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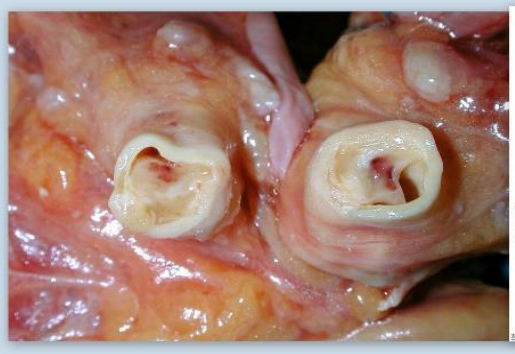
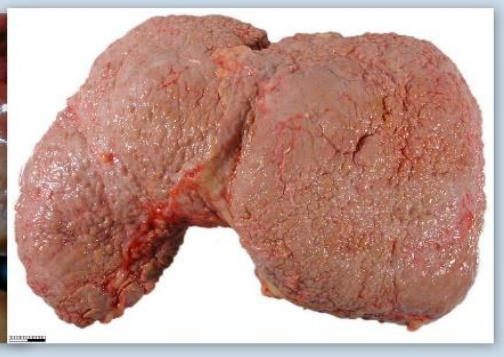

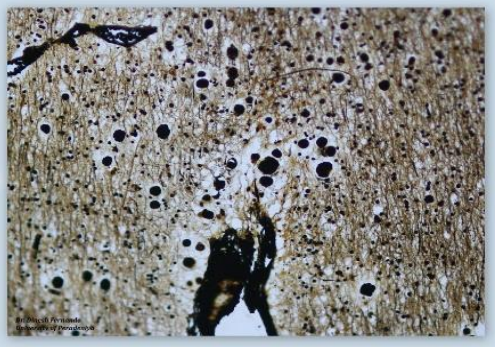

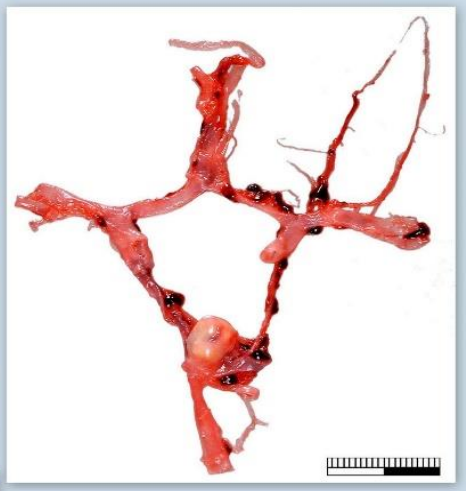
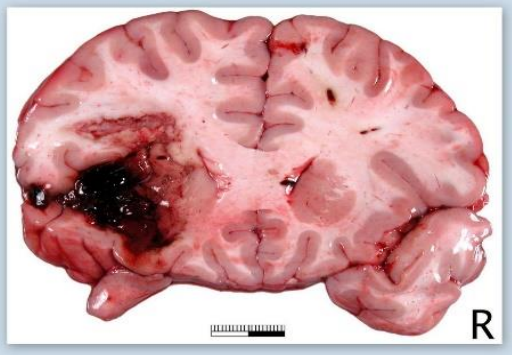

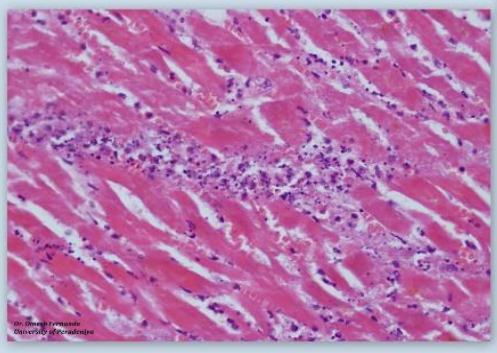
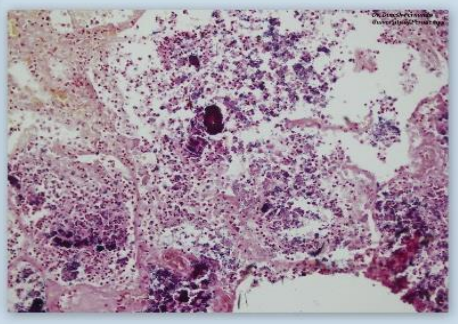

Dinesh Fernando
Sulochana Wijetunge

COLOR ATLAS OF FORENSIC PATHOLOGY

Prof. Dinesh Fernando
MBBS, MD (Forensic Medicine)
DLM, DMJ (Lond)
Dept. of Forensic Medicine

Dr. Sulochana Wijetunge
MBBS, MD (Histopathology)
Dept. of Pathology

Faculty of Medicine,
University of Peradeniya,
Sri Lanka

In case of any questions, comments, suggestions or errors, please mail the authors on dineshf@pdn.ac.lk / dineshmgfdo@yahoo.com or sulochana.wijetunge@med.pdn.ac.lk.

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FOREWORD

The greatest pleasure I experience as a teacher, is to see my students excel in their chosen careers and perform even better than myself. The series of e-booklets prepared to better equip medical officers to handle common conditions likely to be encountered in their day to day forensic practice by Professor Dinesh Fernando, is a good example of one of my students doing better than me!

Dinesh is the son of Emeritus Professor of Community Medicine, Former Head, Department of Community Medicine, Former Dean, Faculty of Medicine and Vice Chancellor of the University of Peradeniya, Malcolm Fernando, who was an illustrious medical academic. Following his father's footsteps, he joined the University of Peradeniya in 2003.

Dinesh was one of my post graduate trainees at the Department of Forensic Medicine and Toxicology, Faculty of Medicine, Colombo, and obtained the doctorate in Forensic Medicine in 2003. He underwent post-doctoral training at the Victorian Institute of Forensic Medicine, Melbourne, Australia, with my colleague and contemporary at Guy's Hospital Medical School, University of London, Professor Stephen Cordner. During this period, he served as the honorary forensic pathologist of the Disaster Victim Identification team in Phuket, Thailand following the tsunami, and was awarded an operations medal by the Australian Federal Police.

He has edited, and contributed chapters to, 'Lecture Notes in Forensic Medicine' authored by the former Chief Judicial Medical Officer, Colombo, Dr. L.B.L. de Alwis and contributed to 'Notes on Forensic Medicine and Medical Law' by Dr. Hemamal Jayawardena. He is the editor of the Sri Lanka Journal of Forensic Medicine, Science and Law. Continuing his writing capabilities, he has compiled an important and unique set of e-booklets which will be a great asset to undergraduate and post-graduate students of Forensic Medicine, and also to our colleagues. Its succinct descriptions of complicated medico-legal issues and clear and educational photographs are excellent. It makes it easy for the students to assimilate the theoretical knowledge of each topic as they have been augmented with histories, examination findings, macroscopic and microscopic photographs of actual cases. In some areas, photographs from multiple cases have been included, so that the students can better appreciate the subtle differences that would be encountered in their practice.

I sincerely thank my ever so grateful student Dinesh, for giving me this great honour and privilege to write the foreword.

Professor Ravindra Fernando

MBBS, MD, FCCP, FCGP, DMJ (London), FRCP (London)
FRCP (Glasgow), FRCP (Edinburgh), FRCPath. (UK)

Senior Professor of Forensic Medicine, General Sir John Kotelawala Defence University, Ratmalana.
Emeritus Professor of Forensic Medicine and Toxicology, Faculty of Medicine, University of Colombo

About the authors.....

Dr. Sulochana Wijetunge is a Senior Lecturer serving at the Department of Pathology, Faculty of Medicine, University of Peradeniya and Teaching Hospital, Peradeniya. She obtained her undergraduate education at the Faculty of Medicine, University of Colombo, and her postgraduate training from Postgraduate Institute of Medicine, University of Colombo, Sri Lanka. International exposure includes training at the University of Southern California, USA and Royal Marsden NHS Foundation Trust, UK. She has 17 years of experience in undergraduate teaching and 12 years of experience as a board certified histopathologist and a post graduate trainer. She has an interest in forensic histopathology and trains the forensic medicine postgraduate students in Pathology.

Dr. Dinesh Fernando is a merit Professor in Forensic Medicine at the Faculty of Medicine, University of Peradeniya and honorary Judicial Medical Officer, Teaching Hospital Peradeniya. He obtained his MBBS in 1994 with Second class honours from the North Colombo Medical College, Sri Lanka, and was board certified as a specialist in Forensic Medicine in 2004. He obtained the postgraduate Diploma in Medical Jurisprudence in Pathology from London in 2005, and possesses a certificate of eligibility for specialist registration by the General Medical Council, UK. He underwent post-doctoral training at the Victorian Institute of Forensic Medicine, Melbourne, Australia. He has also worked at the Wellington hospital, New Zealand, as a locum Forensic Pathologist and as an Honorary Clinical Senior Lecturer at the Wellington School of Medicine and Health Sciences, University of Otago, New Zealand. He was invited to visit and share experiences by the Netherlands Forensic Institute in 2019.

PREFACE

Forensic Medicine in Sri Lanka encompasses, both, examination of patients for medico-legal purposes and conducting autopsies in all unnatural deaths, in addition to those that the cause of death is not known. In the eyes of the justice system in Sri Lanka, all MBBS qualified medical officers are deemed to be competent to conduct, report and give evidence on medico-legal examinations of patients and autopsies conducted by them, as an expert witness. However, during their undergraduate training, they may not get the opportunity to assist, nor observe, a sufficient variety of representative of cases that may be encountered in the future.

Therefore, a series of e-booklets has been prepared to better equip medical officers to handle common conditions that are likely to be encountered in day to day forensic practice. The case histories and macro images are from cases conducted by Prof. Dinesh Fernando, while the microscopic images are from the collections of, either, Prof. Dinesh Fernando or Dr. Sulochana Wijetunge. The selection, photography, reporting of all microscopic images and the short introductions of the pathology of each condition was done by Dr. Sulochana Wijetunge. Most of the macro images used were taken by Louise Goossens – a medical photographer par excellence.

Dr. Madhawa Rajapakshe contributed immensely in preparing the photographs for publication. Ms. Chaya Wickramarathne did a yeomen service in design, lay out and formatting the booklet. If not for the many hours she spent in discussing with the two authors, and editing these cases over several months, these booklets would not have seen the light of day. This is being continued by Ms. Isuruni Thilakarathne.

Prof. Dinesh Fernando
Dr. Sulochana Wijetunge

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CARDIOVASCULAR DISEASES

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CARDIOVASCULAR DISEASE

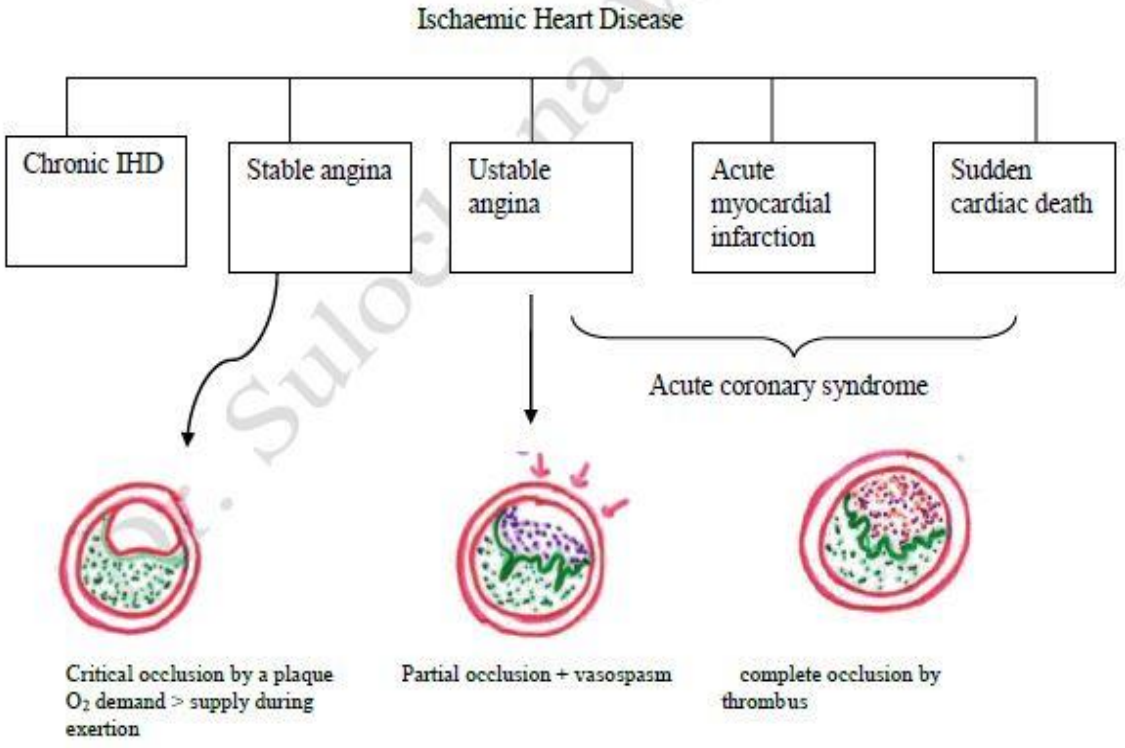
CORONARY ARTERY DISEASE

A) Background

Myocardial ischaemia could be due to reduced blood supply (e.g., coronary artery insufficiency) or increased myocardial demand (e.g, left ventricular hypertrophy) or both (coronary artery insufficiency with left ventricular hypertrophy as in hypertensives).

Myocardial infarction – myocardial death due to severe prolonged ischaemia. Severe acute ischaemia lasting 20 – 40 minutes or more causes irreversible changes in myofibrils and cell death. When such ischaemic states last more than 2 – 4 hours, infarction progresses, involving larger areas.

Coronary artery insufficiency can produce a spectrum of ischaemic manifestations in the myocardium as follows.



B) Coronary artery causes of acute myocardial infarction

1. Atherosclerotic plaques
2. Coronary arterial spasms
3. Myocardial bridging of coronary arteries
4. Coronary artery dissection
5. Coronary thrombo-embolism
6. Vasculitis

1. Coronary artery atherosclerotic plaques

The most common and important cause for ischaemic heart disease (IHD)/ myocardial infarction is occlusion of coronary arteries by atherosclerotic plaques.

The changes that can occur in these plaques are,

- Uninterrupted plaques narrowing the coronary artery lumina
- Eroded plaques with platelet aggregations on the surface without complete obstruction of the arterial lumen
- Ulcerated or eroded plaques with superimposed thrombosis producing complete obstruction of the lumen
- Bleeding into a plaque

Uninterrupted plaques narrowing the coronary artery lumina

When the degree of occlusion reaches a critical level the blood supply through the partially occluded artery may not be sufficient to meet with the increased physiological demands such as running, climbing stairs.

With increased exertion that part of the myocardium supplied by the partially occluded artery become ischaemic and patient may experience an ischaemic pain which is relieved by rest. Such patients
can tolerate a certain level of exertion and with time patients learns the limits of exertion that they can tolerate – stable angina. Stable angina is usually associated with stable plaques. Patients with stable angina usually have chronic IHD.

When there are widespread partial occlusions in the coronary arterial system low grade chronic myocardial ischaemia may lead to diffuse myocardial fibrosis, especially in the left ventricle. Over a period of time the patient may develop Chronic IHD and congestive cardiac failure.

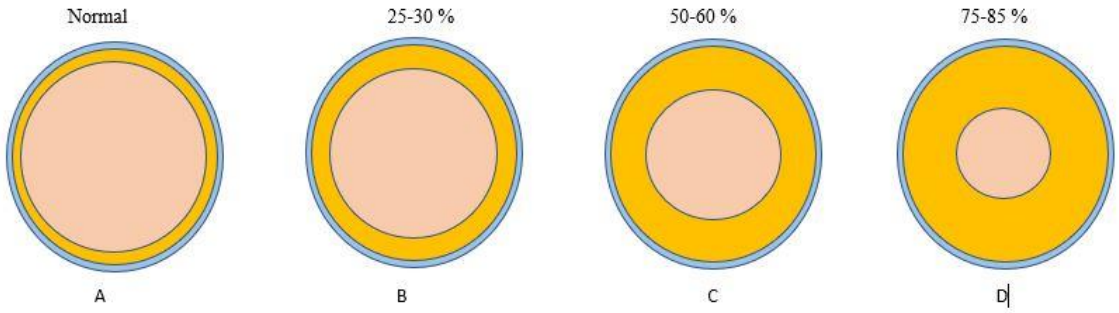


Figure 1: Schematic representation of occlusion by concentric atheroma

Note: Variation of the diameter and the area of the lumen of the coronary artery are compared with respect to the normal coronary artery (A). If diameter is decreased by 10%, area of the lumen is decreased by 20% (B). If diameter is decreased by 25%, area of the lumen is decreased by 50% (C). If diameter is decreased by 50%, area of the lumen is decreased by 75% (D).

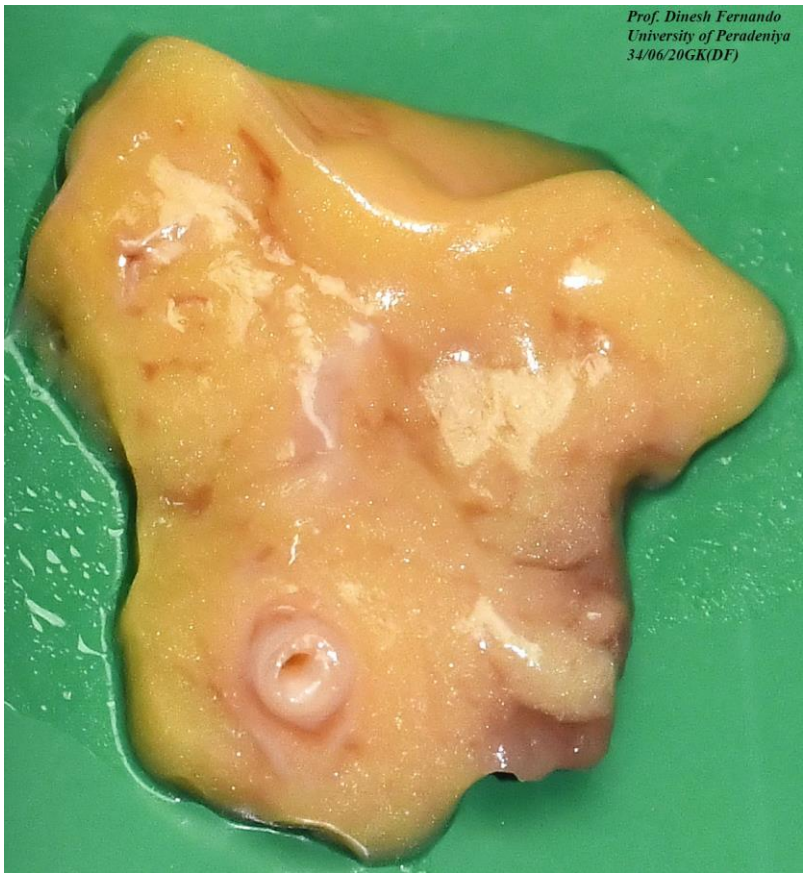


Figure 2: Concentric Atheroma

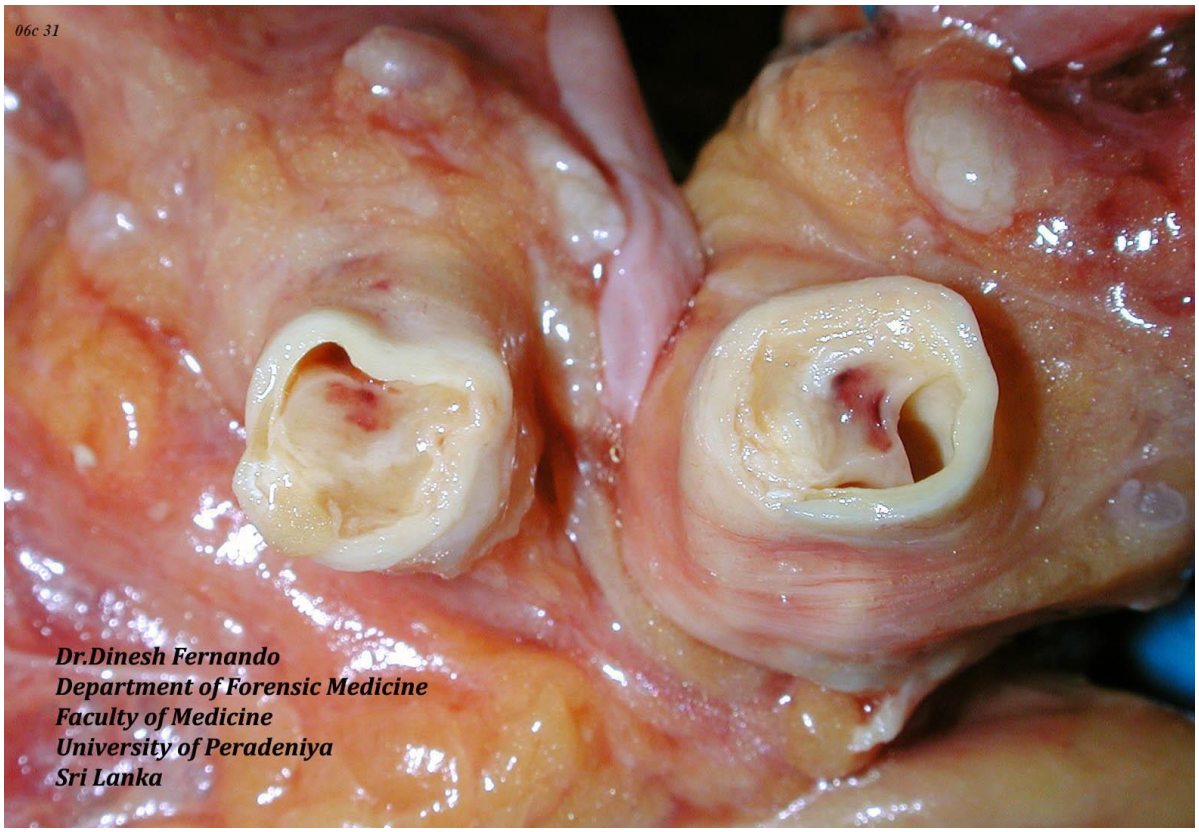


Figure 3: Haemorrhage into an eccentric atheromatous plaque in the coronary artery

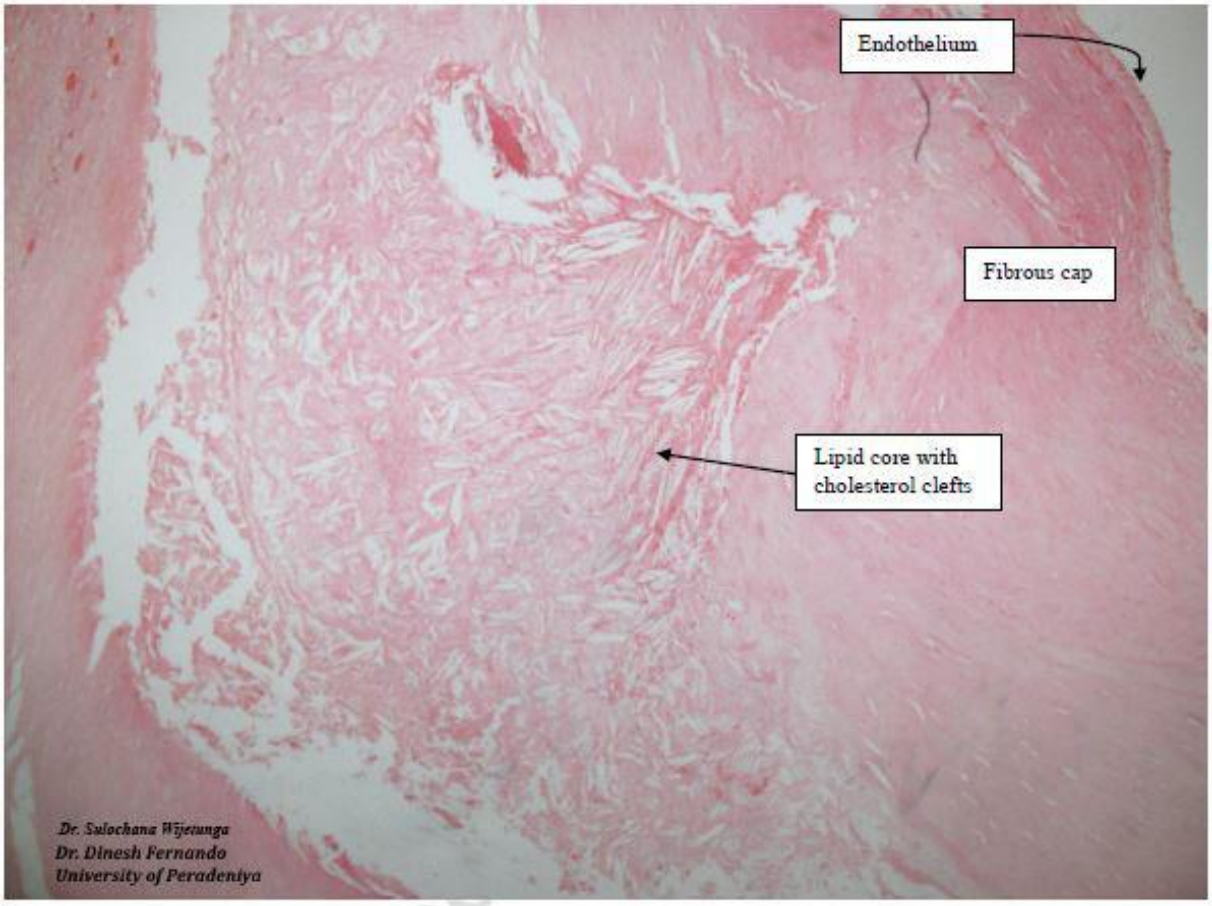


Figure 4: An uninterrupted atheromatous plaque narrowing the coronary artery lumen. Atheromatous plaque is an intimal collection of lipids. The picture shows a typical atheromatous plaque covered with a fibrous cap and a central lipid core. The lipid core can be identified by presence of cholesterol clefts.

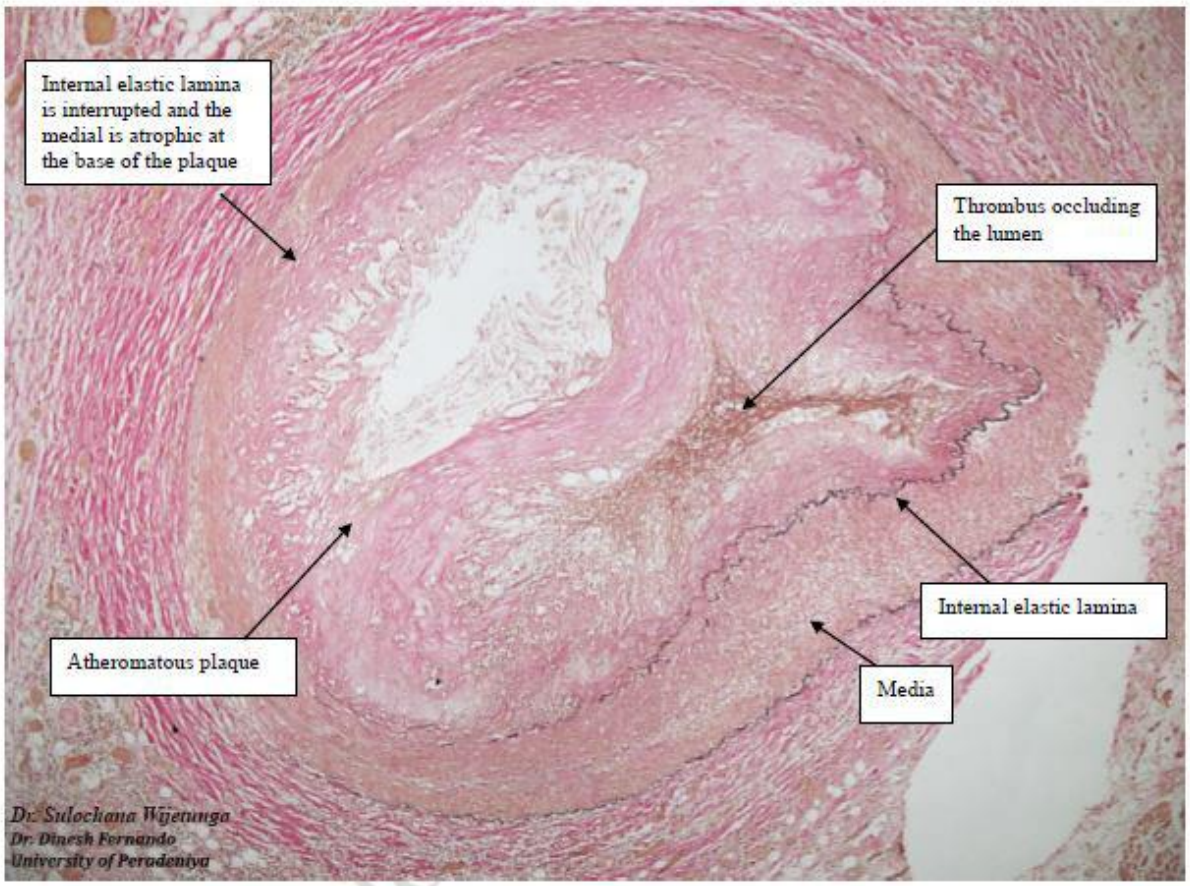


Figure 5: Ulcerated atheromatous plaque with superimposed thrombosis, completely occluding the arterial lumen (elastic van Gieson stain). Staining of the internal elastic lamina shows the intimal location of the plaque. The internal elastic lamina is disrupted and the media is atrophic at the base of the plaque due to pressure.

Figure 6: An uninterrupted atheromatous plaque narrowing the coronary artery lumen. Note that this plaque is almost completely composed of fibrous tissue and the typical lipid core is not seen. In long standing plaques, when there is no active deposition of lipids, the plaques get ultimately replaced by fibrous tissue. Such plaques are less likely to develop acute plaque changes and, are therefore, called stable plaques.

The events that are described below are the acute plaque changes which give rise to acute coronary syndrome, ranging from unstable angina through myocardial infarction to sudden cardiac death
.

Eroded plaques with platelet aggregations on the surface without complete obstruction of the arterial lumen

With superficial plaque erosions there can be platelet aggregation over the erosions, producing sudden increase in the degree of arterial occlusion, without complete occlusion. However, vasoactive amines released by activated platelets causes vasospasm in the uninvolved arterial wall causing temporary complete occlusion of the coronary artery. This could lead to acute coronary syndrome.

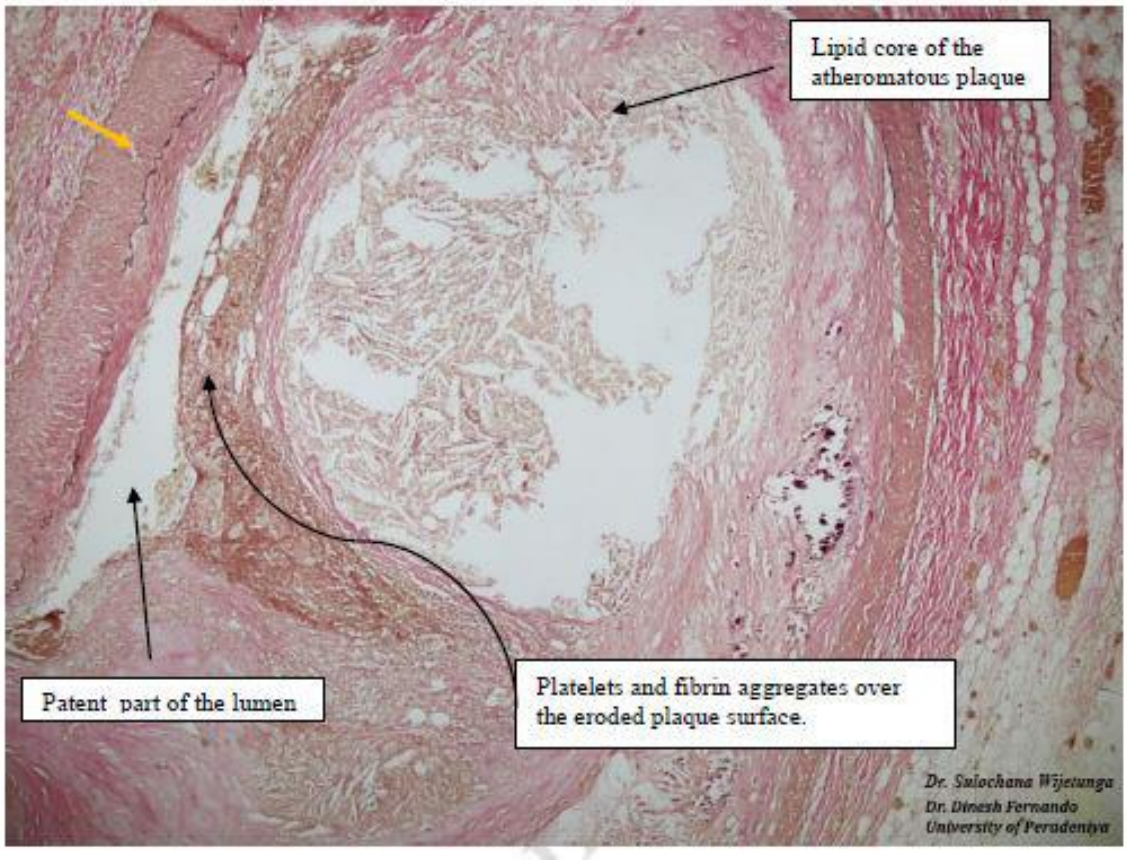


Figure 7: An eroded plaque with platelet aggregations on the surface, without complete obstruction of the arterial lumen. This is a typical plaque that is vulnerable to get acute plaque changes: there is a large lipid core (identified by the presence of cholesterol clefts) with only a thin fibrous cap. Such unstable plaques indicate active deposition of lipids. The yellow arrow indicates the uninvolved part of the arterial wall. The vasoactive amines act on this part of the wall and induce spasms which produce temporary complete occlusion of the lumen.

Ulcerated or eroded plaques with superimposed thrombosis producing complete obstruction of the lumen

Plaque ulceration with superimposed thrombosis can completely occlude the coronary artery and shut down the blood supply to an area of the myocardium for a period of time, sufficient to produce cell death.

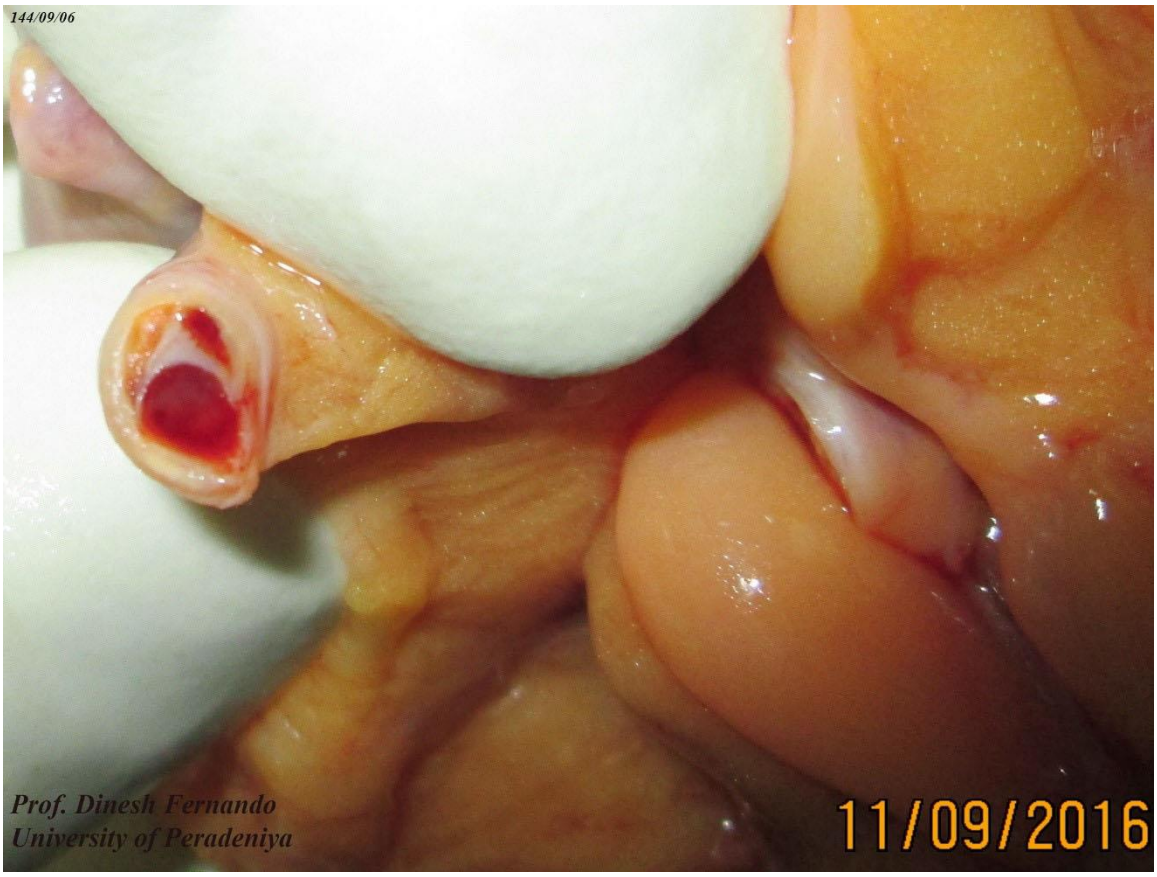


Figure 8: Plaque with superimposed thrombosis

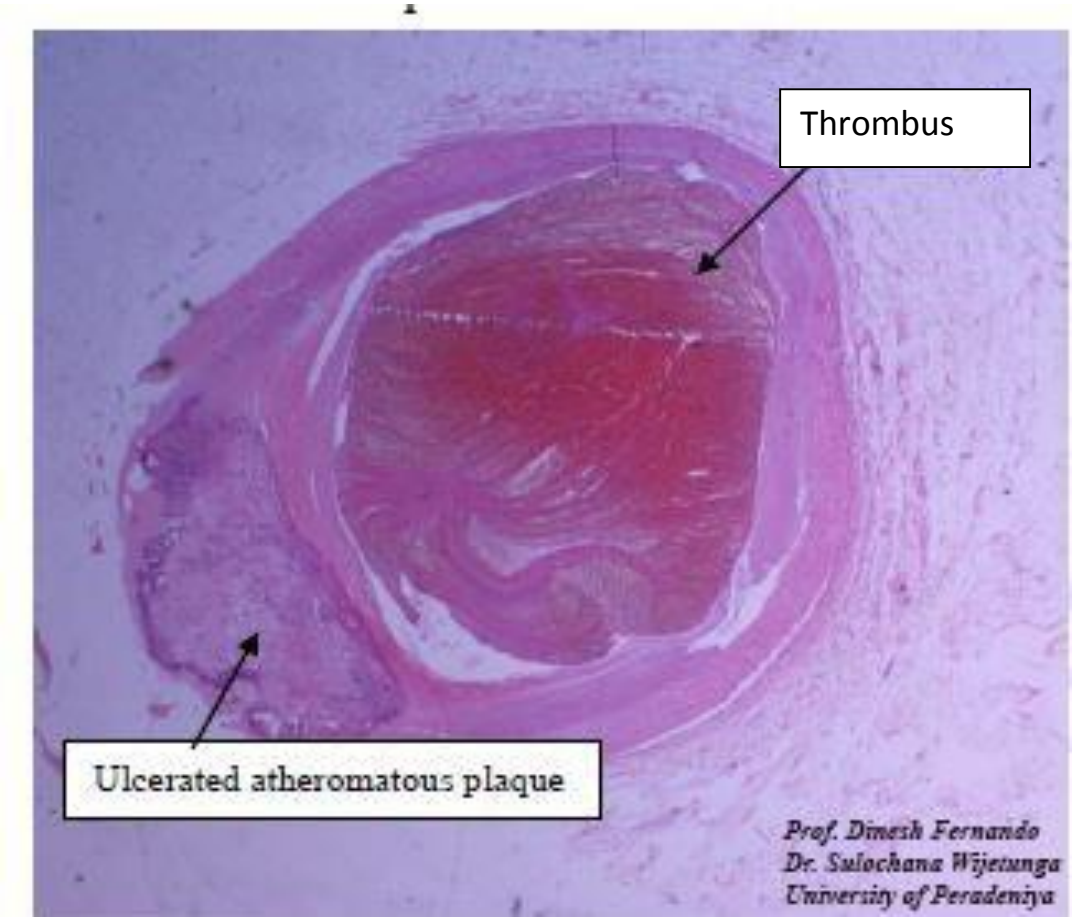


Figure 9: An ulcerated eccentric plaque with superimposed thrombosis, completely occluding the arterial lumen

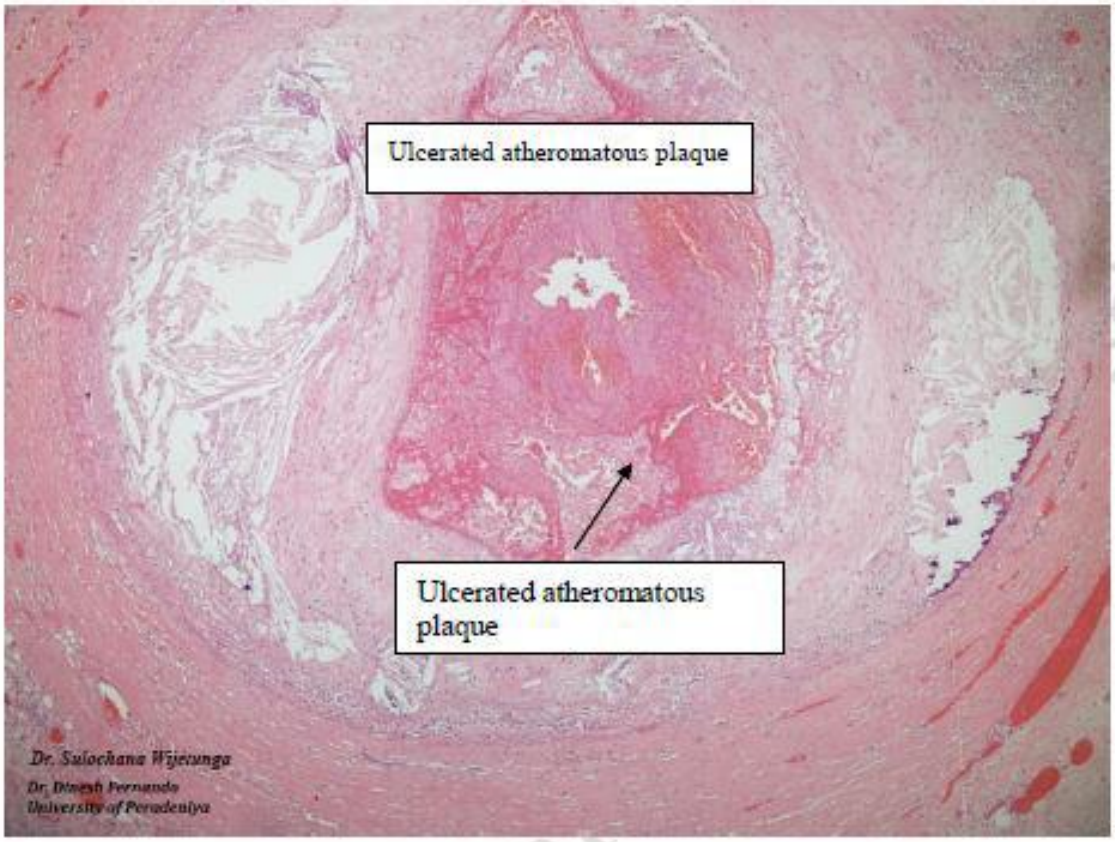


Figure 10: Ulcerated atheromatous plaque with superimposed thrombus, completely occluding the arterial lumen. The atheromatous plaque is more circumferential in this example. The cholesterol clefts in the lipid core as clearly seen.

Bleeding into a plaque

Bleeding into the atheromatous plaque can produce an acute increase of the degree of arterial occlusion. This can produce acute coronary syndrome.

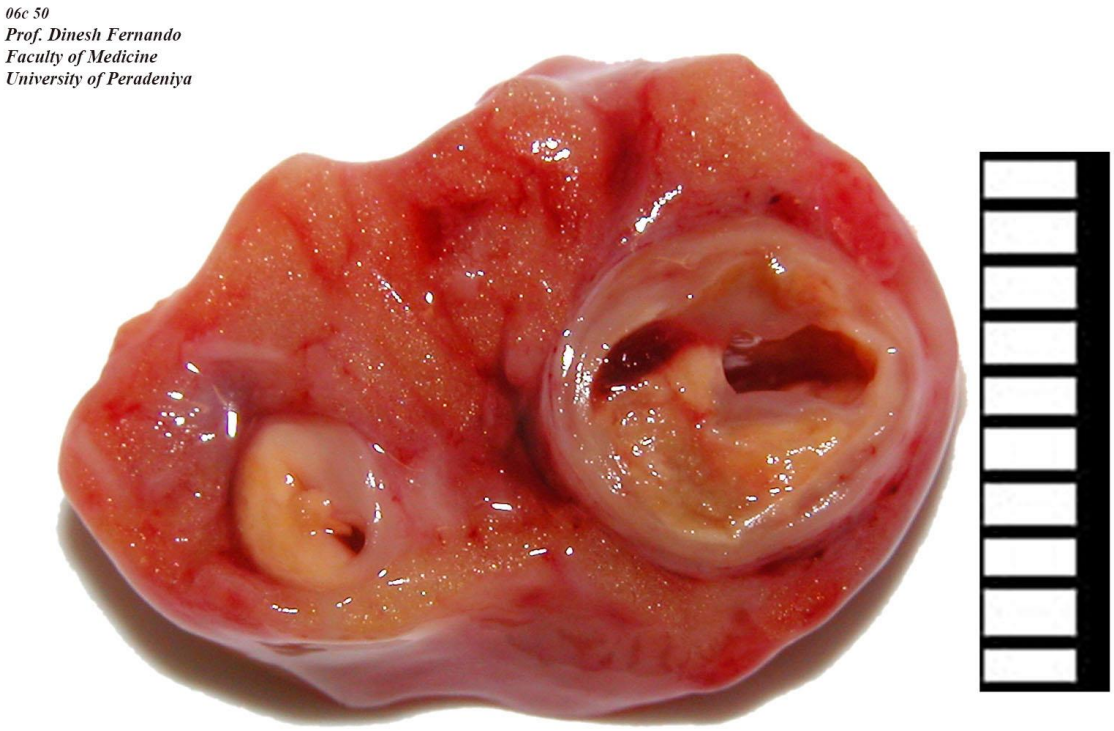


Figure 11: Bleeding into an eccentric atheromatous plaque

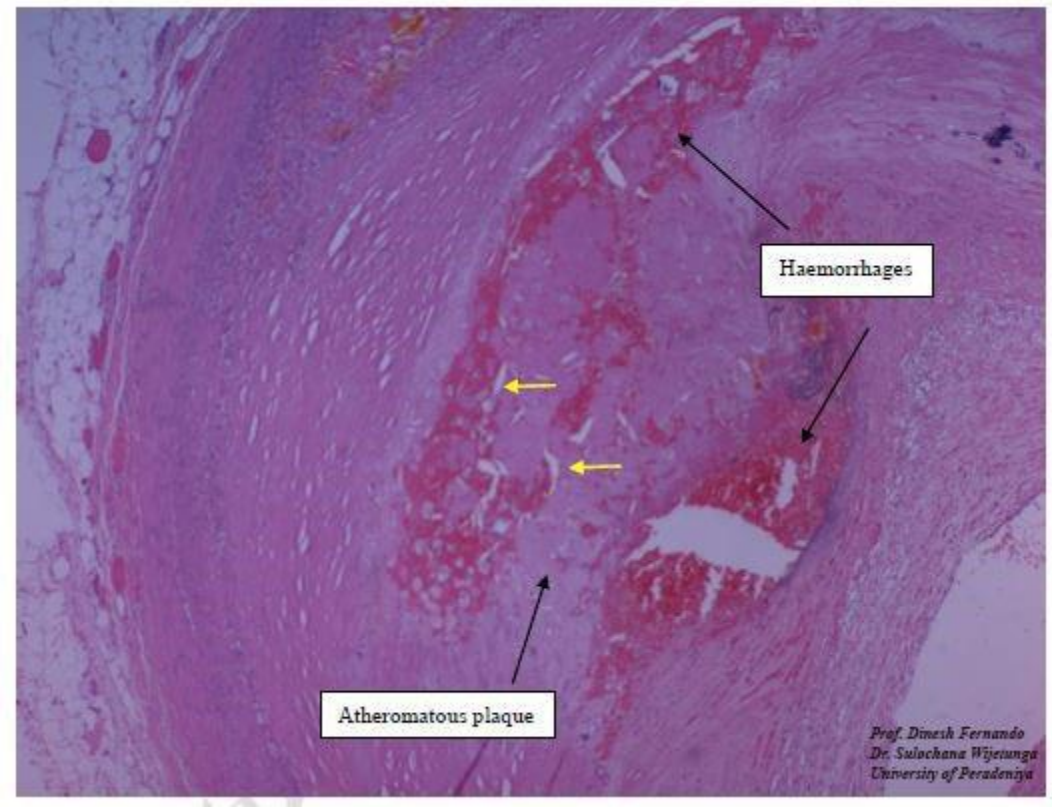


Figure 12: Bleeding into a plaque

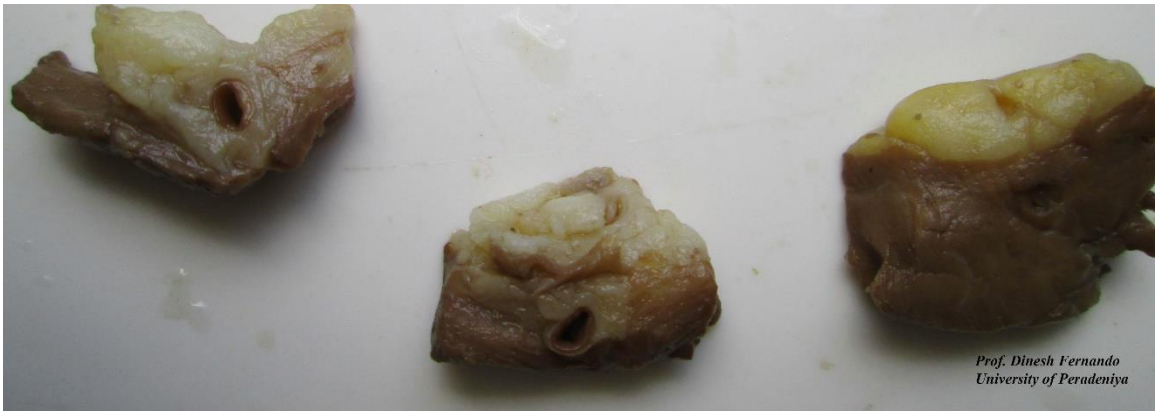
2. Coronary arterial spasms

Intense and prolonged coronary arterial spasms can produce acute coronary syndrome. However, these cannot be demonstrated by morphological means. Therefore, the conclusions are made based on circumstantial evidence and exclusion of other coronary causes.

3. Myocardial bridging of coronary arteries

Normally coronary arteries and their main branches are located within epicardial fat. Occasionally segments of these branches can have an intra-myocardial course and get compressed during systole. Usually, such events are asymptomatic. However, severe bridging of a major coronary artery can produce acute coronary syndrome.

(a)



(b)

Figure 13(a, b): Myocardial bridging of coronary arteries

Figure 14: Myocardial bridging of a coronary artery branch

4. Coronary artery dissection

Spontaneous coronary artery dissection is a rare cause of acute coronary syndrome, that typically occurs in young, otherwise healthy, women.

In arterial dissection, blood tracts down along the planes of media, forming a blood-filled channel within the arterial wall. Arterial dissection is more common in the aorta. However, it can rarely occur in muscular arteries such as the coronary arteries.

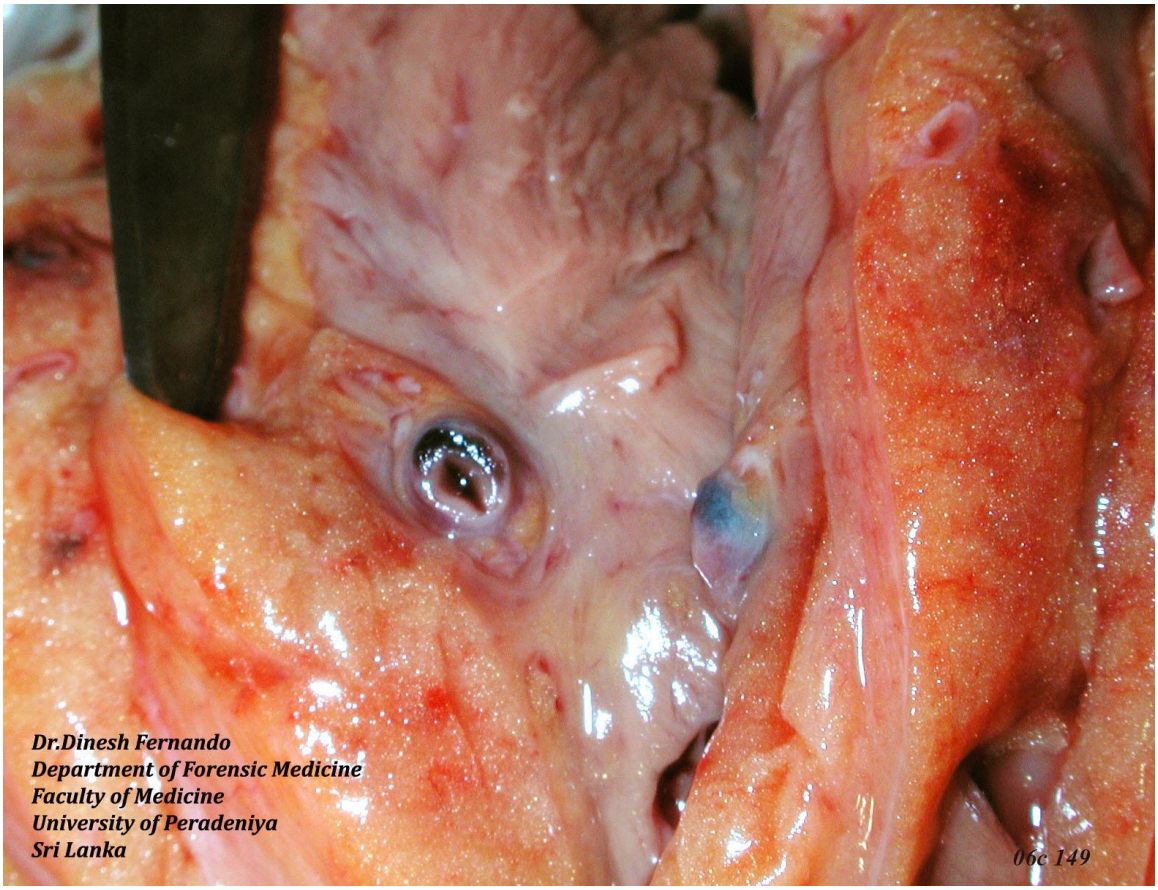
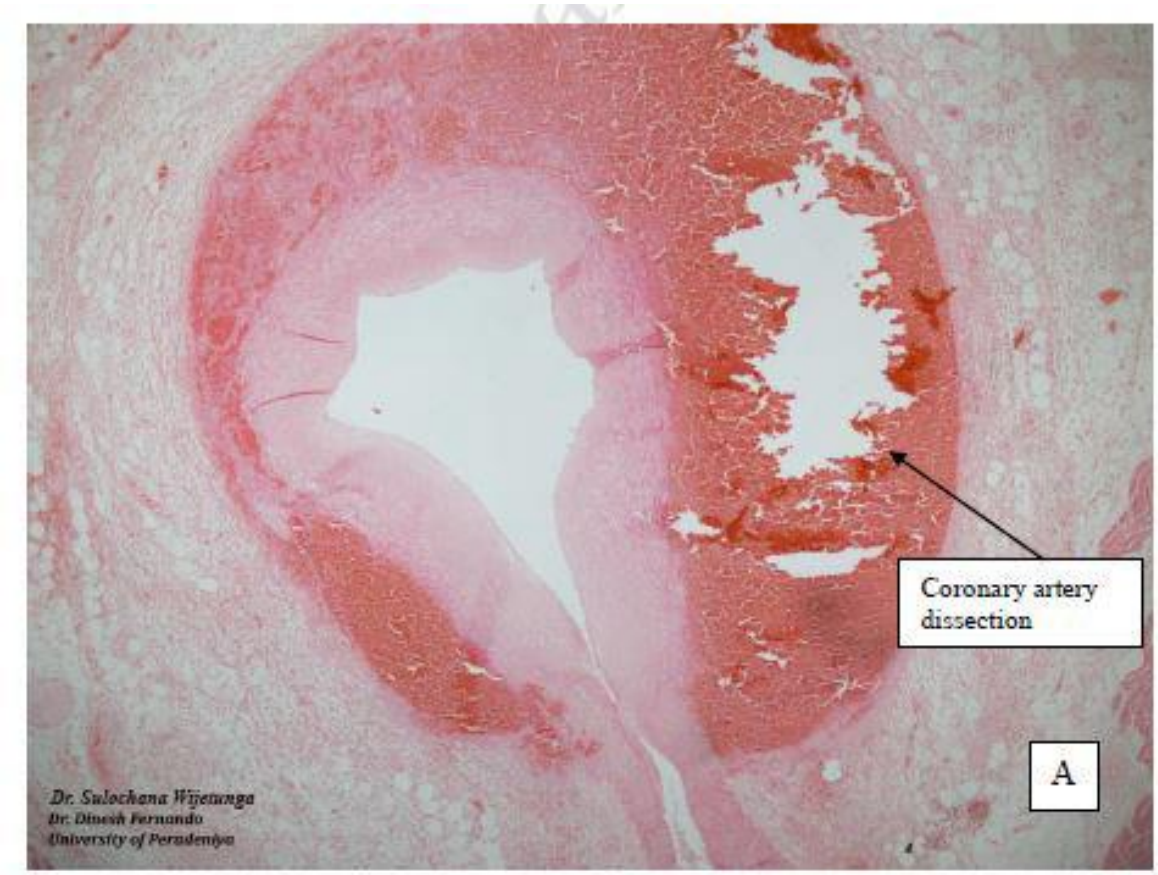


Figure 15: Coronary artery dissection


Coronary artery dissection
A
Dr. Sulochana Wijetunga
Dr. Dinesh Fernando
University of Peradeniya

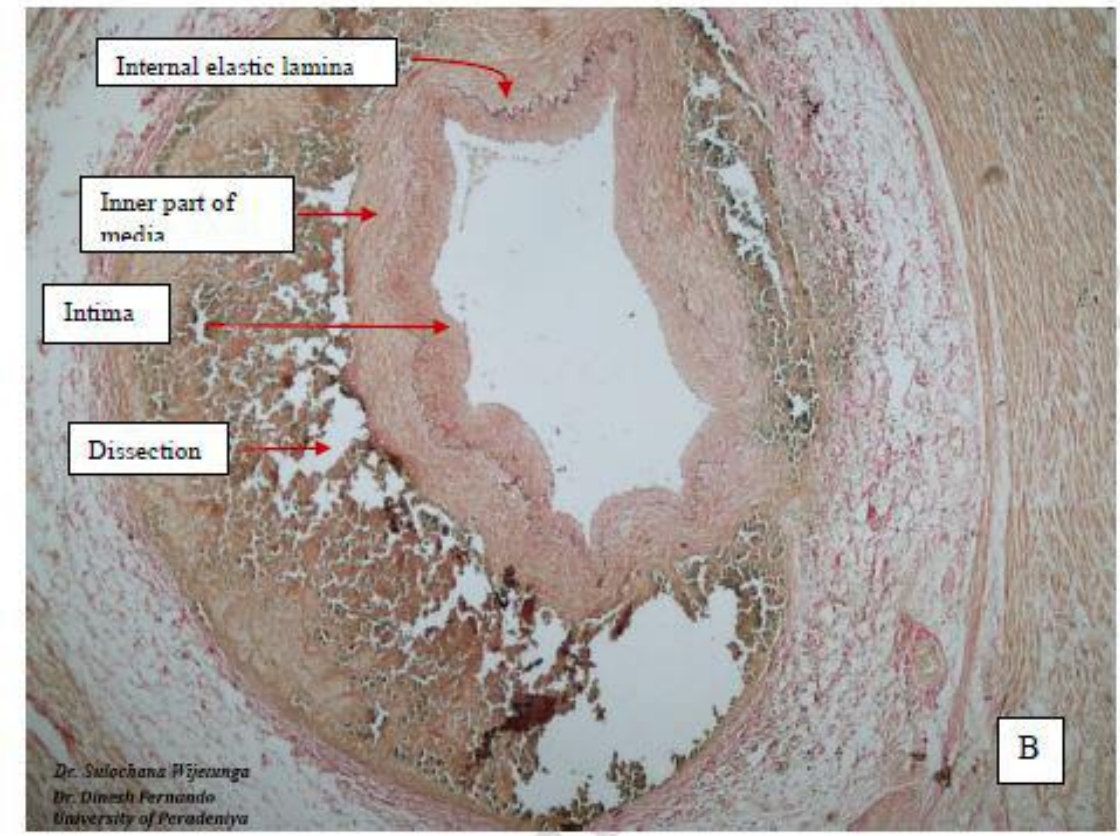


Figure 16: Coronary artery dissection
A) Haematoxylin and eosin stain show a blood-filled channel within the arterial wall
B) Elastin stain demonstrates that the dissection has occurred within the medial planes

5. Coronary thrombo-embolism

Thrombi in coronary arteries are commonly due to in-situ thrombosis on ulcerated coronary arterial atherosclerotic plaques. However, rarely coronary arteries can get occluded due to thrombo-emboli.

The sources of such thrombo-emboli could be

- Left atrial thrombi – in atrial fibrillation
- Ventricular mural thrombi – following a transmural myocardial infarction
- Vegetations in bacterial endocarditis
- Paradoxical thrombi – venous thrombi entering the left heart through an atria septal defect when reversal of blood flow has occurred (Eisenmenger's syndrome).

6. Vasculitides

Vasculitis is a rare cause of coronary artery thrombosis. Kawasaki disease, a type of childhood vasculitis shows a predilection for coronary arteries. Kawasaki disease is the commonest cause of childhood myocardial infarctions.

In adults, polyarteritis nodosa can rarely affect the coronary arteries.

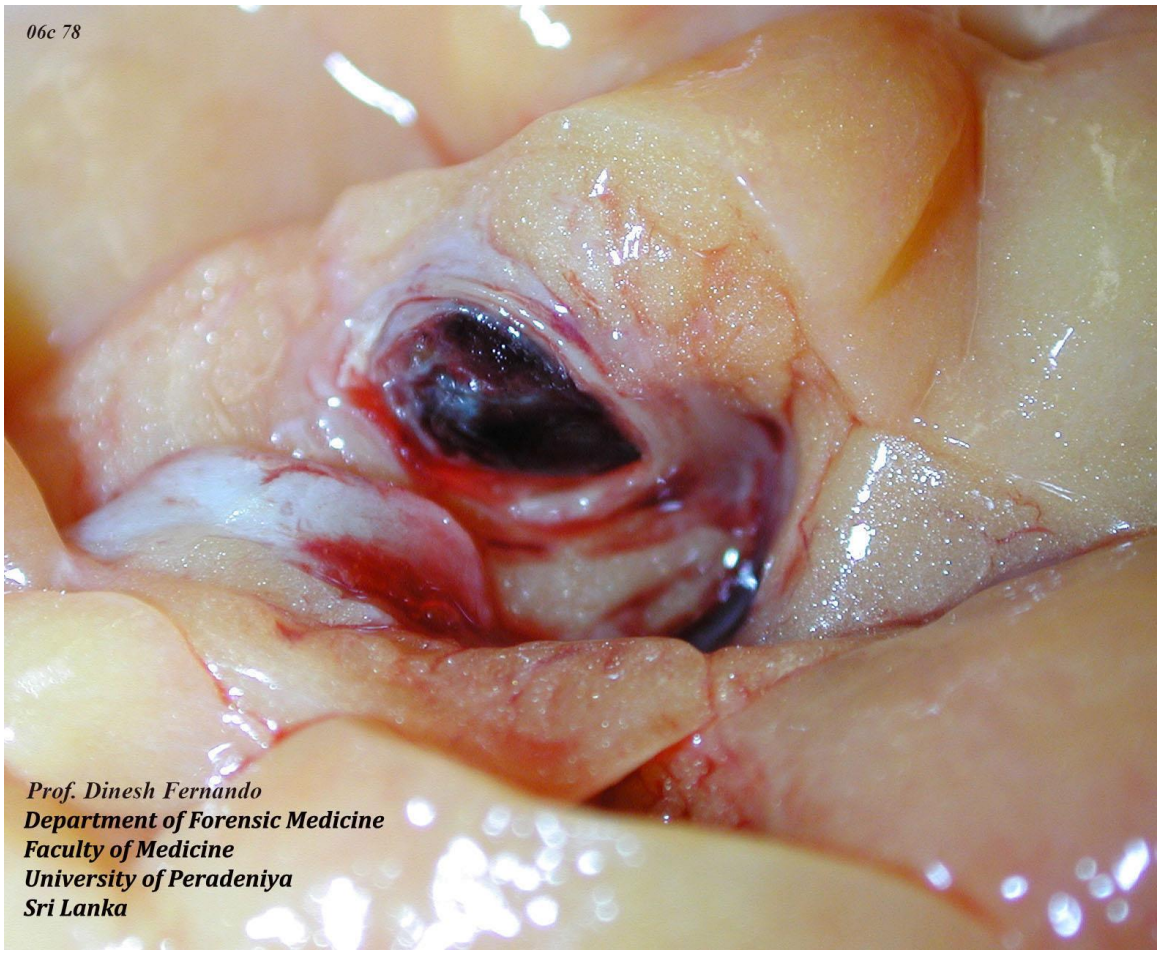
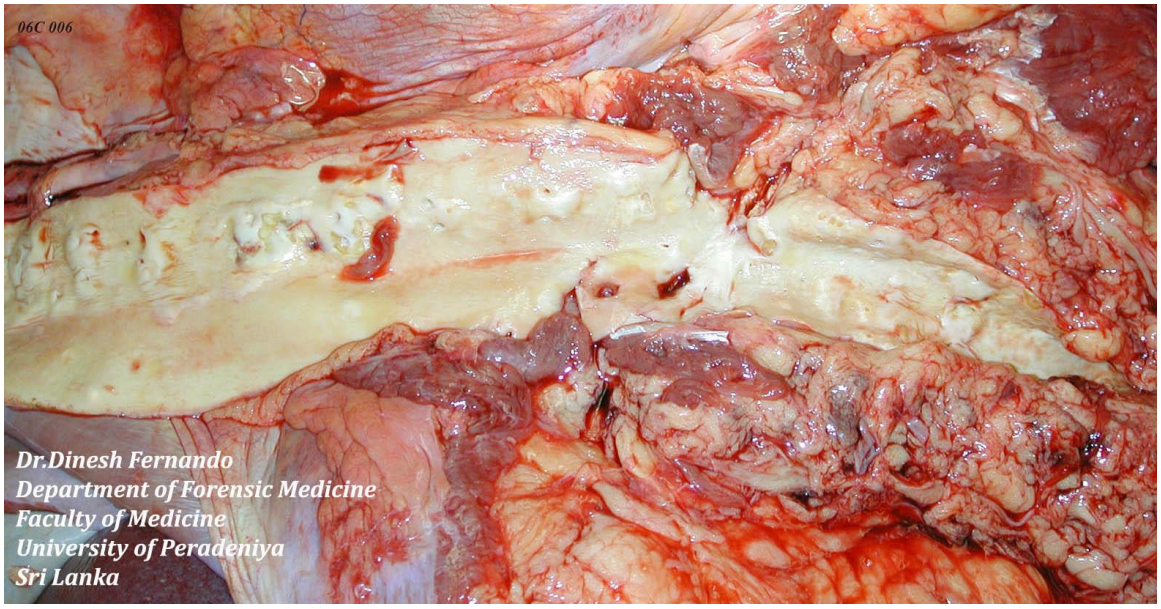


Figure 17: Coronary thrombo-embolism

C) Atheroma in other vessels



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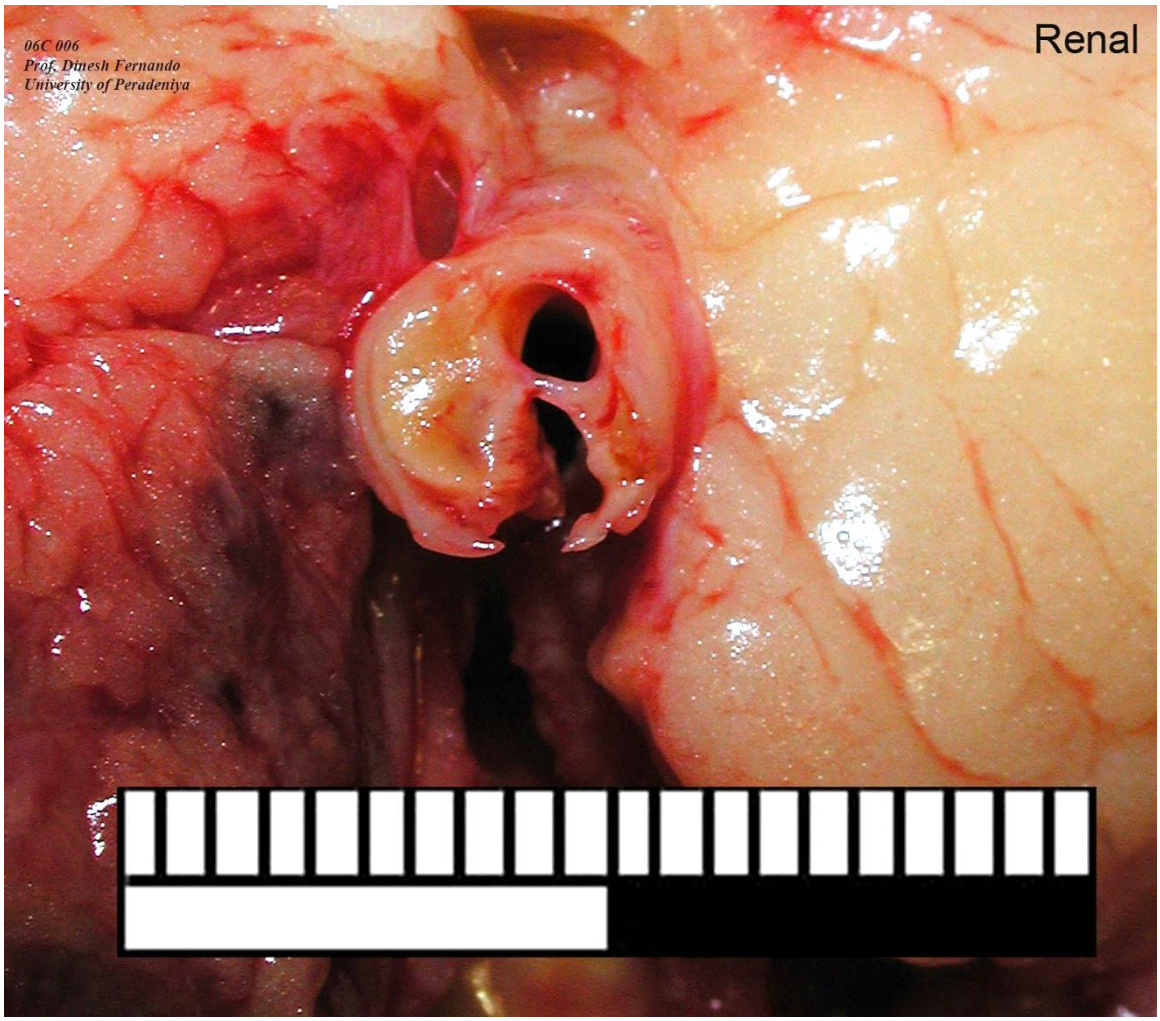
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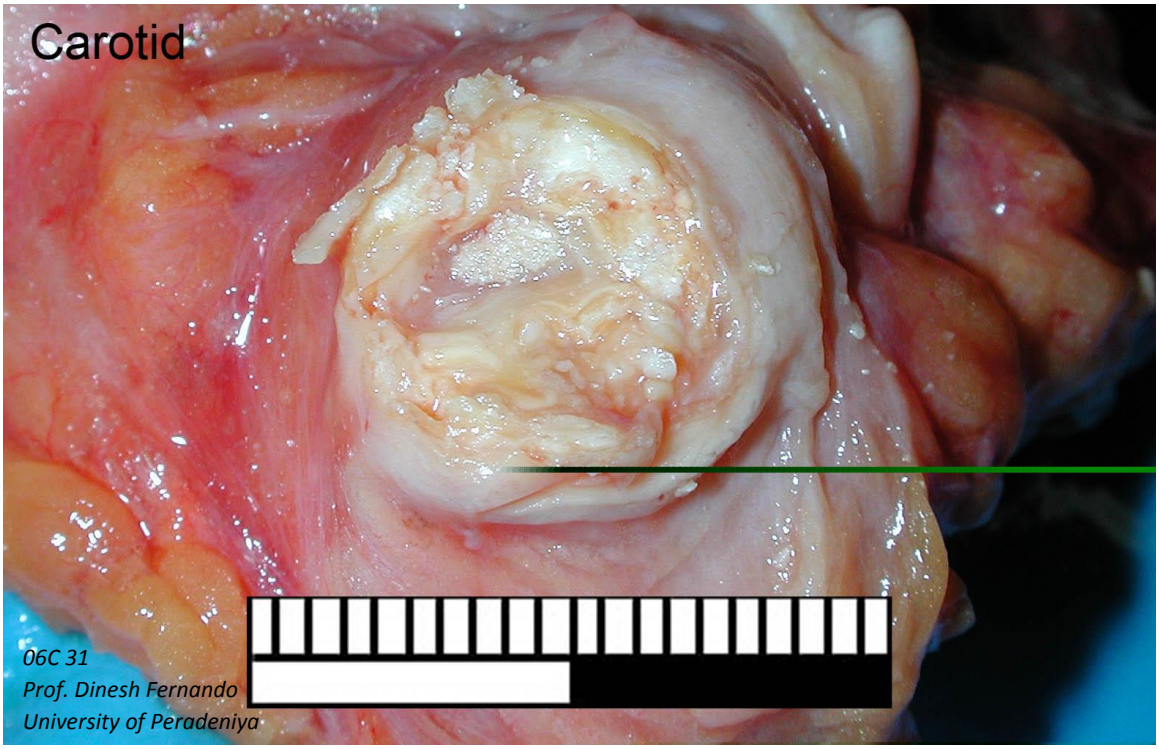
Prof. Dinesh Fernando
University of Peradeniya
Renal

(b)



(c)

Figure 18: Atheroma in the aorta (a), Renal artery (b) and Carotid artery (c)

Myocardial Infarction

Identification and dating of myocardial infarction

CARDIOVASCULAR DISEASE

MYOCARDIAL INFARCTION

Identification and dating of an acute myocardial infarction

Immediately after an acute myocardial infarction (AMI) there are usually no macroscopically or microscopically detectable changes in the myocardium. Initial macroscopic changes occur after about 12 hours and microscopic changes after about 4-6 hours. Therefore, post mortems performed on patients died soon after an AMI may not show any changes in the myocardium.

Given below are the histological criteria to identify and determine the age of a myocardial infarction. It should be noted that the given time sequence for evolution of an infarction could vary depending on many variables including the macro and micro environmental changes. Furthermore, neither of these changes starts or ends abruptly; they appear gradually, reach a peak and then wane off.

Overall, predominant changes in the first week are those of necrosis and acute inflammatory cells (neutrophils and their debris); the second week macrophages and other mononuclear inflammatory cells predominate and the third week onwards granulation tissue formation and collagen accumulation. However, not infrequently necrotic myocytes may be seen in the centres of the infarcts even in the third week.

The time required for complete healing of an infarction to form a scar is variable and may take about 6 to 7 weeks or longer.

First 12 hours

Macroscopy: No changes usually. However, necrotic areas may be highlighted by nitro-blue tetrazolium

Microscopy

Reliable recognition of myocardial infarctions within the first 12 hours with haematoxylin and eosin stain could be challenging, often the changes are either absent or too subtle to be identified.

Ancillary methods that may be useful to identify changes are:

1. Special stains – nitro- blue tetrazolium
   This special stain can be used on macroscopic sections by painting them or on fresh (unfixed) histology sections.

   Dehydrogenase enzyme present in viable myocytes turns the chemical into a deep blue colour. Therefore, the viable tissue stains blue and dead tissue remains unstained.

Figure 1:Negative, showing an even purple stained myocardium without discolouration, implying no ischaemic changes

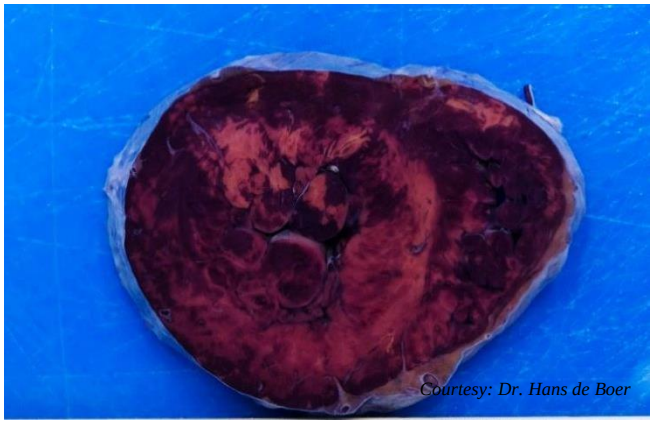


Figure 2: Yellow area in the dorsal part of the left and right ventricle. This implies necrosis, signalling ischaemic damage of at least about a day

2. Immunohistochemistry

Complement 9 (C9) staining is absent in normal myocardial cells but expressed hypoxic cells and therefore, stains

myocytes which has undergone recent necrosis.

Troponin T is expressed by normal myocardial cells but shows negative staining in necrotic myocardial cells. However, immunohistochemistry is still not widely used to date early infarctions.

Wavy myocardial fibres and contraction bands (see notes below) could appear even during the first 12 hours

12 – 24 hours

Macroscopy: congested appearance or dark mottling due to extravasated red cells. If bleeding is not prominent may appear pale.

Microscopy: Main features are

- Wavy myocardial fibres
- Contraction bands
- Loss of cross striations and cytoplasmic eosinophilia
- Cytoplasmic degeneration may also appear
- Nuclear pyknosis, karyorrhexis and karyolitic.

Wavy myocardial fibres with intercellular oedema and contraction bands start appearing first. The other changes appear later and evolve to be more prominent in the 2nd day.

Wavy myocardial fibres are thinned and stretched myocardial fibres which has gained a wavy appearance and associated with intercellular oedema. Wavy fibres may have resulted from stretching of noncontractile myocytes due to bulging of ischaemic regions during systole.

These fibres may also be seen at the peripheries of older infarcts.

Contraction bands are irregular eosinophilic transverse bands on H &E stain. These bands are clearly seen when stained with phosphotungstic acid haematoxylin (PTAH) which stains the myocytes blue.

This phenomenon is due to intense hypercontraction in dying myocytes due to increase influx of calcium into the cytoplasm that occur with plasma membrane damage. Contraction bands are more prominent in reperfusion injuries and death following ventricular fibrillation; changes are diffuse in the latter situation. Focal collection of myocytes with contractions bands is a reliable indicator of early infarction.

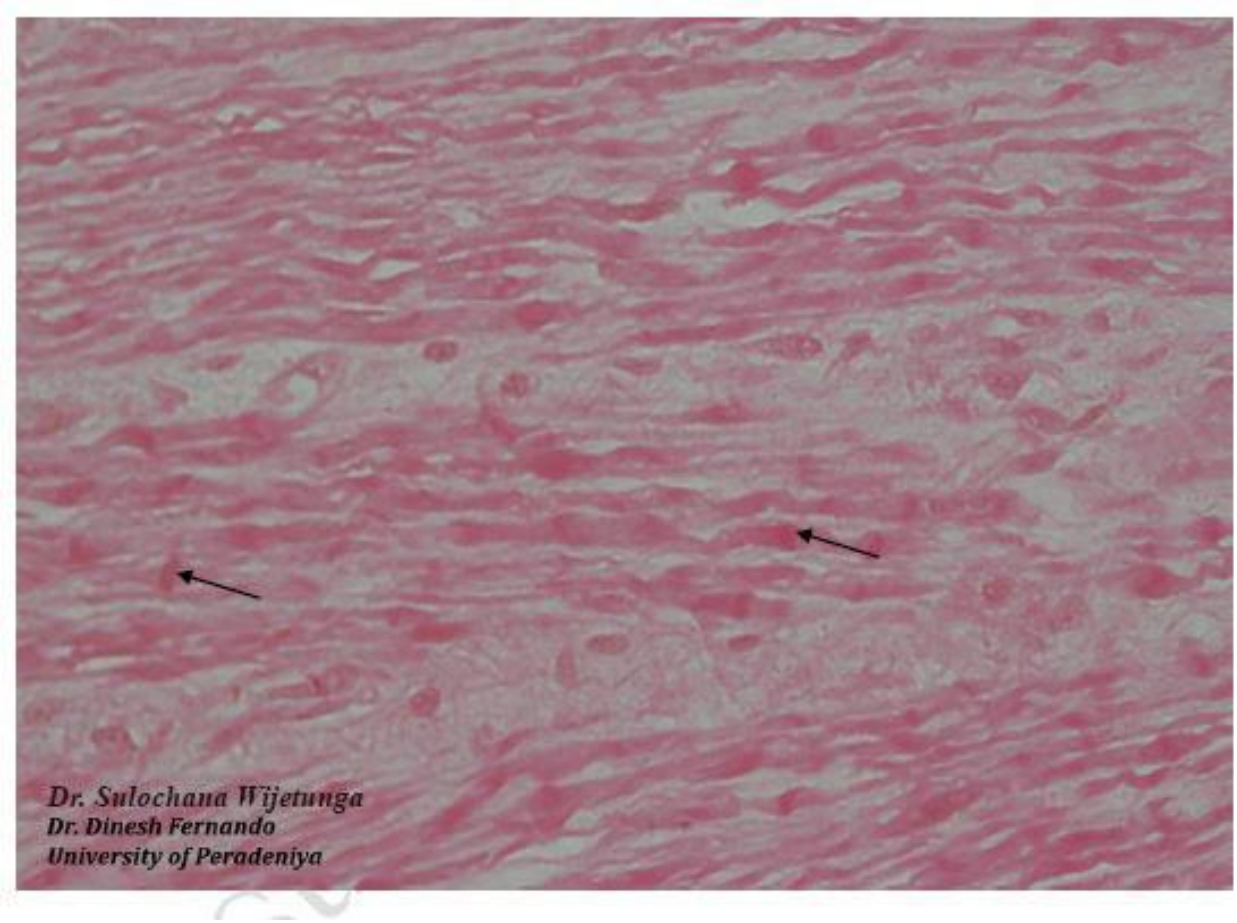


Figure 1: Early myocardial infarction 24 to 48 hours

Contraction bands (arrows) and wavy myocardial fibres

24 to 48 hours

Macroscopy: The infarcted area becomes pale with hyperaemic borders

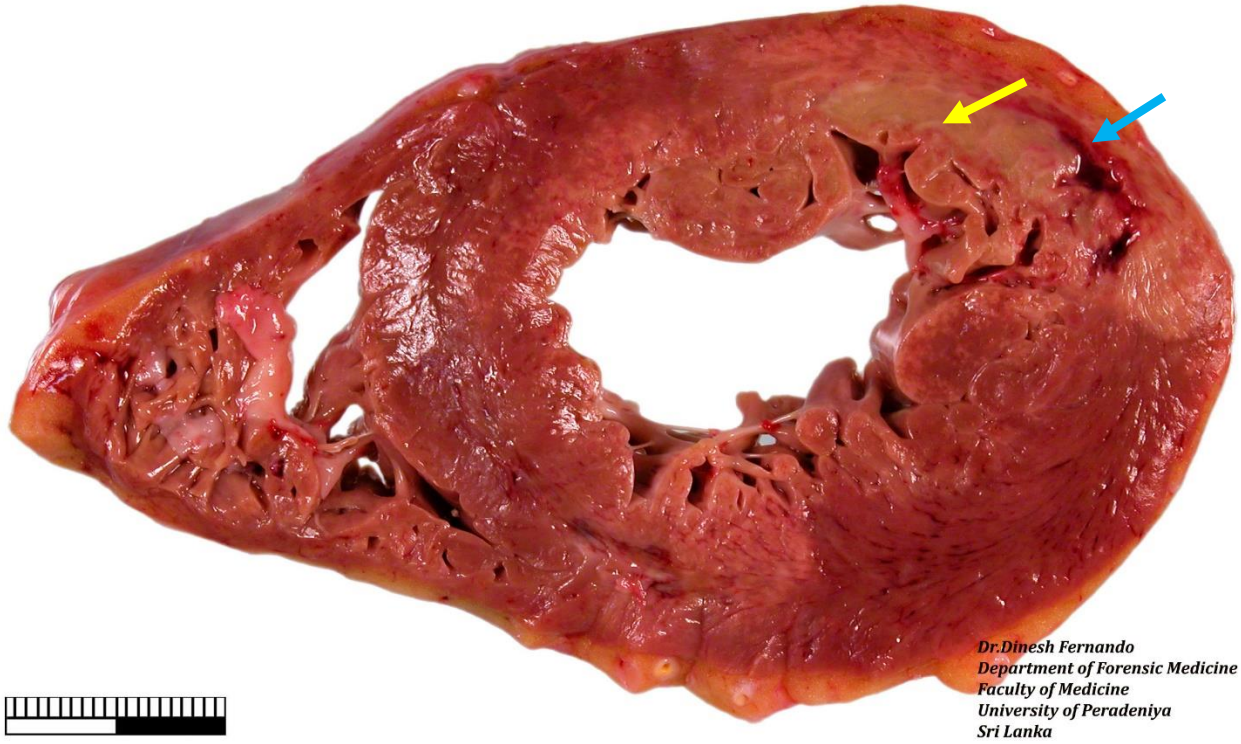


Figure 2: Infarcted area which had become pale (yellow arrow) with hyperaemic borders (blue arrow)

Microscopy: Features are

- Nuclear pyknosis and karyorrhexis.
- Cytoplasmic eosinophilia and degeneration
- Focal areas of haemorrhage into the infarcted area.
- Congested blood vessels in the surrounding viable tissue due to acute inflammatory response in the viable tissue.

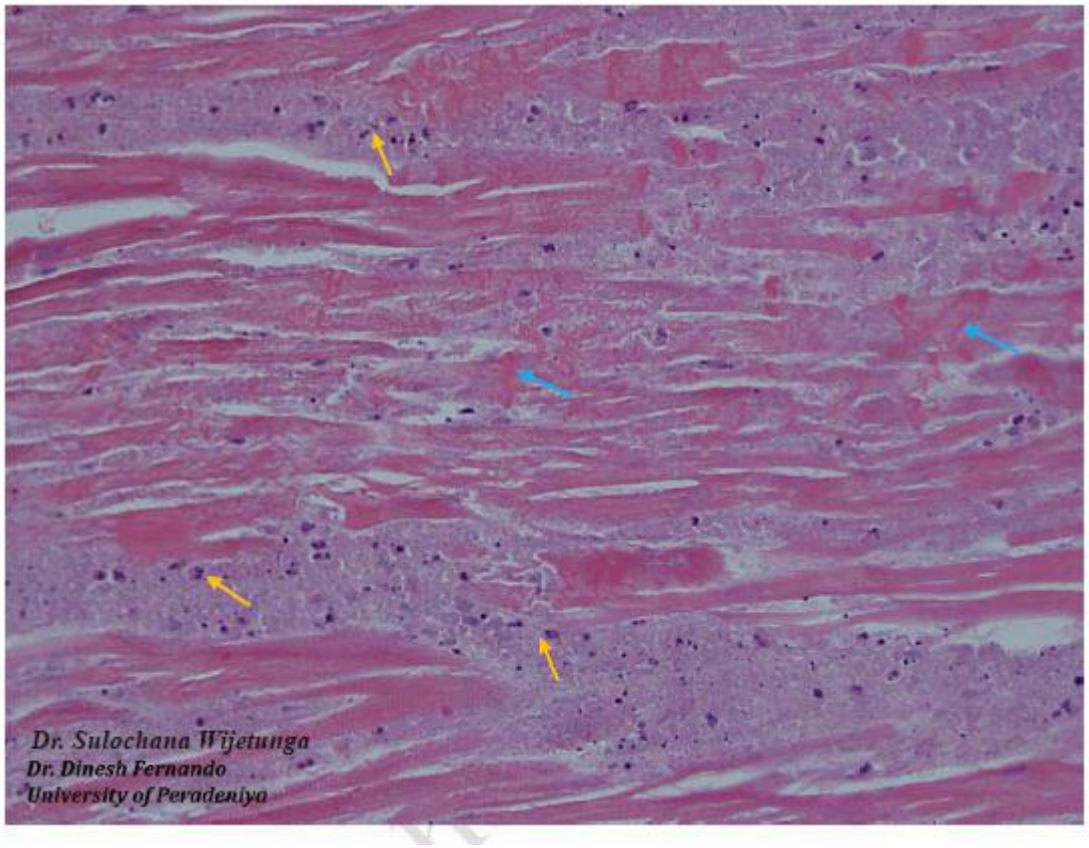


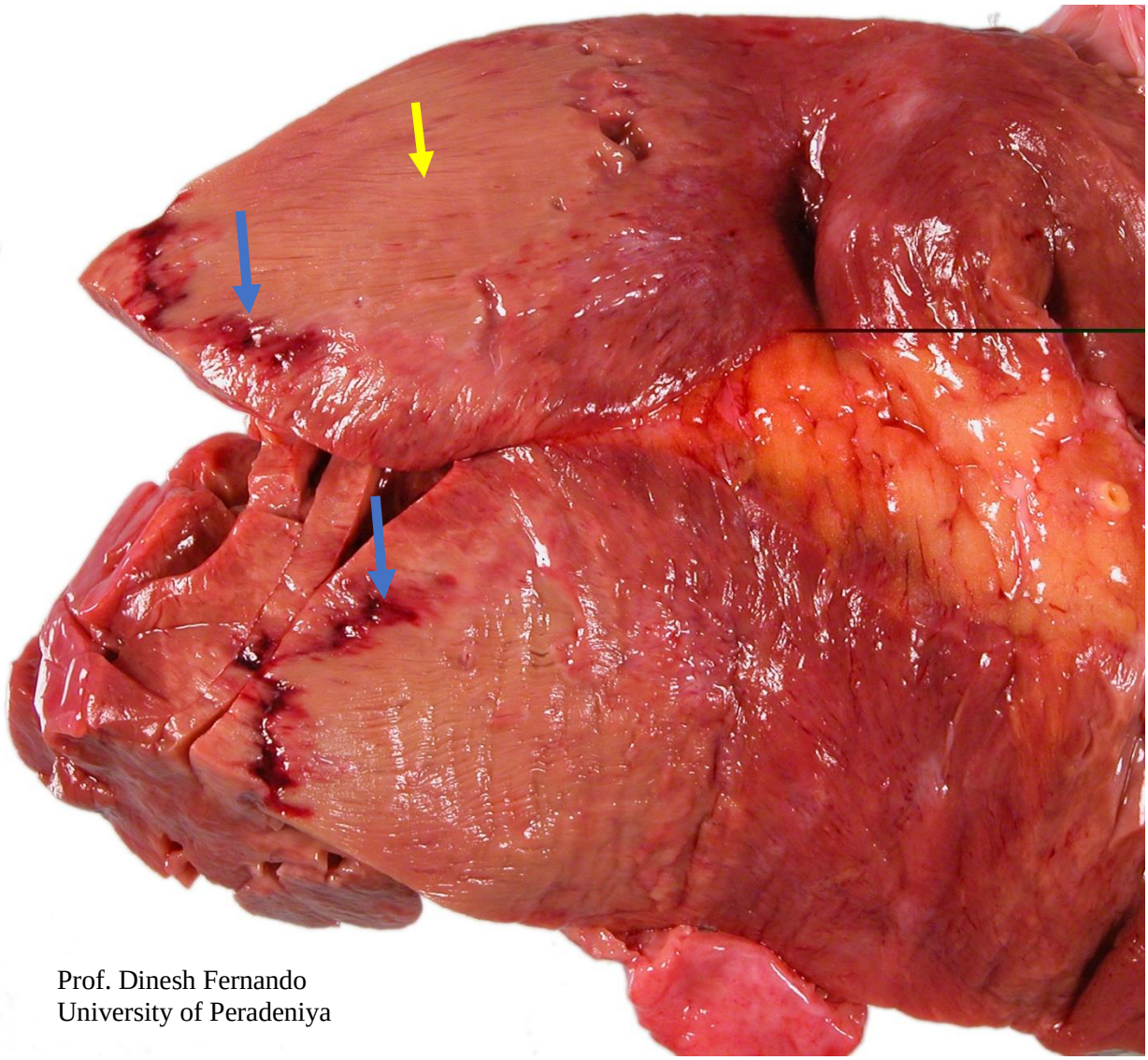
Figure 3: Myocardial infarction 24 to 48 hours

Note the fragmented myocytes. Karyorrhectic debris are seen as densely basophilic granules (yellow arrows).

Note the contraction bands in remaining parts of myocytes (blue arrows)

48 to 72 hours

Macroscopy: The infarcted area becomes pale with hyperaemic borders



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University of Peradeniya

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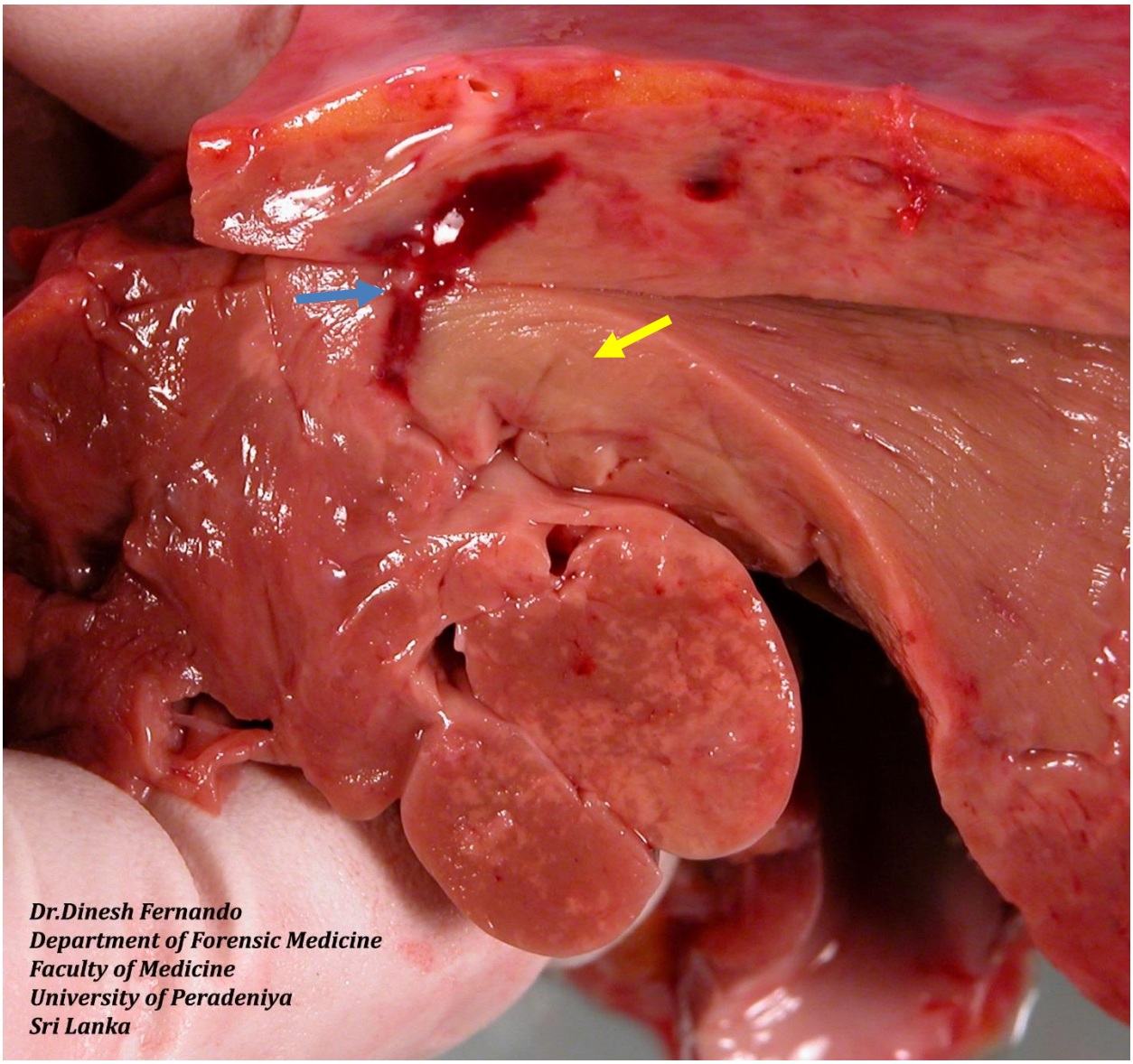


(b)

Figure 4(a,b): Myocardial Infarction 48-72 hours. Infarcted area is pale (yellow arrow) with hyperaemic borders (blue arrow)

Microscopy

The main feature is necrotic changes in cells, characterized by cytoplasmic degeneration and fragmentation. Prevalence of these necrotic changes decreases with increasing age of the infarct. Neutrophils start invading the degenerating myocardial fibres.

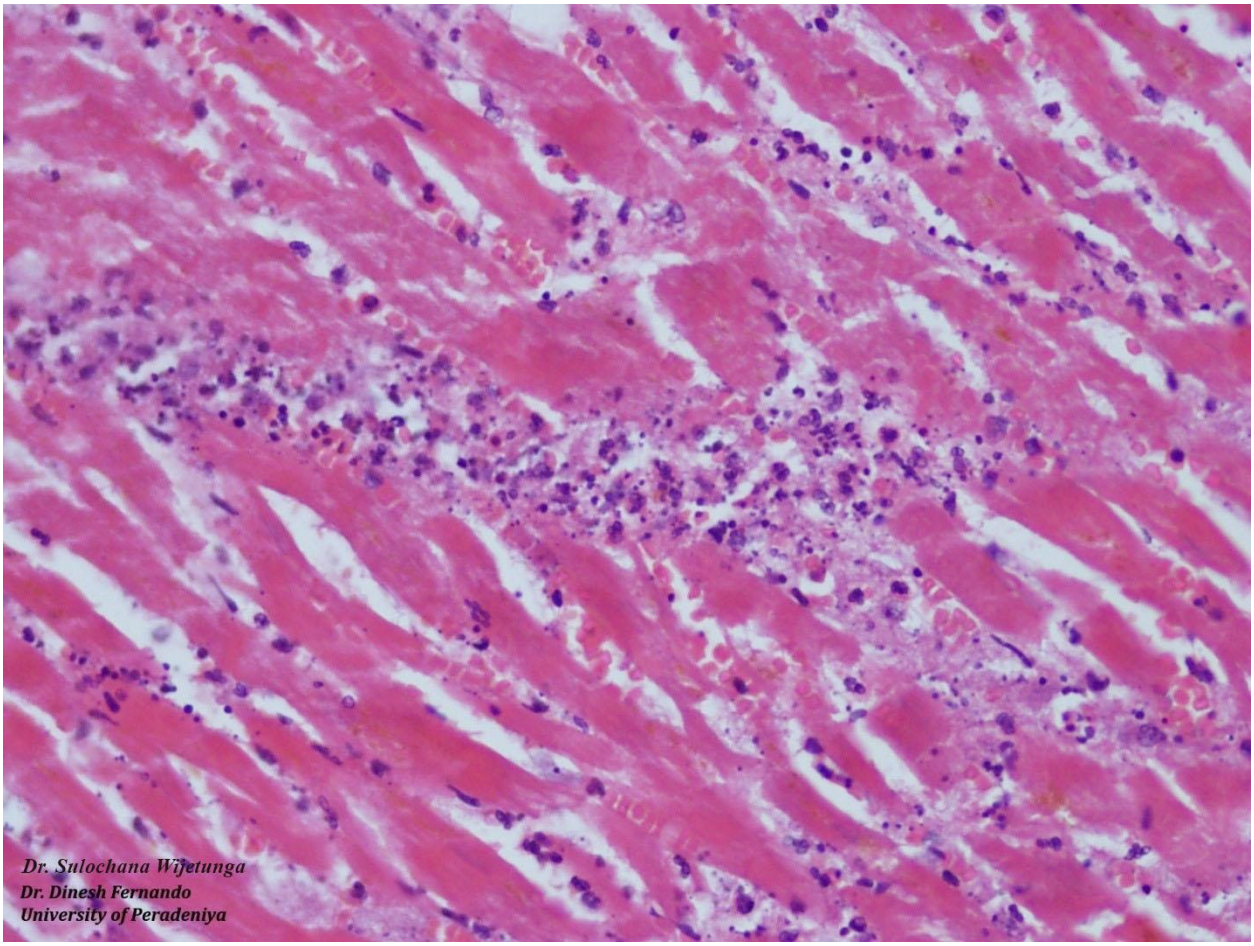


Figure 5: Myocardial infarction 48 to 72 hours. Fragmented myocytes are getting infiltrated by neutrophils. The neutrophil infiltration is not heavy. Karyorrhectic debris are still abundant.

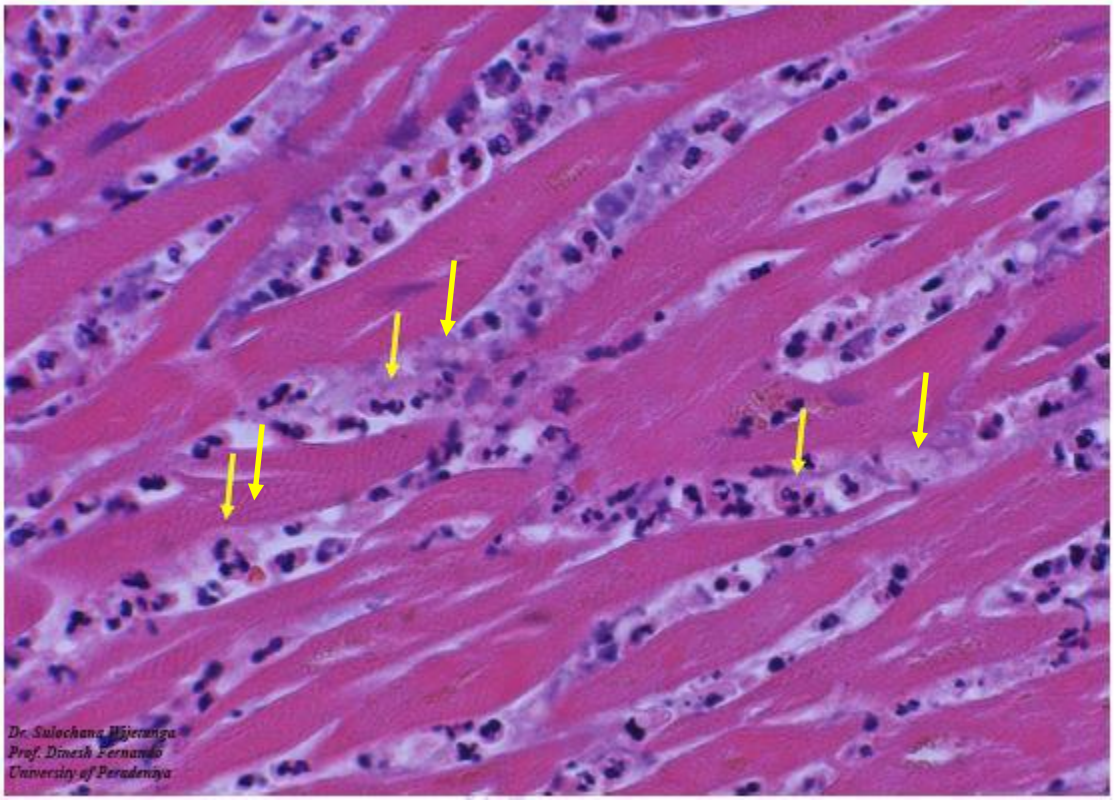
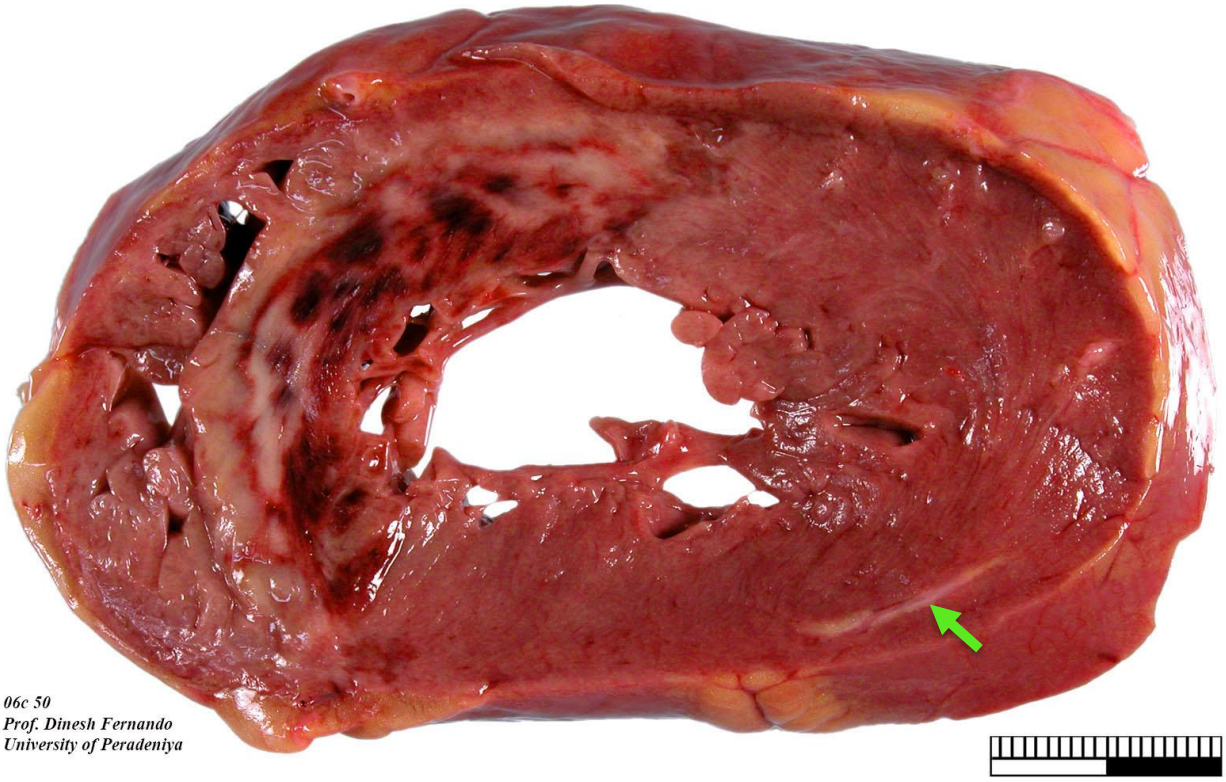


Figure 6: Myocardial infarction 48 to 72 hours (higher power). Dead myocytes bieng infiltrated by neutrophils (arrows). Neutrophils can be identified by their lobulated nuclei.

3 to 7 days

Macroscopy: The pale region gradually become yellow due to neutrophil action in dead myocytes and the hyperaemia in the borders become more intense giving rise to a 'tigroid' appearance.



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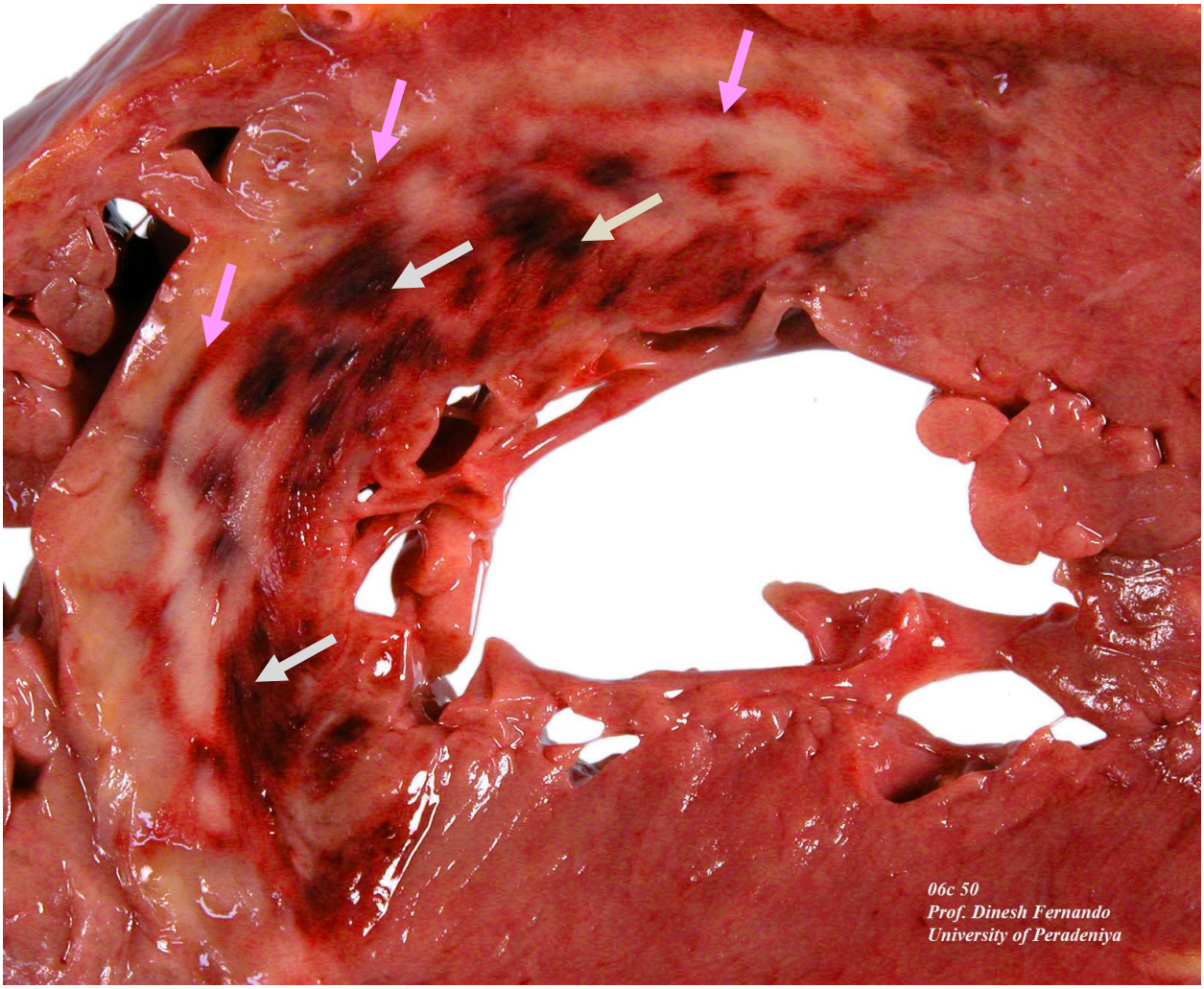

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Figure 7(a,b): The 'Tigroid' appearance caused by the streaks of red (due to dilated vascular channels and inter fibre haemorrhage) (pink arrows) between the yellow infarcted areas seen around 3-4 days gradually enlarges and by about the 10th day the infarct becomes maximally yellow with marked hyperaemia seen as reddish blotches on a yellow background transforming into a "leopards rosette" like appearance (grey arrows). Fibrosis is depicted by a green arrow.

Microscopy:

The main feature is neutrophils invading and digesting the necrotic myocardial fibres. There are basophilic neutrophils debris in the interstitium. Neutrophil infiltration persists until about 6th or 7th day and then gradually declines.

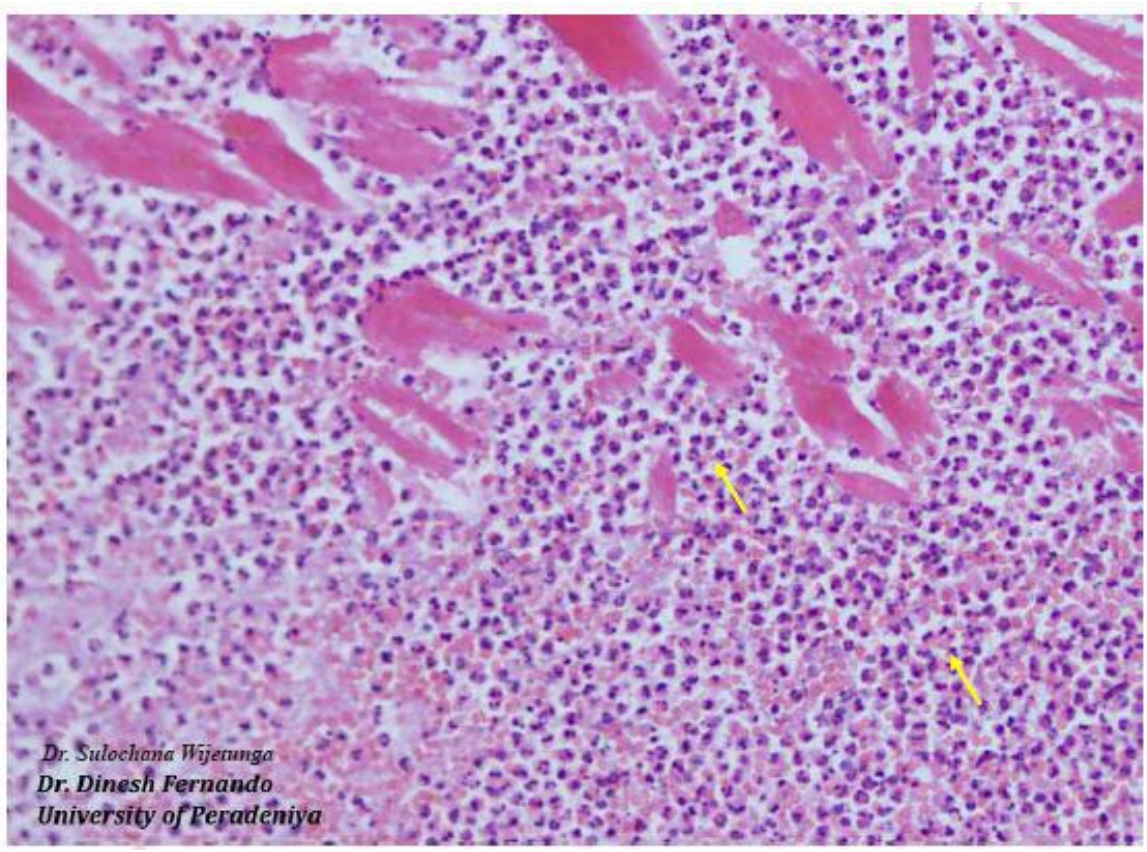


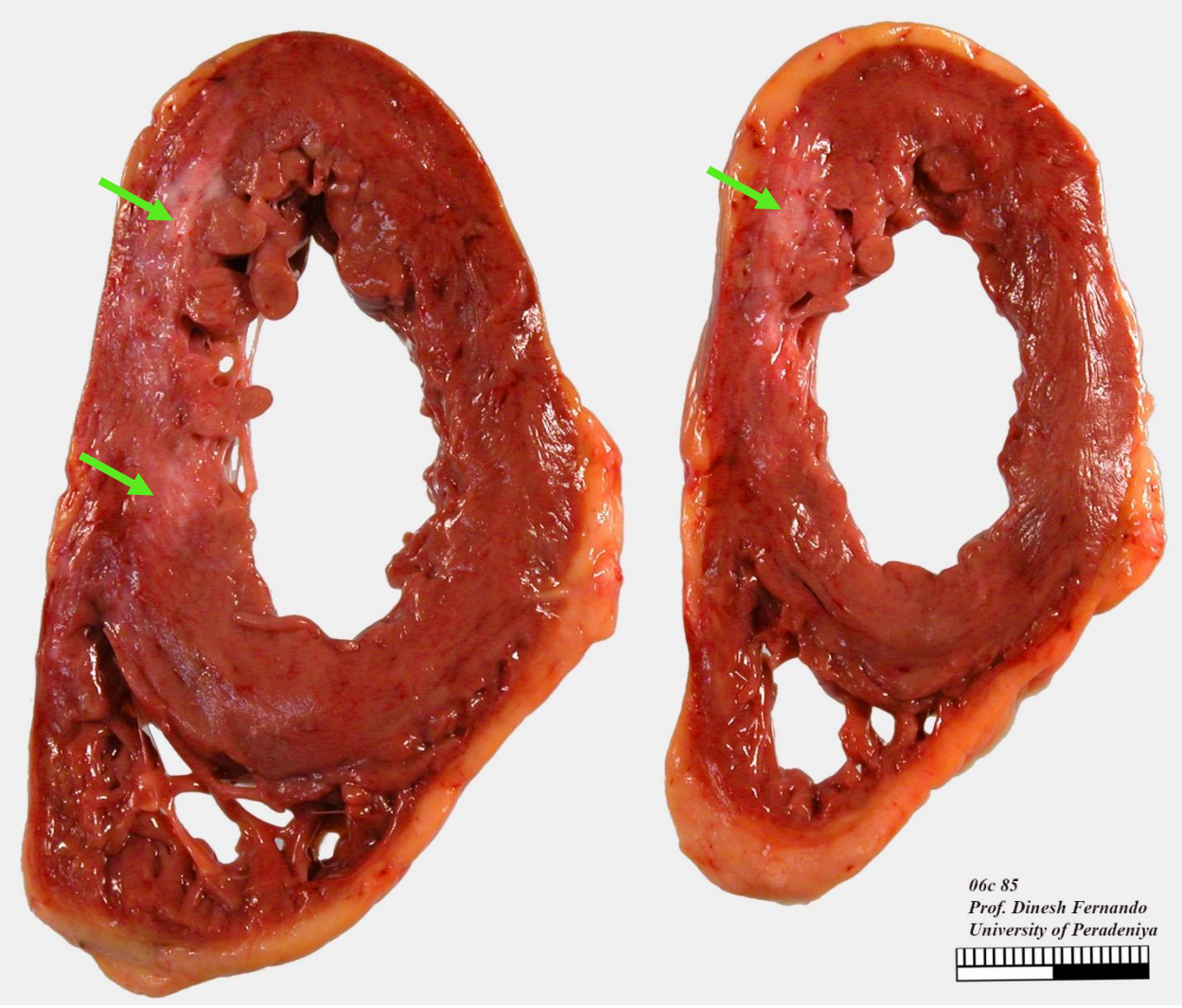
Figure 8: Myocardial infarction – about 3 to 5 days old

Myocardial fibres are more degenerated and the neutrophil infiltration is dense (arrows)

7th day onwards

Macroscopy:

With increasing collagen accumulation, the infarcted area gradually become whitish and ultimately the infarcted region is seen as a whitish scar – old infarct.



(a)

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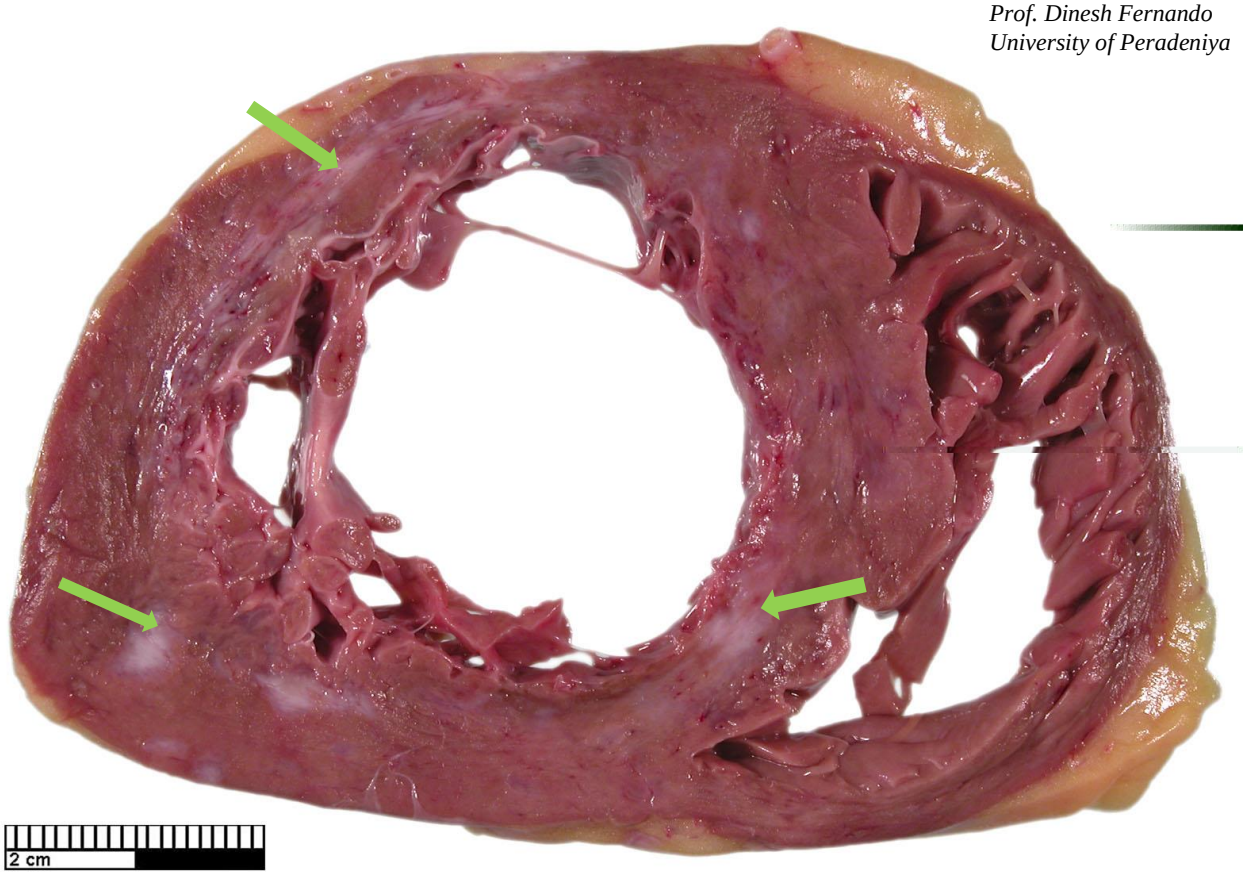
University of Peradeniya



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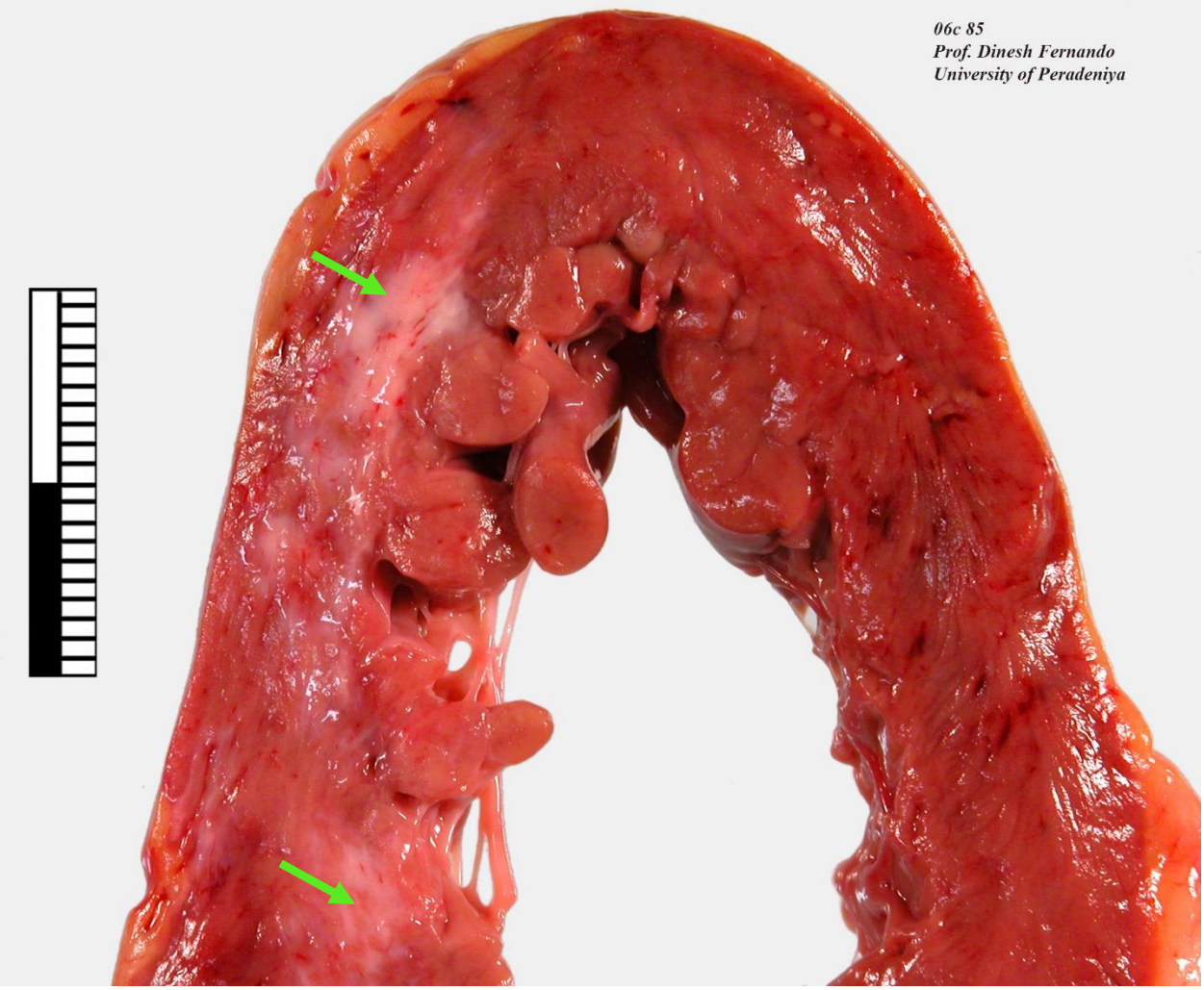

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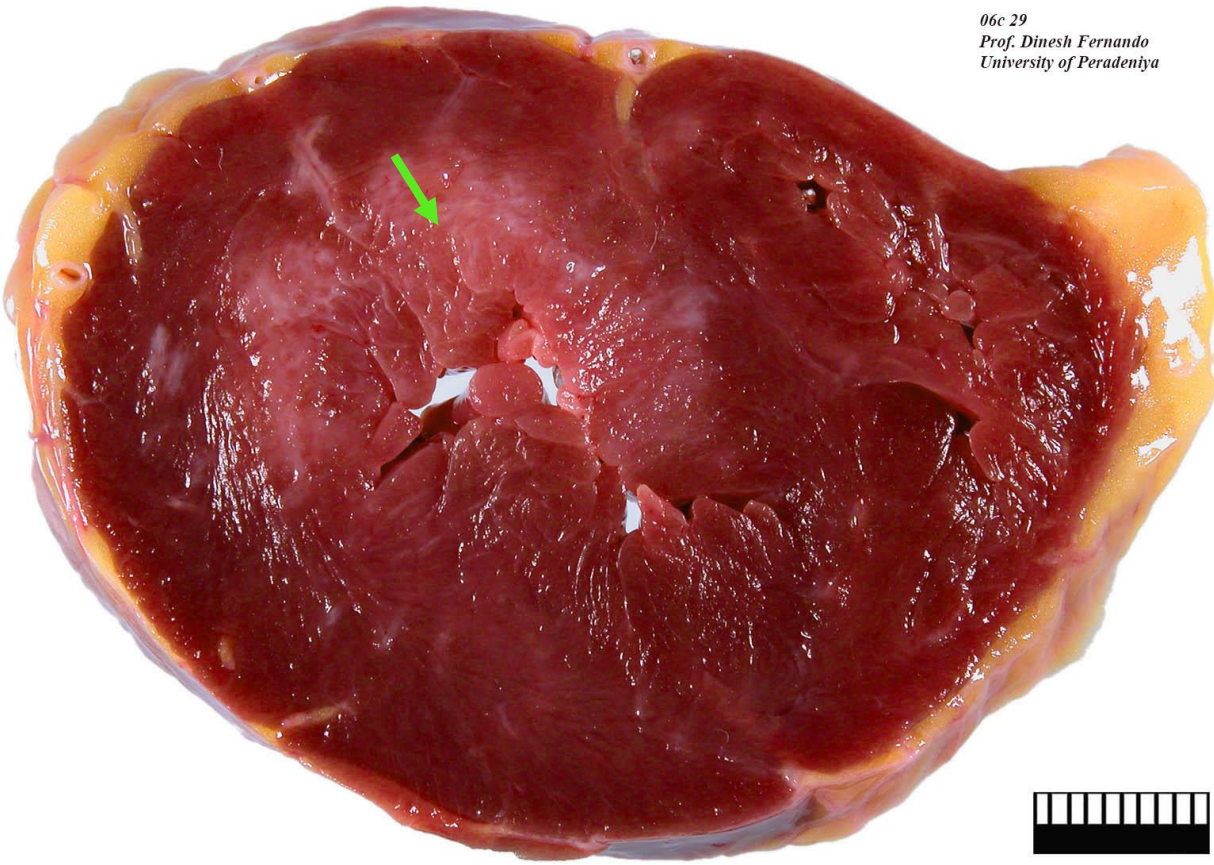

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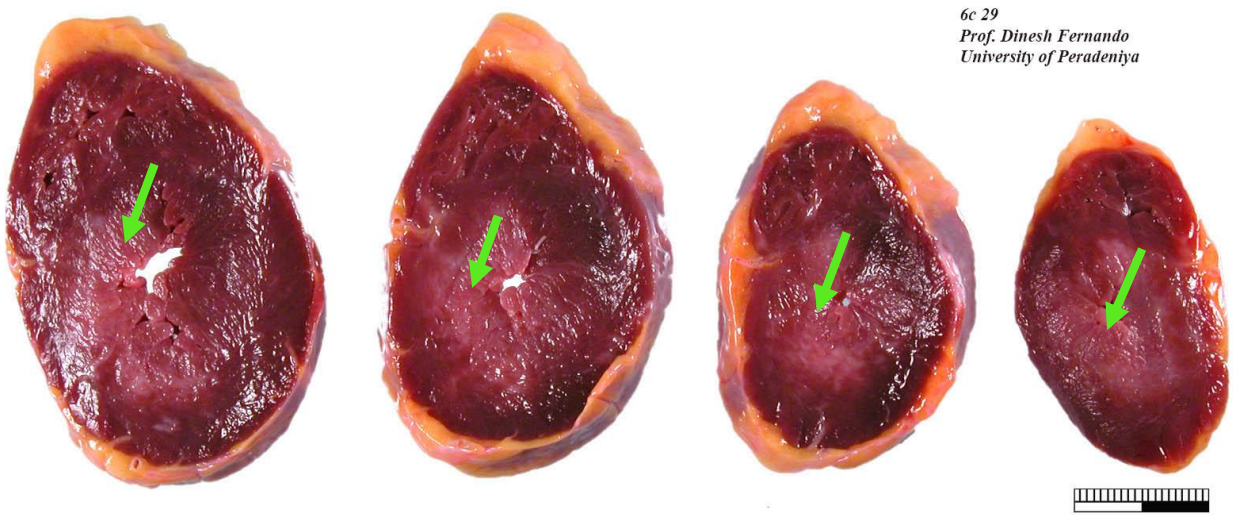


(e)

Figure 9 (a to e): Advanced Fibrosis over several weeks (green arrows)

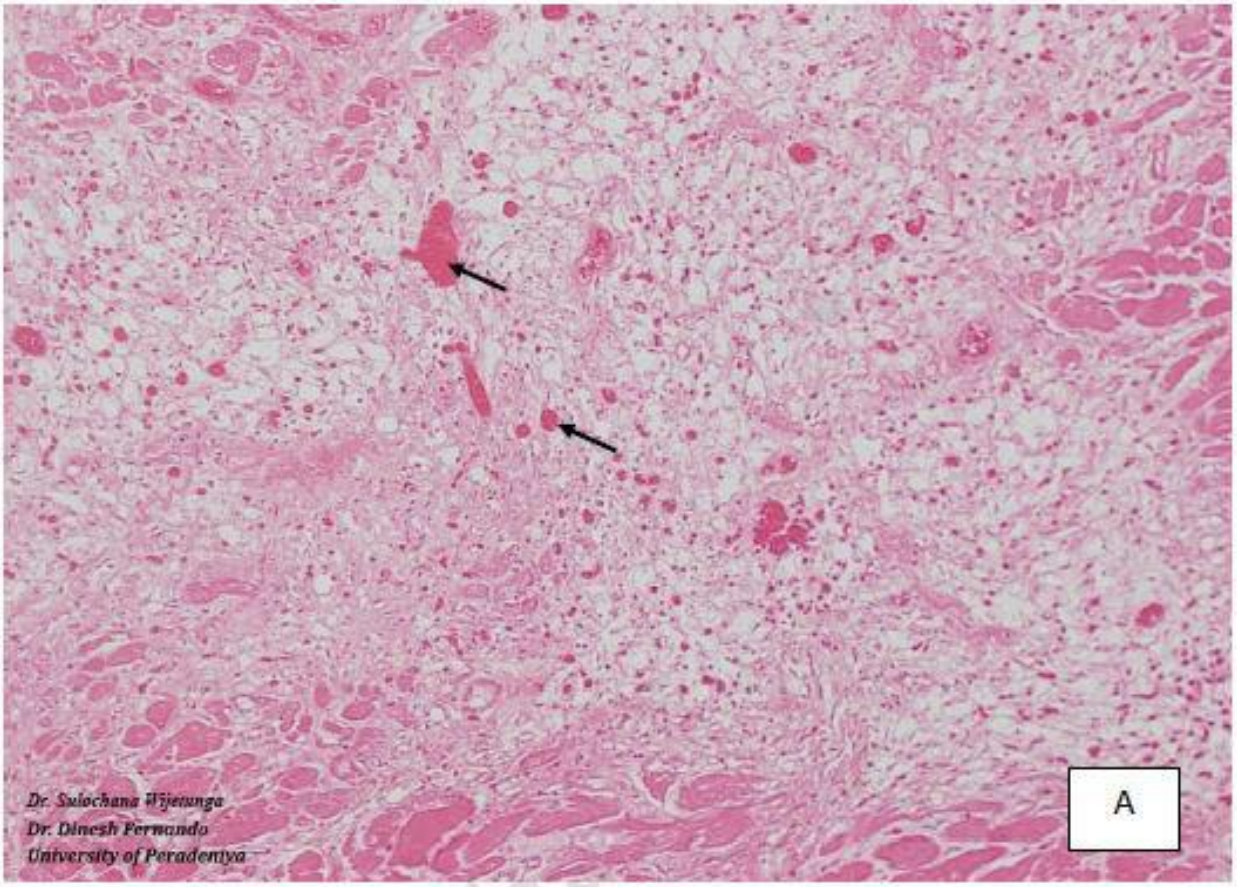
Microscopy:

In the second week macrophages and other mononuclear inflammatory cells predominate. The purpose of macrophages is scavenging and secretion of cytokines to lay the background for healing process.

By about the 10th day the necrotic changes become well developed with granulation tissue formation in the margin.

Granulation tissue formation starts from the 7th day onwards and become the main feature in the 3rd week. This begins from the edges of the infarct. Granulation tissue is characterized by numerous capillaries and proliferating fibroblasts. These fibroblasts synthesize and lay down collagen. This is the repair process of a myocardial infarction, i.e since myocardial cells are permanent cells repair of an infarction is done solely by fibrous tissue formation (scar).

With advancing age of the infarct there is progressive accumulation of collagen with decreasing vascular and fibroblast density. This takes place over several weeks and ultimately the infarcted area gets replaced by an acellular collagen scar. Although the time taken to produce this scar is quite variable, roughly it takes about 7 weeks. Once the scar tissue is formed the infarction cannot be dated any longer.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Dr. Dinesh Fernando
University of Peradeniya
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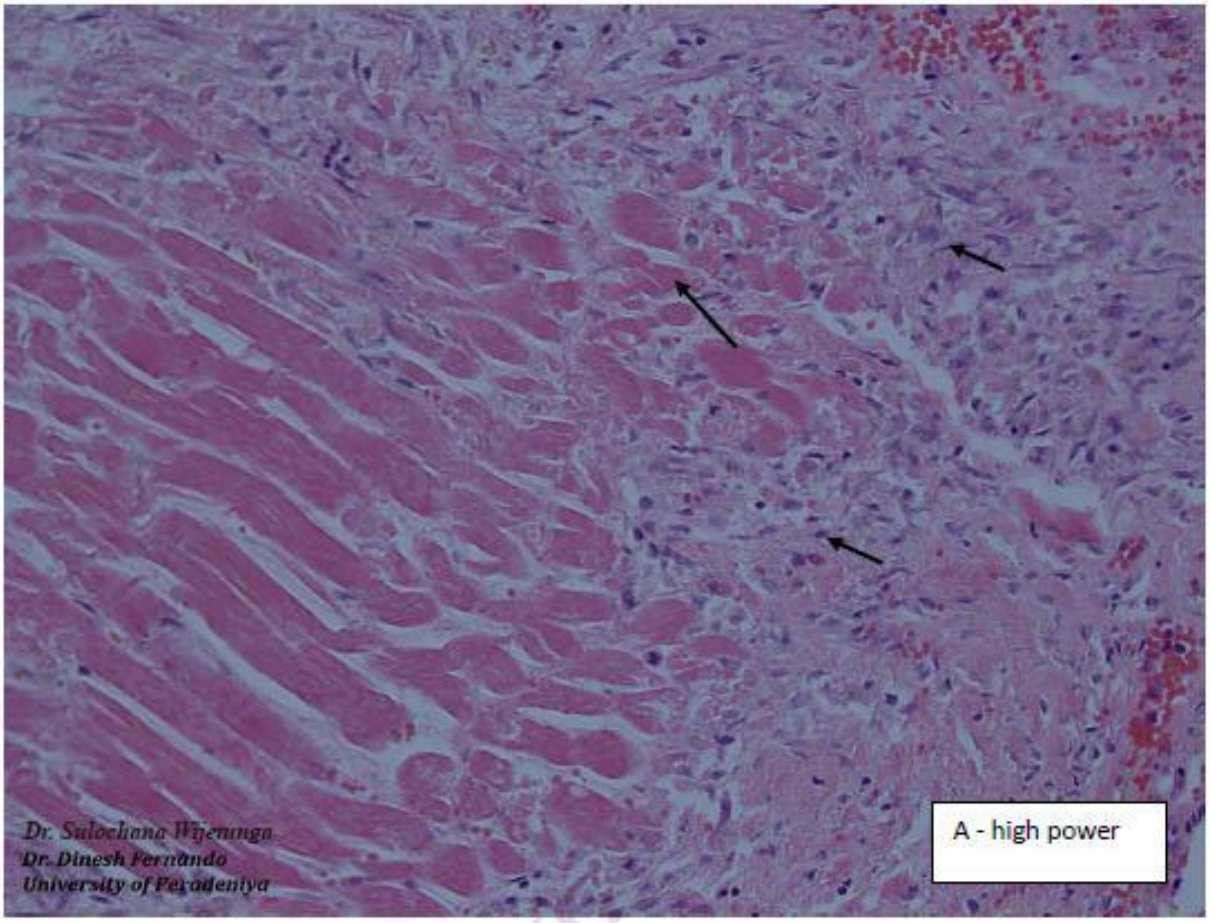
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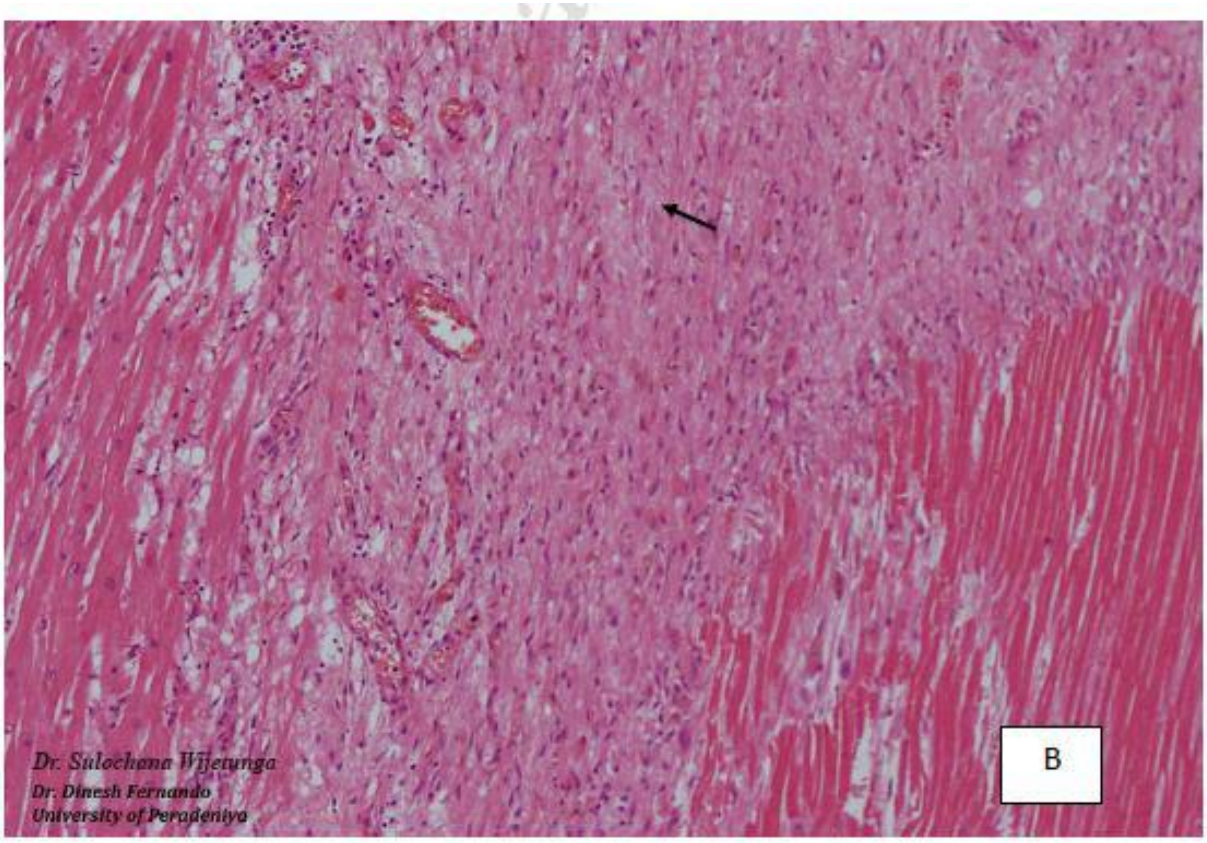
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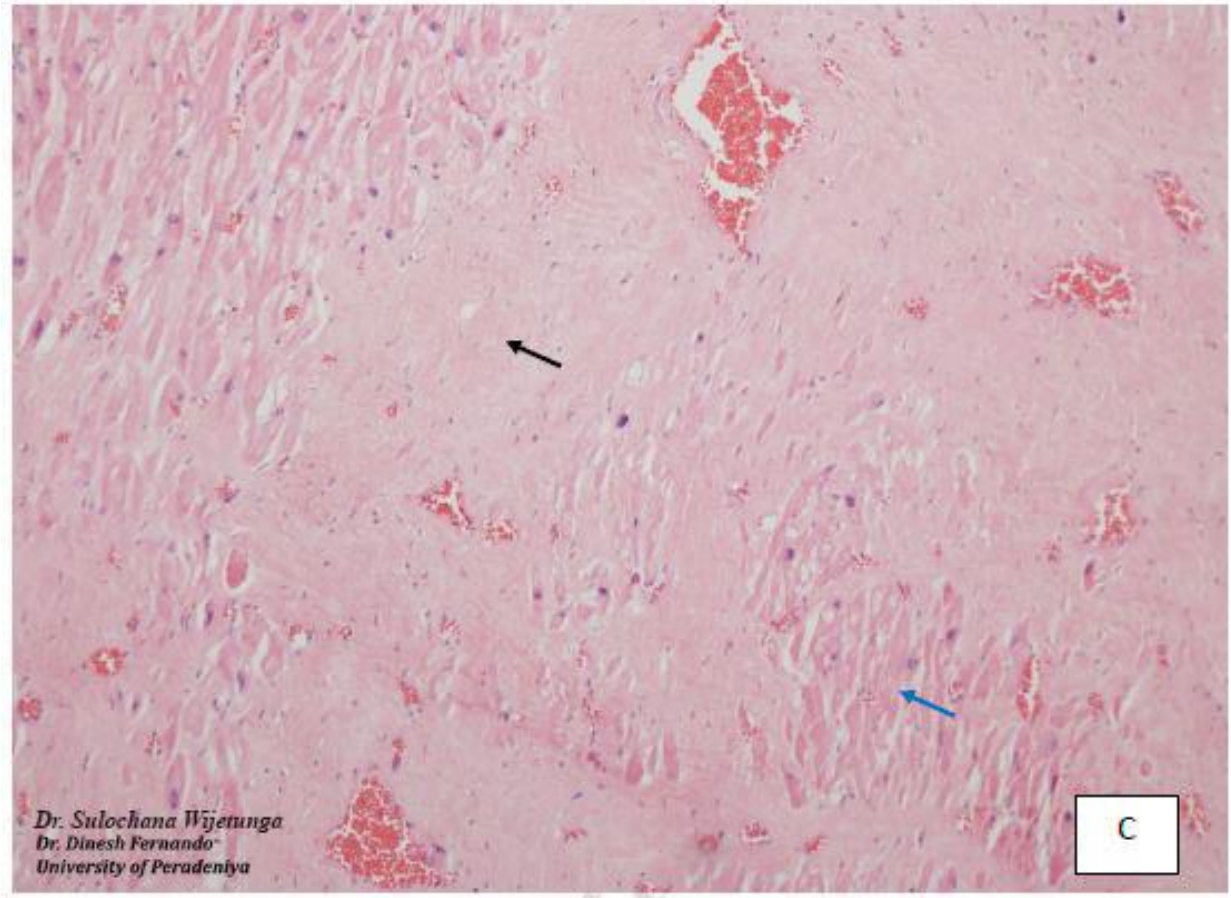

Figure 10 (A, B & C): Scar tissue formation in a myocardial infarction (repaired by organization)

A) Early phase – granulation tissue formation. Note the high vascularity. Higher power view show higher cellularity due to fibroblasts (arrows). (fibroblast synthesize and lay down collagen)

B) Intermediate - Vascularity and cellularity have decreased and more collagen has been laid down. Collagen is seen as eosinophilic acellular material (arrow).

C) Advanced scar – granulation tissue is replaced by masses of acellular collagen (black arrow). Residual viable myocardial fibres are also present (blue arrow)

PULMONARY EMBOLISM

Pulmonary embolism (PE) is the blockage of the pulmonary vasculature by an embolus. A thrombus commences as a platelet aggregate. Then fibrin and red cells form a mesh until the lumen of the vein wall is occluded. This coralline thrombus then progresses as a propagated loose red fibrin clot containing many red cells. This is likely to extend up to the next large venous branch and it is possible for the clot to break off and embolize to the lung as a pulmonary embolus. More than 95% of all pulmonary emboli arise from thrombi within the large deep veins of the lower legs, typically originating in the popliteal vein and larger veins above it. Large or long thrombi can embolize to the main pulmonary artery trunk, the bifurcation or left or right pulmonary artery and produce massive pulmonary embolism. If total occlusion of the pulmonary arteries occurs, typically, from a large saddle embolus, acute cor pulmonale, shock or death due to acute right ventricular failure results.

The vast majority (60% to 80%) of emboli are clinically silent, and no more than one third is diagnosed before death. The minority (5%) are symptomatic with a sudden onset of unexplained dyspnoea which is the most common, and often, the only symptom of it. On examination there will be tachypnoea, localized pleural rub, and course crackles.

Embolization of medium sized pulmonary arteries by smaller thrombi usually do not produce any significant effect in otherwise healthy individuals due to compensation by bronchial arterial perfusion; however, they can produce haemorrhagic infarctions in those with already congested and compromised pulmonary perfusion, such as in congestive cardiac failure and mitral stenosis. Multiple recurrent pulmonary emboli cause increased breathlessness, often, over weeks or months due to recurrent emboli. About three fourths of all infarcts affect the lower lobes and more than half are multiple. Characteristically, they are wedge-shaped, with their base at the pleural surface and the apex pointing toward the hilus of the lung. Pleuritic chest pain and haemoptysis are present only when infarction has occurred. Since a saddle embolus causes a sudden death there is no time for morphologic alterations in the lung. However, when smaller emboli are impacted in medium-sized and small pulmonary arteries, alveolar haemorrhage may occur as a result of ischemic damage to the endothelial cells.

Risk factors for pulmonary embolism are obesity, immobility, pregnancy, oestrogen therapy, disorders of hypercoagulability, diseases like polycythaemia, inflammatory bowel disease, disseminated cancer, surgical procedures or trauma including multiple burns or fractures.
Autopsy data on the incidence of pulmonary embolism varies; ranging from 1% in the general hospitalized population, to 30% in persons dying after a predisposing factor of the patient to venous thrombosis in the legs. In a study done by Knight, more than three-quarters of the victims had predisposing factors but the remaining 20% were ambulant and apparently healthy.

Pulmonary infarcts typically are haemorrhagic, and appear as raised, red-blue areas in the early stages. The red cells begin to lyse within 48 hours, and the infarct becomes pale, eventually becoming red-brown as hemosiderin is produced. In time, fibrous replacement begins at the margins and converts the infarct into a scar. An ante-mortem embolus (especially if several days old) is firm. Although it may appear to be a cast of the large vessel in which it is impacted, it may often be unravelled to form a long length, that obviously originated in a leg vein. Post-mortem clots may be adherent to the ante-mortem embolus, and sometimes forms a sheath around it.

History

A 59-year-old male was treated in hospital for 12 days for a bowel obstruction and subsequent pneumonia. While going for a shower in hospital he fell and was declared deceased.

Internal examination

Cardiovascular System: The heart weighed 384 grams. Bilateral pulmonary arteries contained coiled thrombi which were approximately 2 mm in diameter. These were present in the main pulmonary arteries and their branches.

Respiratory system: The right and left lungs weighed 680 grams and 530 grams respectively. Sectioning of the lung revealed thrombi in the pulmonary arteries. The lower lobes of both lungs were solid to touch and friable.

Musculoskeletal System: Dissection of the left calf muscle revealed thrombi in the deep veins.

Prof. Dinesh Fernando
Department of Forensic Medicine
Faculty of Medicine
University of Peradeniya
Sri Lanka

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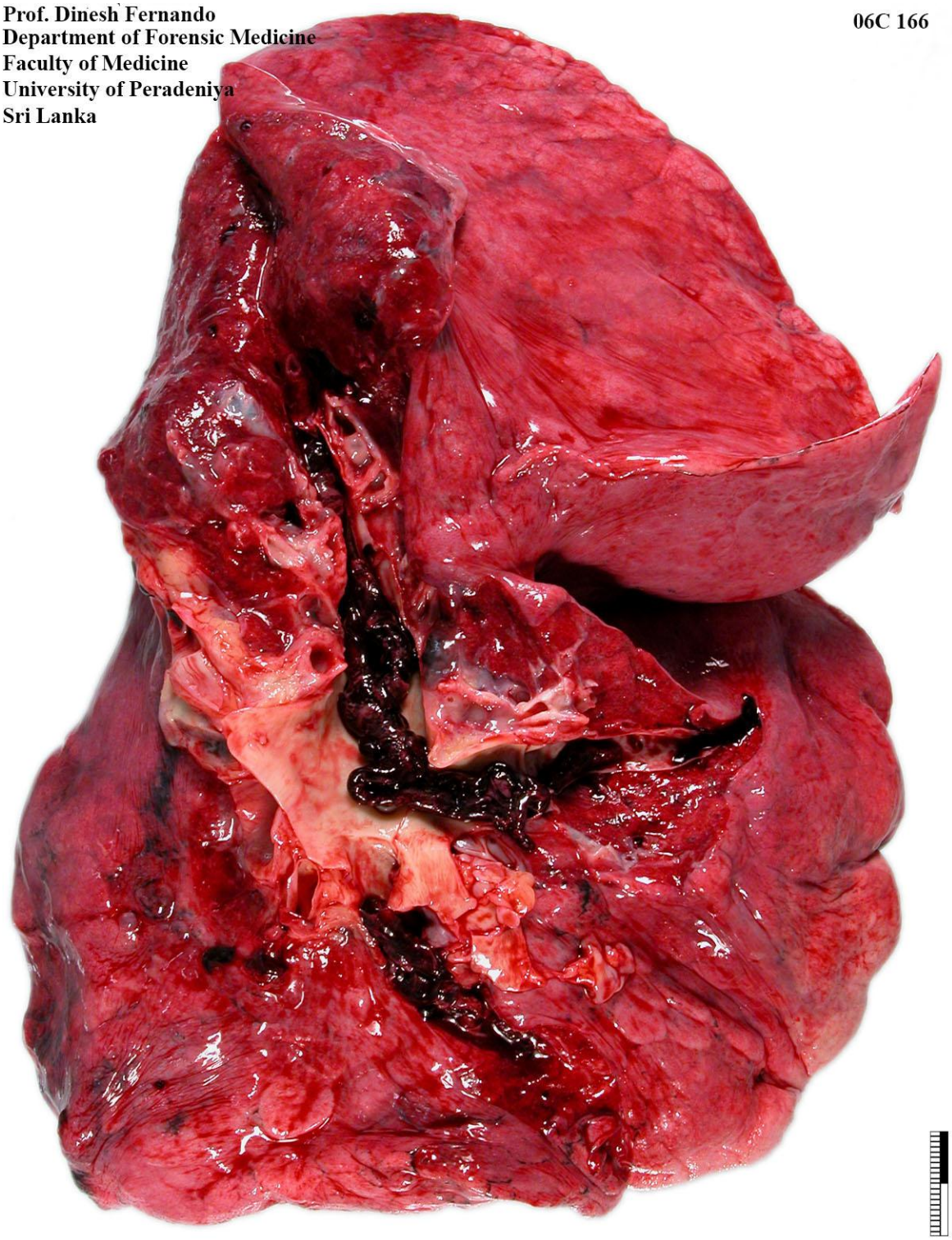

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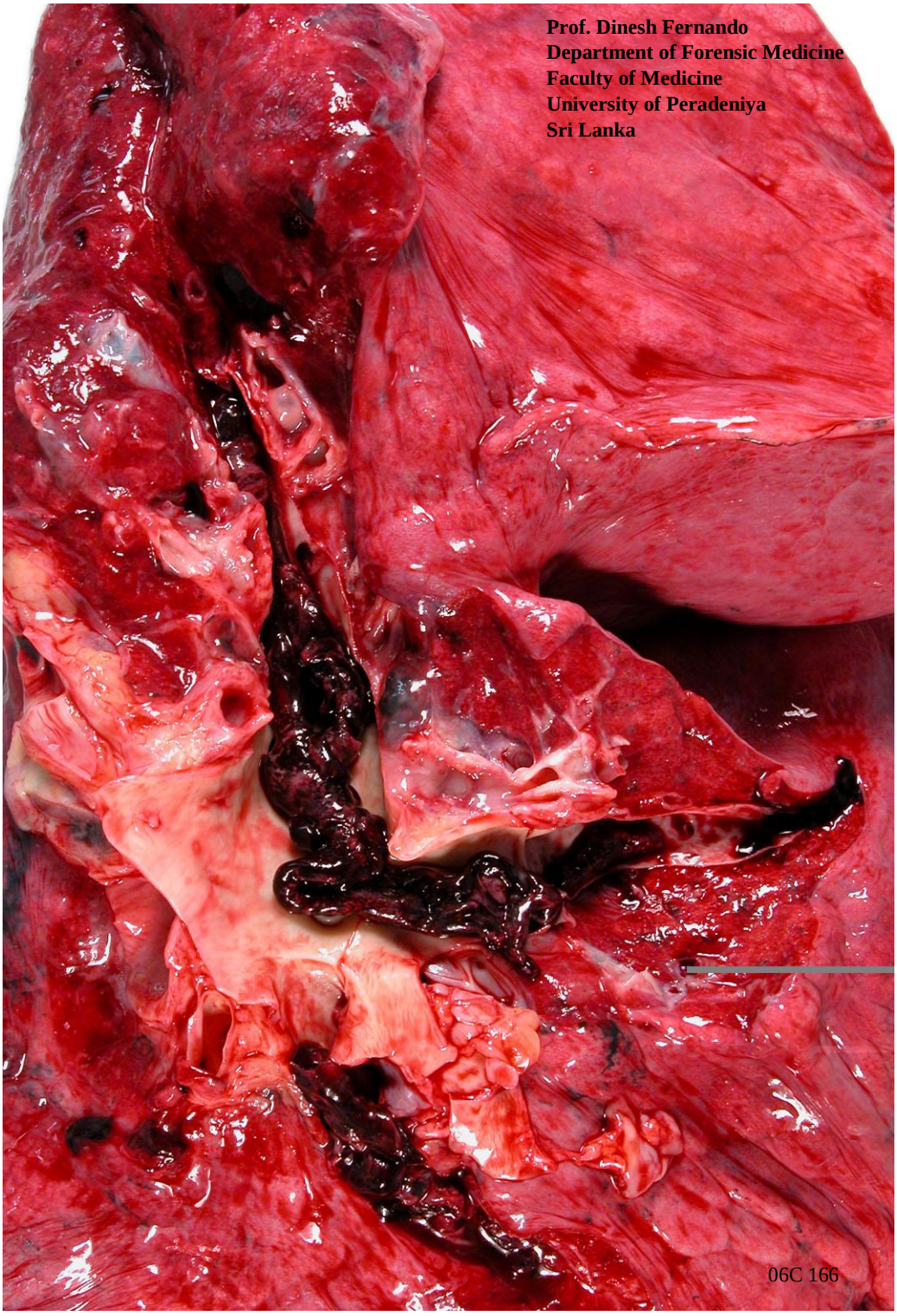
Prof. Dinesh Fernando
Department of Forensic Medicine
Faculty of Medicine
University of Peradeniya
Sri Lanka
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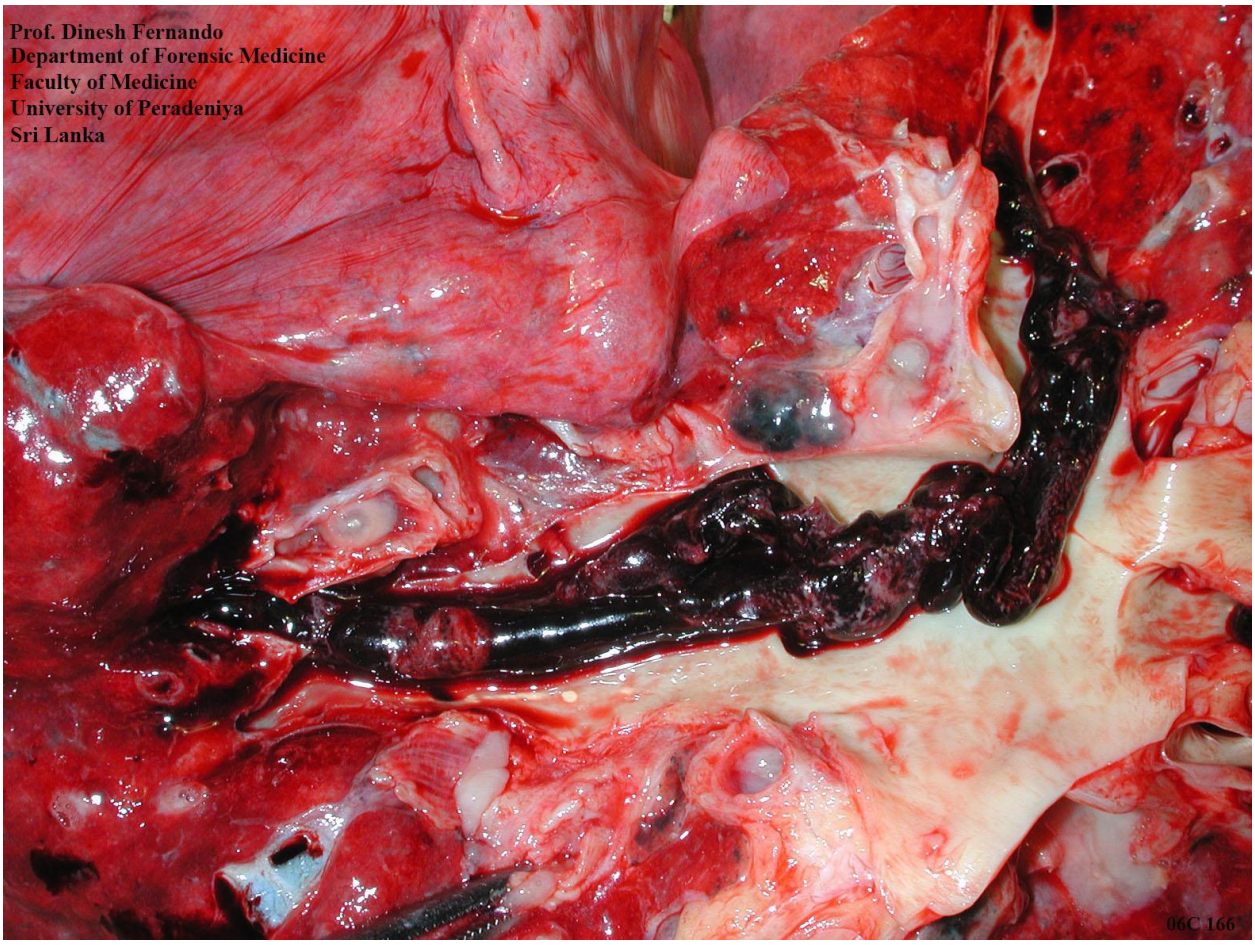
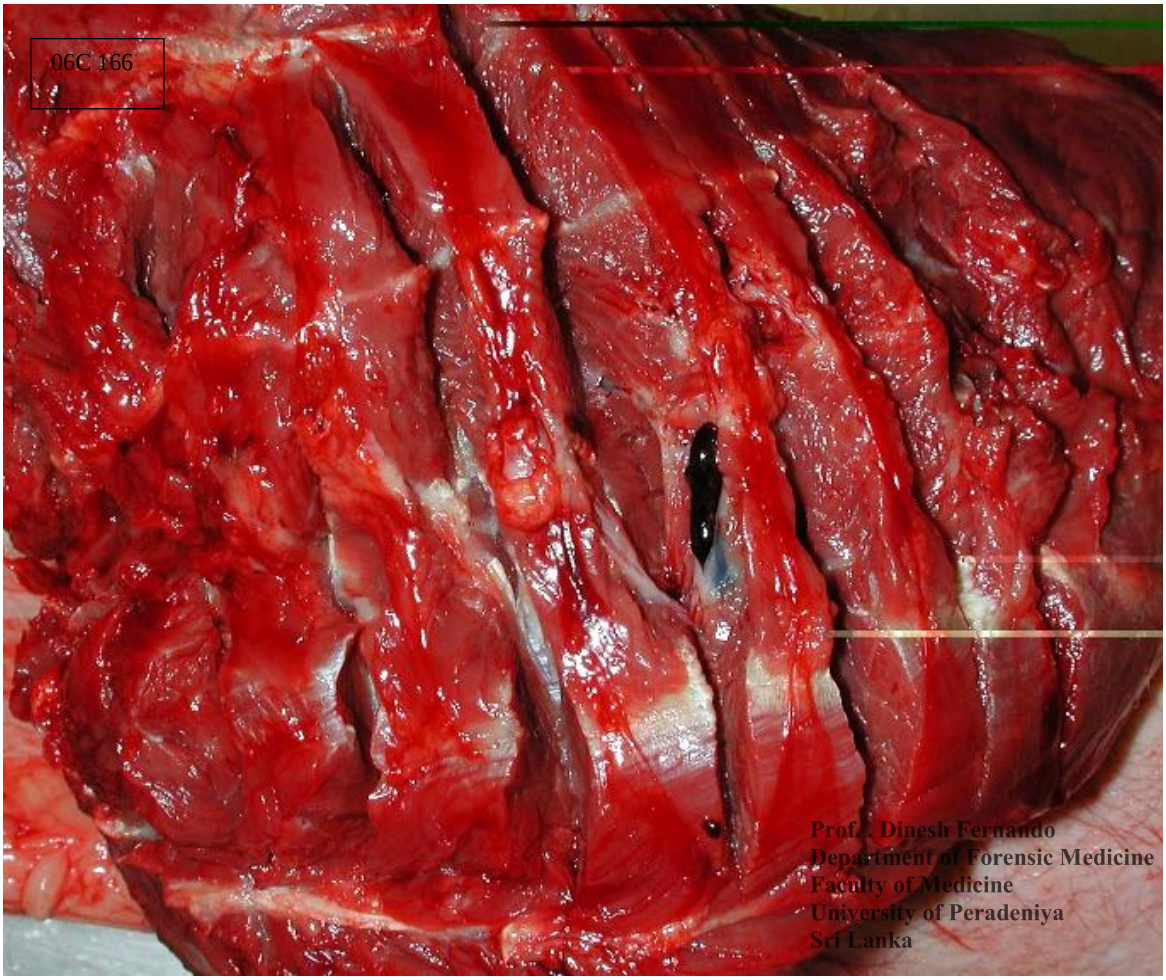


(c)

Figure 1(a, b, & c): Thromboembolus in the main pulmonary artery


06C 166
Prof. Dinesh Fernando
Department of Forensic Medicine
Faculty of Medicine
University of Peradeniya
Sri Lanka

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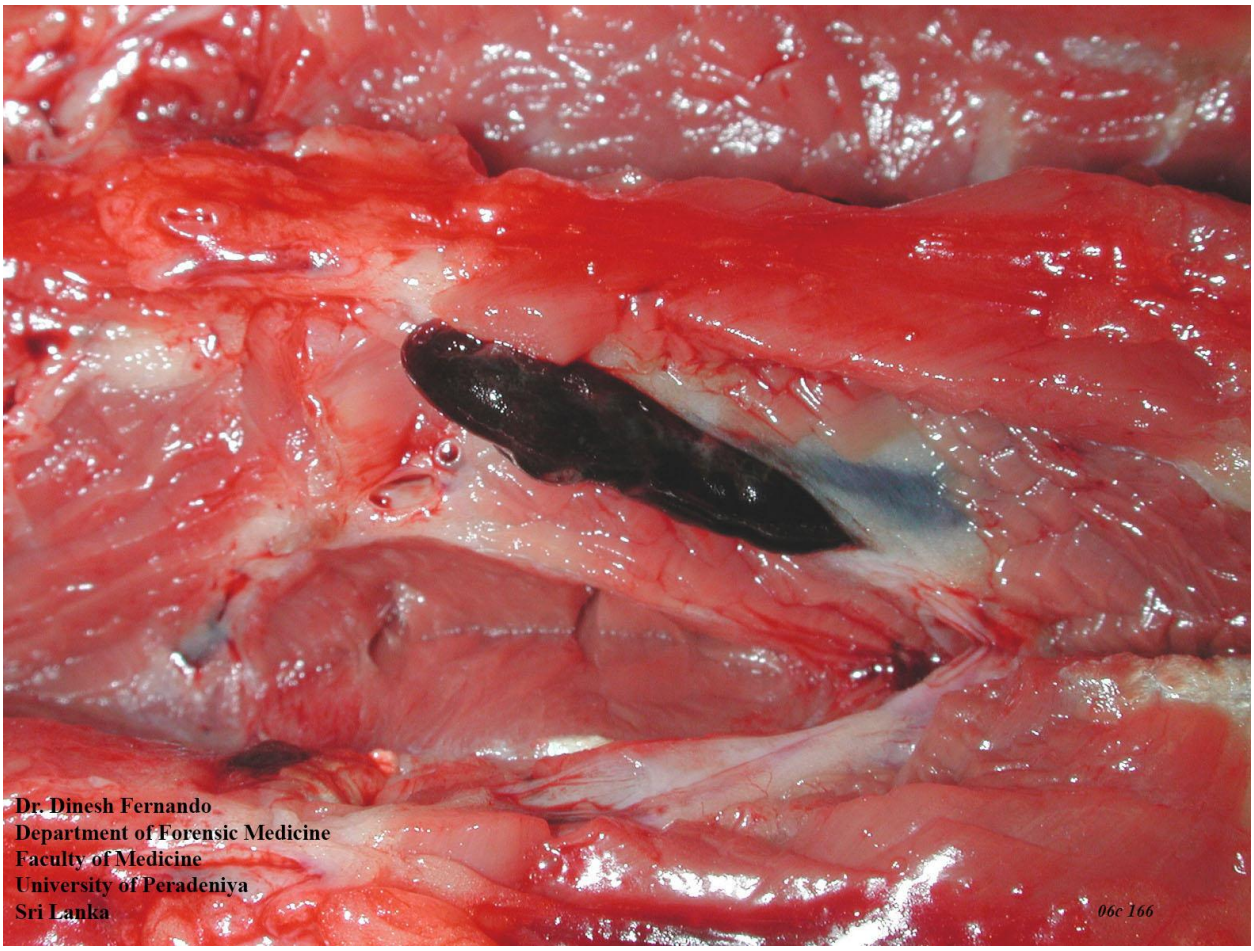

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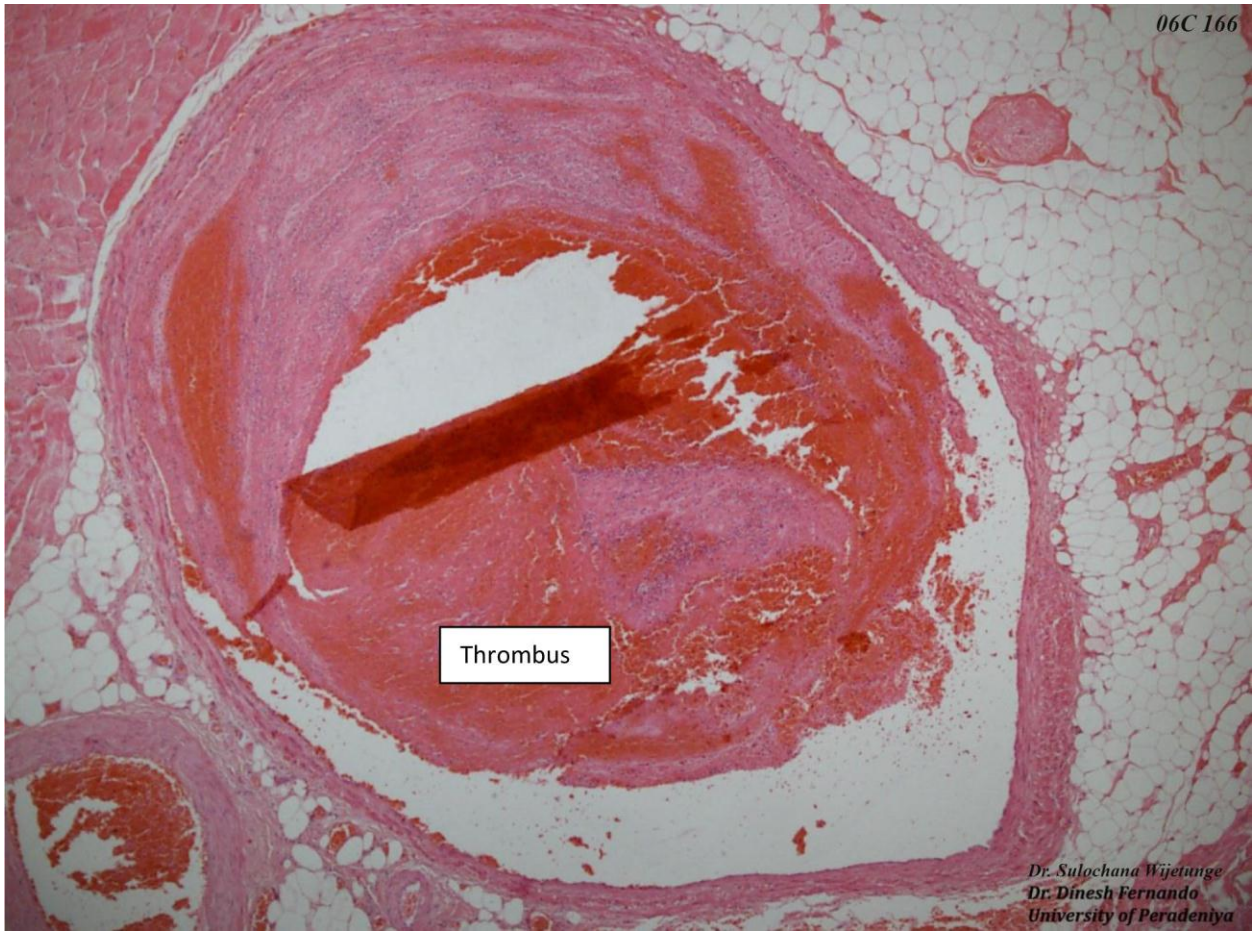
Figure 2(a & b): Calf muscle sliced to show thrombi in deep veins

Microscopic Examination



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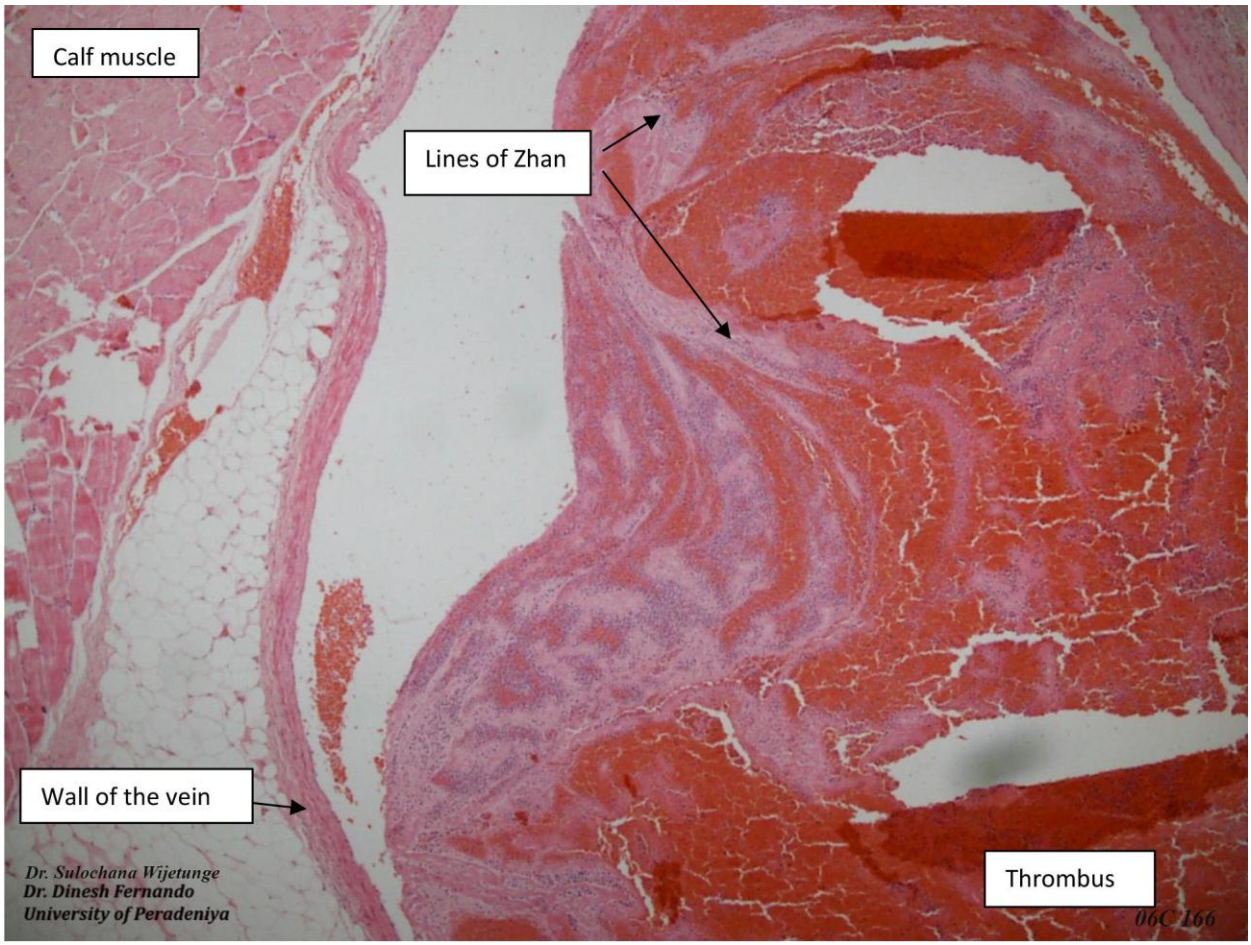

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Figure 3(a & b): Thrombosis of deep calf veins

- Lines of Zhan, pale pink areas composed of fibrin and platelets (bright pink areas are predominantly composed of red blood cells) are clearly seen in both thrombi. Lines of Zhan are useful to differentiate an ante mortem clot from a post mortem clot.

- These deep vein thrombi can get detached and embolize to the pulmonary circulation. Large or long thrombi can embolize to the main pulmonary artery trunk, the bifurcation or left or right pulmonary artery and produce massive pulmonary embolism., which leads to instantaneous death due to acute right ventricular failure.

- Embolization of medium sized pulmonary arteries by smaller thrombi usually do not produce any significant effect in otherwise healthy individuals due to compensation by bronchial arterial perfusion; however, they can produce haemorrhagic infarctions in those with already congested and compromised pulmonary perfusion, such as in congestive cardiac failure and mitral stenosis.

Cause of death

Pulmonary embolism due to deep vein thrombosis due to prolonged immobilization resulting from bowel obstruction and aspiration pneumonia

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CORONARY ARTERY DISSECTION

Coronary artery dissection can be spontaneous, traumatic, or iatrogenic. In most cases, a predisposing arterial disease is identified and up to 20% are idiopathic. Spontaneous coronary artery dissection is a rare condition which usually presents with sudden death. Around 80% of spontaneous dissections occur in females, particularly in the peri-partum period. Three quarters of the cases involve the left anterior descending coronary artery. The artery wall has three layers; namely tunica intima, tunica media -which is the muscle layer- and the tunica externa. Coronary arterial dissection may be primary, if a tear occurs at some point in the artery's lining with the formation of an intimal flap, or, it may be secondary to extension of an aortic root dissection.

Non-atherosclerotic spontaneous coronary artery dissection arises from a tear in the tunica intima of the blood vessel, with blood entering and separating the layers of the arterial wall. The increasing pressure within the false lumen, by the enlarging haematoma, causes compression of the true coronary lumen. This restricts the coronary blood flow, eventually leading to coronary insufficiency, ischemia and death.

Aortic dissection occurs in the laminar planes of the media to form a blood-filled channel within the aortic wall. Almost 90% of aortic dissections are seen in men aged 40 to 60 with antecedent hypertension. It may rarely occur in younger patients with connective tissue abnormalities that affect the aorta. These patients present with sudden onset of excruciating tearing or stabbing pain, usually beginning in the anterior chest, radiating to the back between the scapulae, and moving downward as the dissection progresses. The most common cause of death is rupture of the dissection into the pericardial, pleural, or peritoneal cavity.

In most dissections, the intimal tear marking the point of origin is found in the ascending aorta within 10 cm of the aortic valve. Such tears usually are transverse or oblique in orientation and 1 to 5 cm long, with sharp, jagged edges. The dissection plane can extend retrograde toward the heart or distally, occasionally as far as the iliac and femoral arteries, and usually lies between the middle and outer thirds of the media. External rupture causes massive haemorrhage, or results in cardiac tamponade, if it occurs into the pericardial sac.

In some (fortunate) instances, the dissecting haematoma re-enters the lumen of the aorta through a second distal intimal tear, creating a second vascular channel within the media (so-called double-barrelled aorta). Over time, such false channels become endothelialized to give rise to chronic dissections. In most instances, no specific underlying causal pathology is identified in the aortic wall.

History

A 52-year-old female with no medical history was found dead in bed, lying on her left-hand side.

External Examination

Rigor mortis was absent. Hypostasis was present over the left side of the body anteriorly, laterally and posteriorly with sparing of the left forearm and left buttock. The right side was free of livor. Greenish discolouration of the right iliac fossa and peeling of skin on the left medial thigh were present.

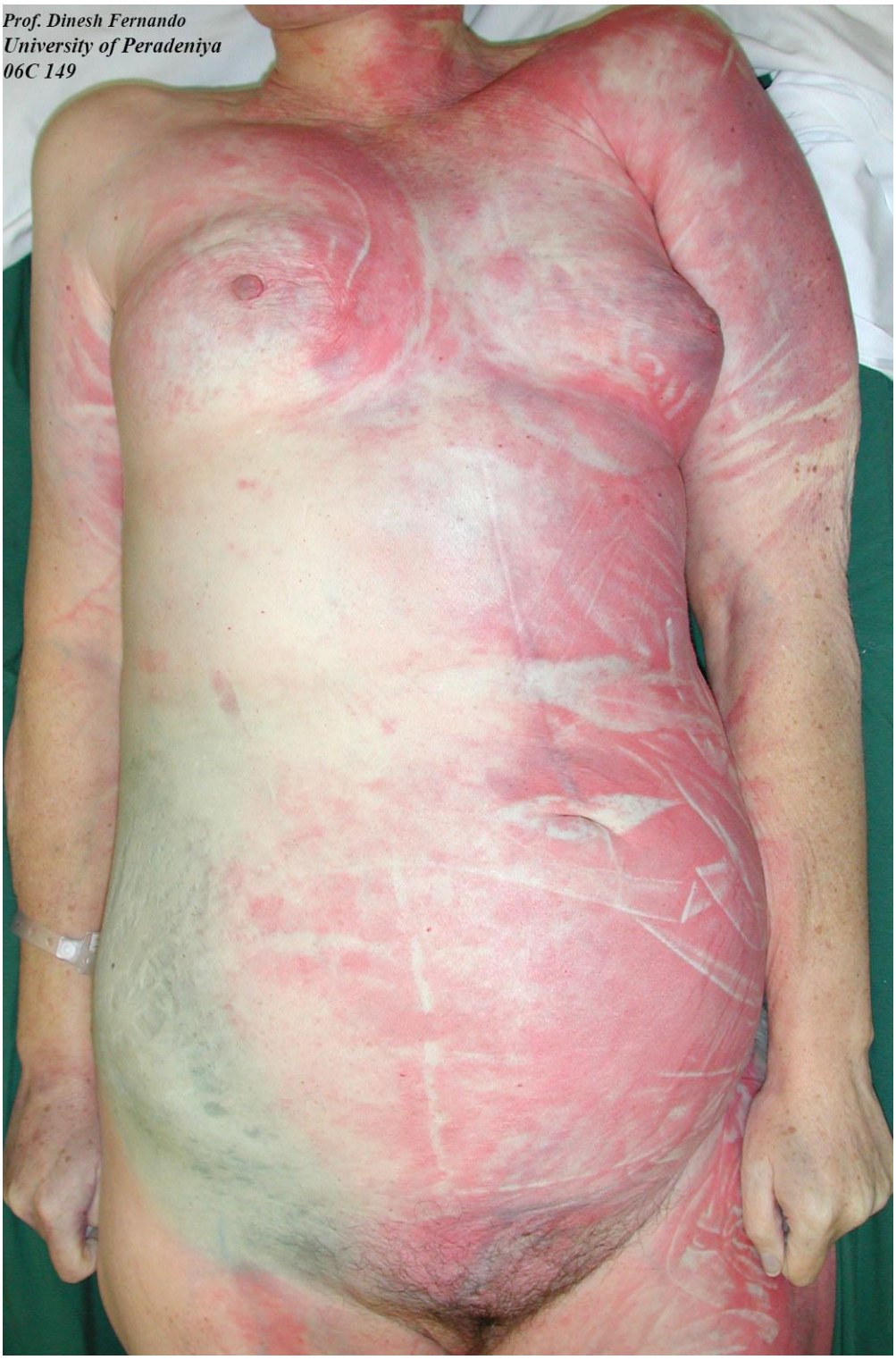


Figure 1:Hypostatis on the left side of the body. Note sparing of left forearm due to pressure and fold marks of sheet on abdomen

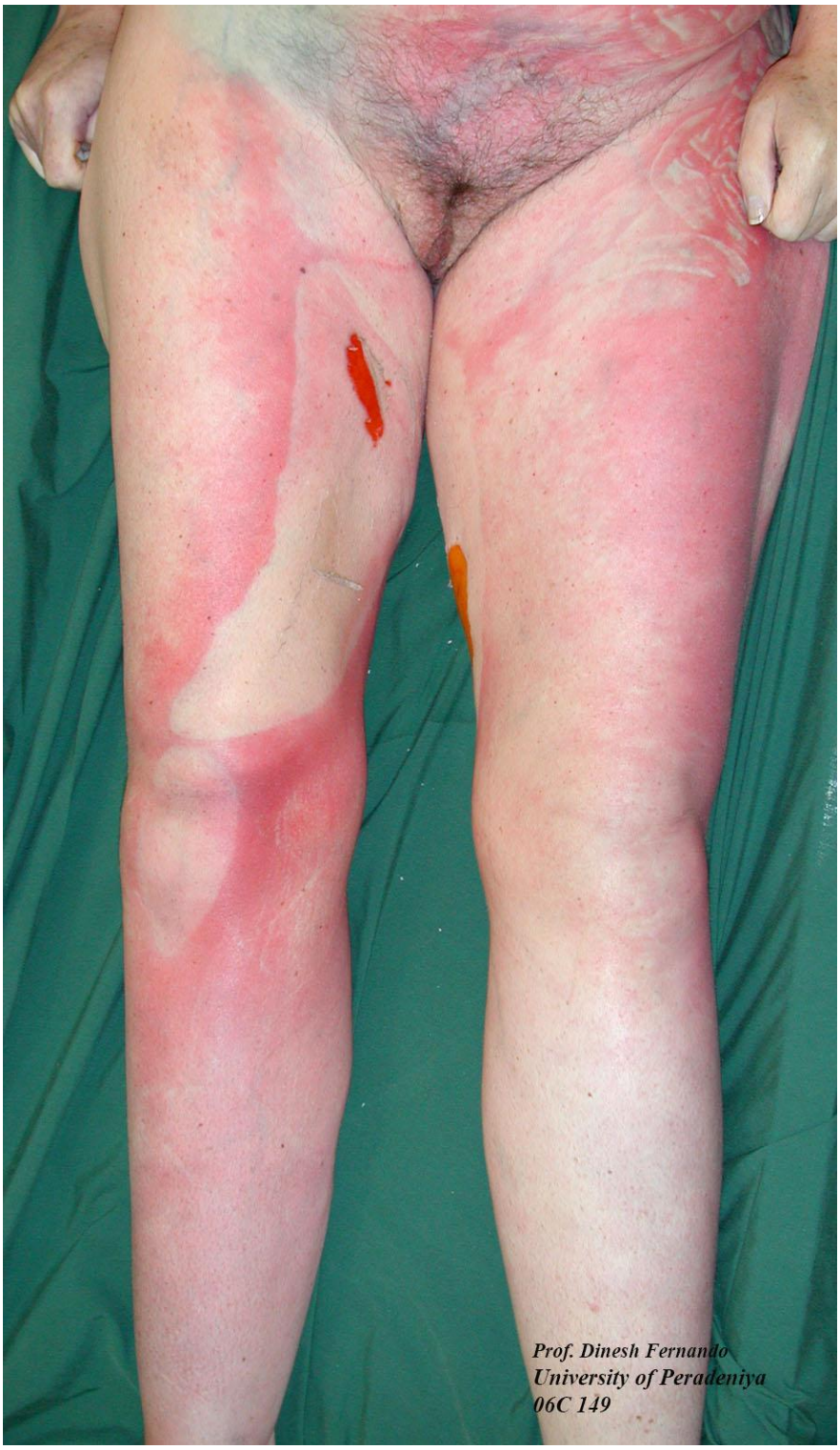
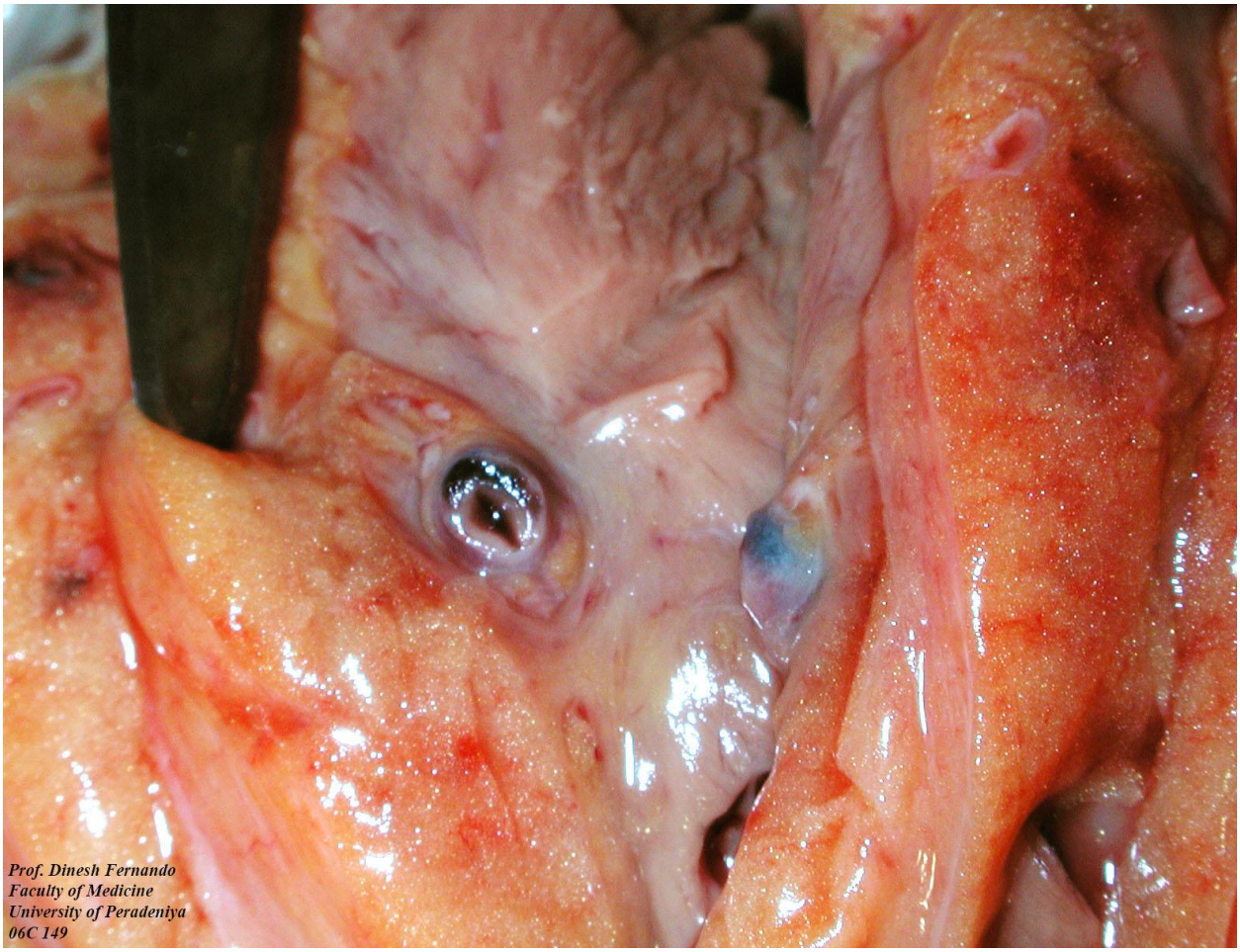


Figure 2: Hypostasis with an area of sparing on the right thigh due to being placed on the left thigh

Internal Examination

Cardiovascular System: Circumferential haemorrhage was present within the wall of the middle and distal thirds of the left anterior descending coronary artery. The myocardium was red-brown, flabby, and without evidence of fibrosis or necrosis grossly. Congestion was present in the posterior wall of the left ventricle.



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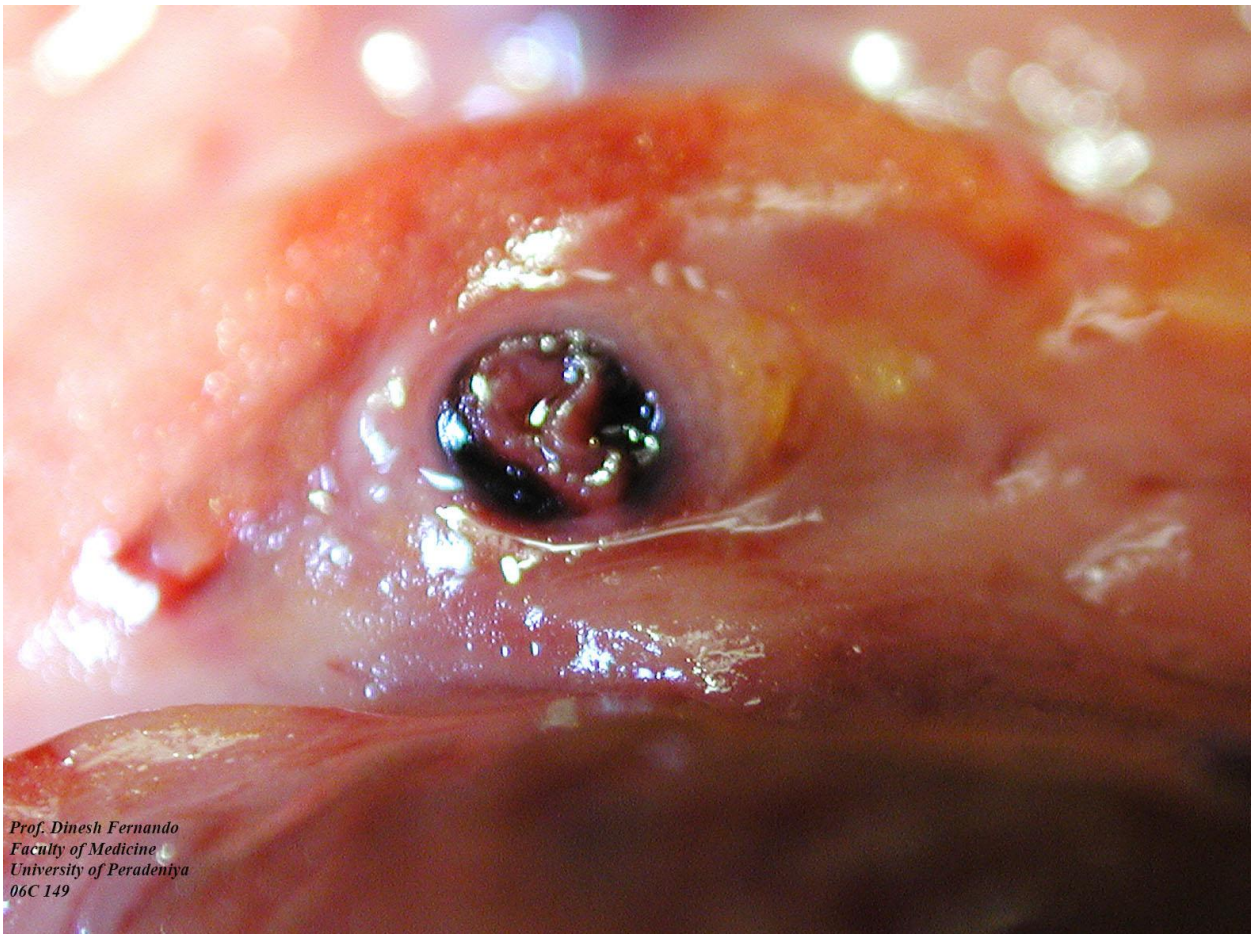

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Figure 3(a & b): Cut-section of coronary artery showing haemorrhage

Prof. Dinesh Fernando
Faculty of Medicine
University of Peradeniya
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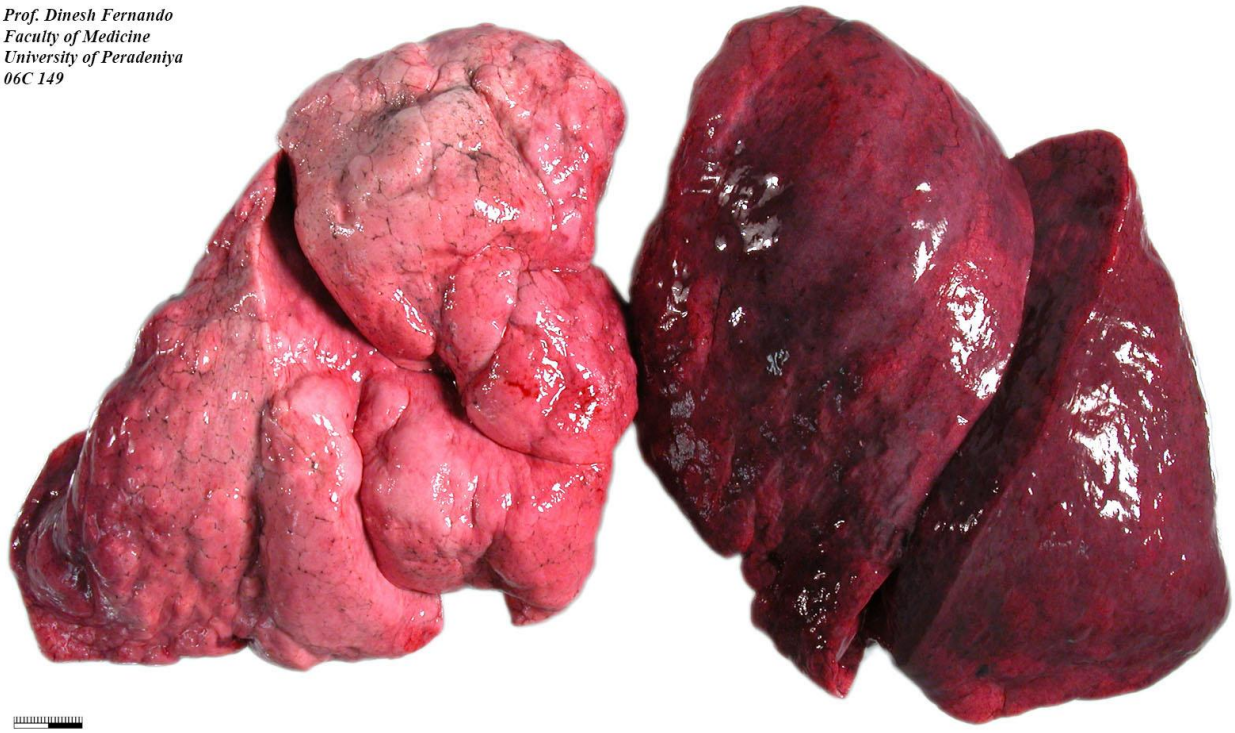

Figure 2: Hypostasis of left lung

Microscopic Examination

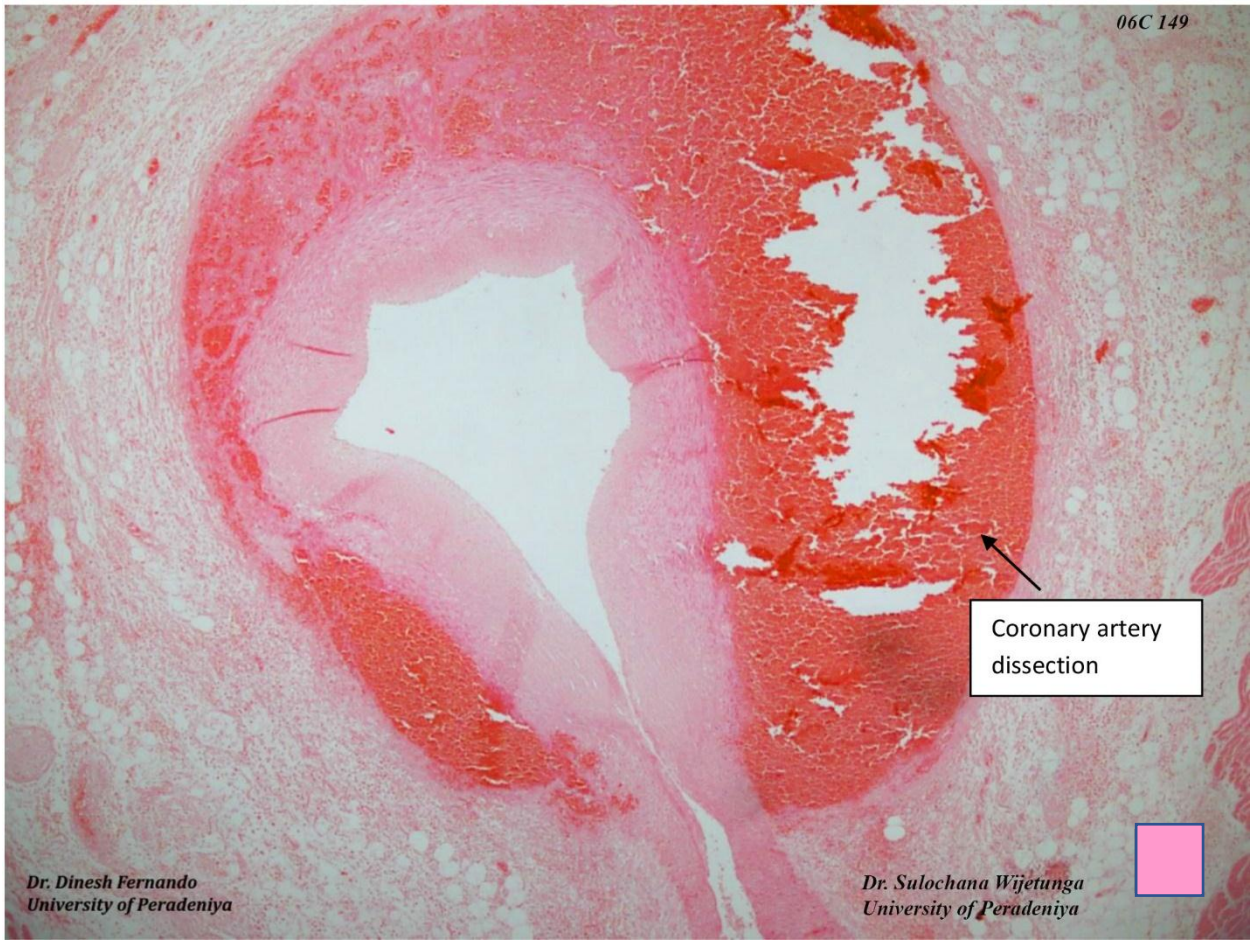

Figure 5: A blood filled channel within the arterial wall under Haematoxylin and Eosin stains

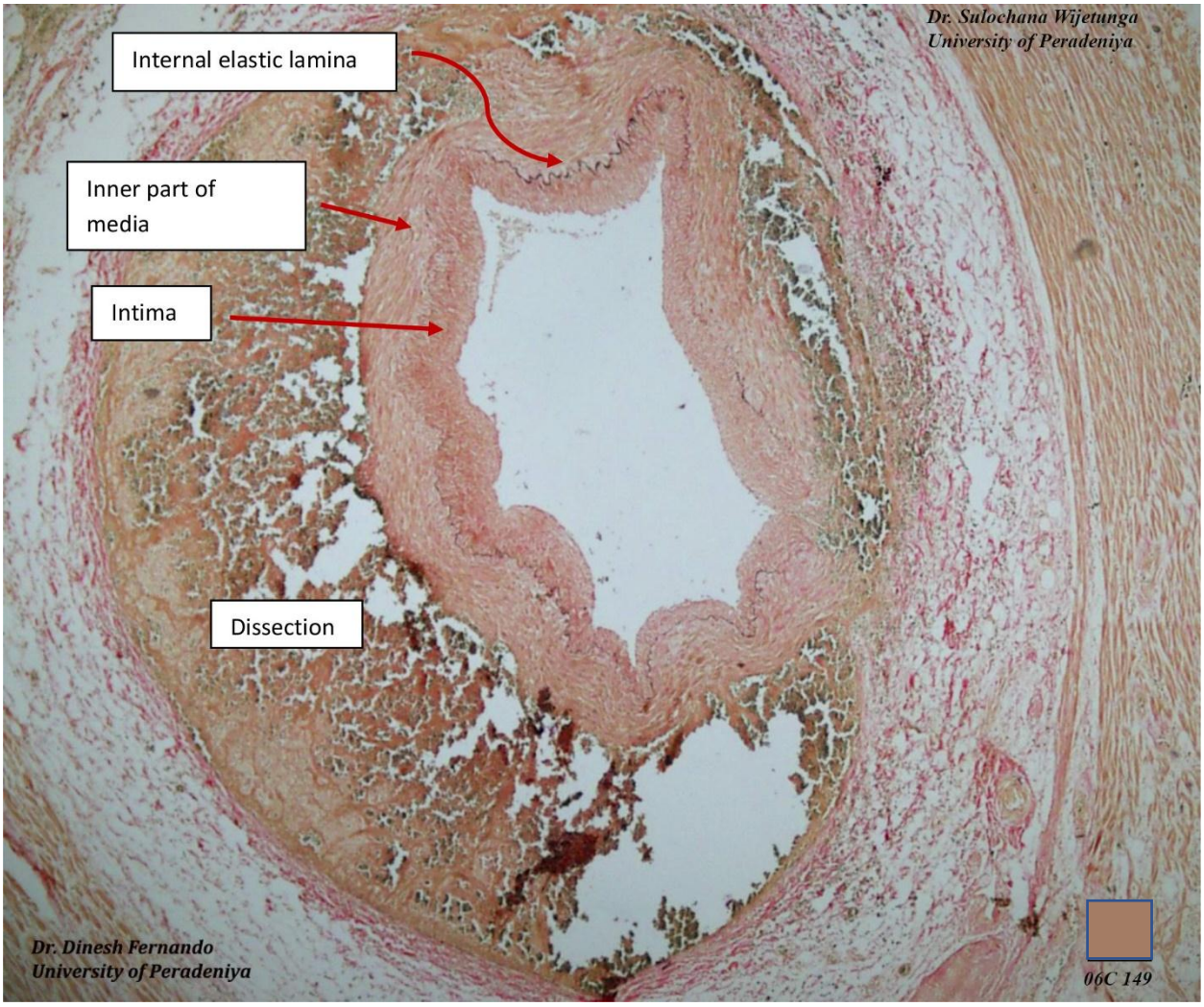

Figure 6: Elastin stain demonstrates that the dissection has occurred within the medial planes

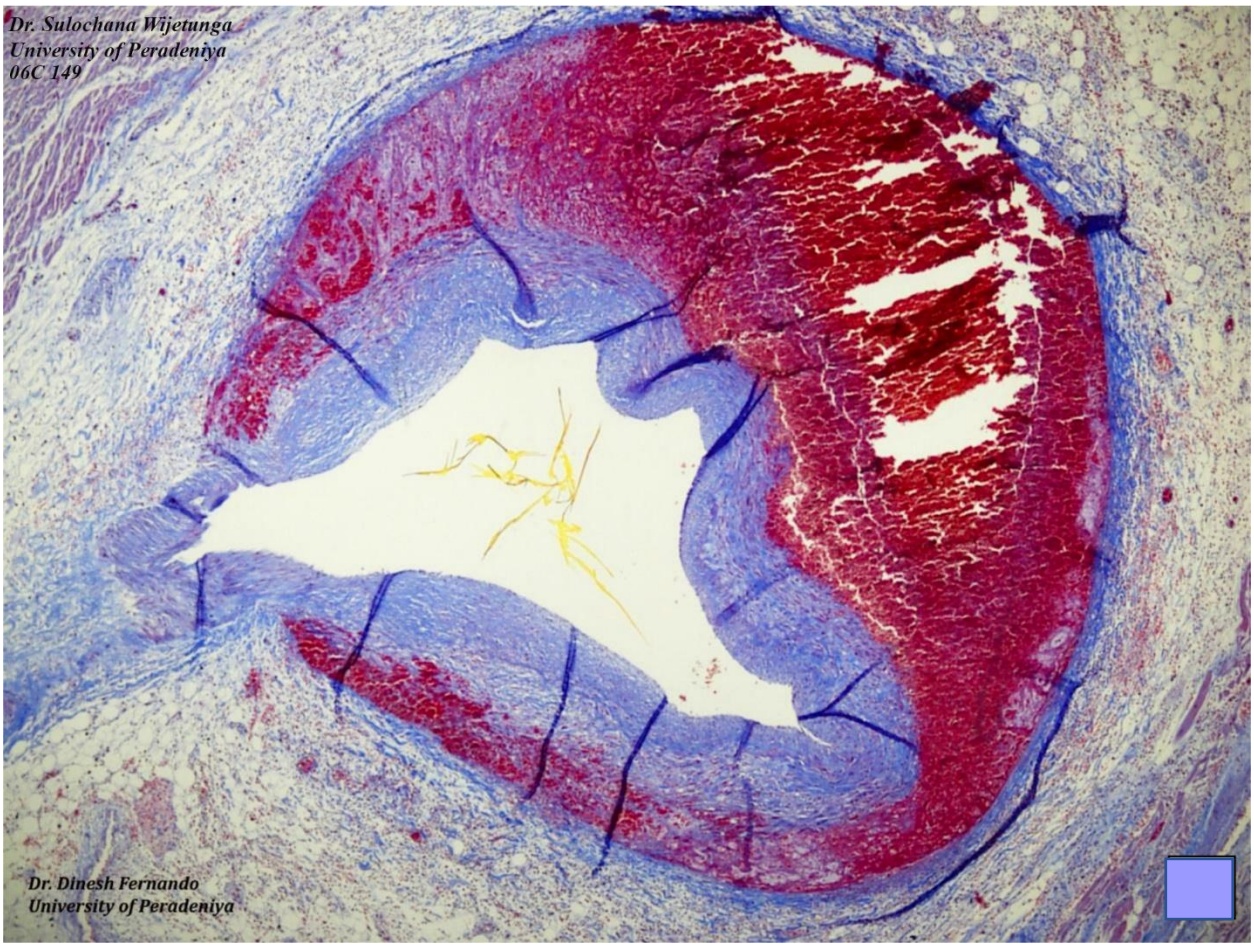

Figure 7: Masson trichrome stain shows blood tracking down through muscularis propria

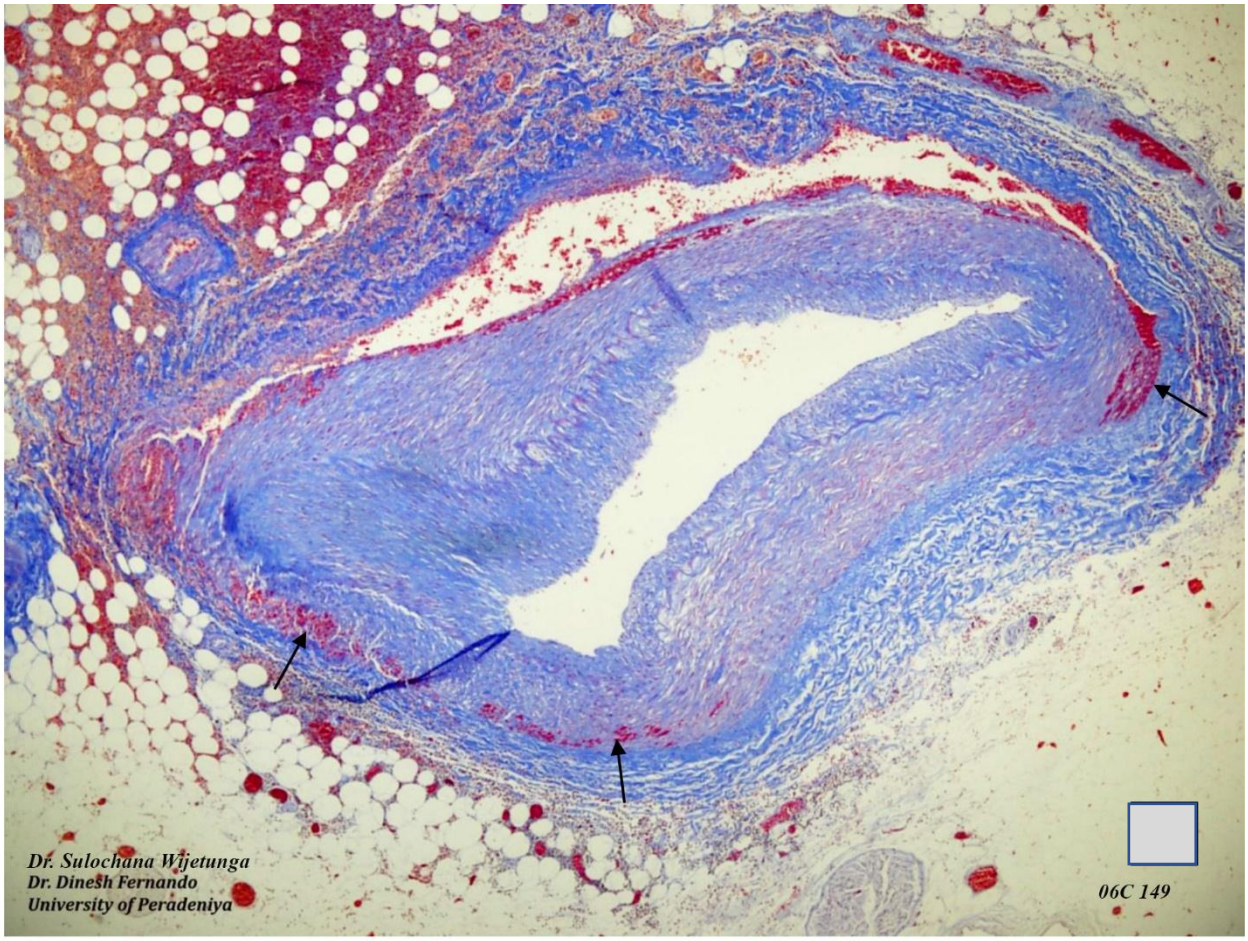

Figure 8: Masson trichrome stain shows blood tracking down through muscularis propria. Arrows show early stages of blood tracking down through the media.

Spontaneous coronary artery dissection (SCAD) is a rare cause of coronary artery occlusion and acute coronary syndrome. SCAD typically occurs in young otherwise healthy women.

In arterial dissection, blood tracks down along the planes of media, forming a blood-filled channel within the arterial wall. Arterial dissection is more common in the aorta; however, it can rarely occur in muscular arteries like coronaries.

Cause of death

Acute coronary insufficiency due to coronary artery dissection

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COLOR ATLAS OF

FORENSIC PATHOLOGY

BRAIN AND SPINAL CORD

ACUTE SUBDURAL HAEMATOMA

Subdural haematoma is an accumulation of blood in the subdural space without extension into the depths of sulci. Rapid movement of the brain during trauma can tear the bridging veins, which extend from the cerebral hemispheres through the subarachnoid and subdural space into the dural sinuses. A large number of subdural haematomas are caused by falls, commonly in the elderly and alcoholics. High energy trauma results in expanding haematoma with primary brain injury, causing rapid deterioration. Subdural haematoma is also seen following trivial low energy injury; especially in extremes of ages, mostly in elderly patients who are on anticoagulants. Infants have thin walled veins which tend to rupture easily. Subdural haematomas typically manifest within the first 48 hours after injury. However, the interval between injury and symptoms can be days, or extend to weeks or months.

SDH is categorized according to the duration between the injury and the onset of symptoms and signs. These are; acute, sub-acute and chronic SDH. Acute subdural haematoma presents within 72 hours, sub-acute SDH presents between 72 hours to 3 weeks, and presentation thereafter, is termed as a chronic SDH. Acute on chronic subdural haematomas refers to a second episode of acute haemorrhage into a pre-existing chronic subdural haematoma. Chronic SDH can re-bleed either spontaneously or as a result of a minor trauma.

SDH usually presents with headache, drowsiness and confusion. Focal deficits may develop later.
During the first week, the subdural haematoma organizes by lysis of the clot. Granulation tissue starts to grow into the haematoma from the dural surfaces during the 2nd week, followed by fibrosis in 1 to 3 months. Organized haematomas are attached to the dura, but not to the underlying arachnoid. Usually fibrosing parts get pulled away, leaving a thin layer of connective tissue which is known as “subdural membranes”.

A subdural haematoma acts as a space occupying lesion. This eventually increases the intracranial pressure. Rigid dural folds, the falx and tentorium, divide the cranial vault, and focal expansion of the brain causes displacement, in relation to these partitions. If the expansion is sufficiently large, herniation occurs. The main types of herniation are; subfalcine (cingulate gyrus) herniation, ascending or descending transtentorial (uncal) herniation and tonsillar herniation. In addition, central and transcalvarial herniation may be seen

Subfalcial herniation occurs when the ipsilateral cingulate gyrus is compressed beneath the free edge of the falx, usually due to a rapidly expanding mass in one cerebral hemisphere.

Transtentorial or uncal herniation occurs due to a rapidly expanding supratentorial mass lesion. It compresses the temporal lobe against the free margin of the tentorium. It may be either unilateral or bilateral; since rapidly expanding lesions are usually unilateral, ipsilateral uncal herniation can be expected. Transtentorial herniation can be divided into two types based on the direction of herniation: downwards due to supratentorial mass effect and upward due to infratentorial mass effect.

In large transtentorial herniations, the pressure on the midbrain compresses the contralateral cerebral peduncle against the incisura of the tentorium. This creates a deformation – the 'Kernohan's notch'. This will manifest with false localizing signs, which is known as Kernohan's phenomenon.
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Subfalcial herniation compresses the anterior cerebral artery (which supplies the orbital surfaces of the frontal lobes and medial surfaces of the cerebral hemisphere) and transtentorial herniation compresses the posterior cerebral artery, (which supplies the occipital lobe and infero-medial part of the temporal lobe) leading to secondary infarctions. When the intracranial pressure equals or exceeds the arterial blood pressure, the blood flow to the brain will stop. This will cause additional cerebral oedema which, in turn, causes further herniations.

Tonsillar herniation is the displacement of the cerebellar tonsils through the foramen magnum which is life threatening. Herniation may be either symmetrical, due to brain swelling, or asymmetrical. Symmetrical herniation of the cerebellar tonsils without brain stem haemorrhage is usually seen in diffuse brain swelling. As a result of tonsillar herniation, the brainstem is compressed, causing impairment of brain stem reflexes such as corneal, gag and swallowing. The brain stem and cerebellar tonsils are forced into the foramen magnum, with resultant dysfunction or even infarction of the brain stem.

Rapid development of a subdural haematoma with mass displacement of the brain, with or without generalized cerebral oedema, may result in compression of the brain stem and development of secondary, linear or flame shaped haemorrhages, known as Duret's haemorrhages. They range from small streaks to massive confluent haemorrhage. They are in the midline and are most commonly associated with asymmetrical herniation of the brain stem. These are caused by tearing of penetrating vessels which supply the upper brain stem resulting in secondary herniation haemorrhages of the midbrain and pons. Duret haemorrhages may develop in only 30 min.

Increase in pressure can damage the brain, by decreasing perfusion, by displacing tissue across dural partitions inside the skull or through openings in the skull (herniations). Cushing reflex is a compensatory mechanism in order to maintain brain perfusion which includes a triad of symptoms; bradycardia, hypertension and respiratory irregularity.

Death occurs as a result of central respiratory failure due to compression of the midbrain and downward displacement of the cerebellar tonsils and compression of the medulla.

History

An 88-year-old female who had been on long term treatment with Warfarin was found dead in her house, seated in front of the television.

External Examination

An abrasion measuring 0.5 cm in diameter was situated on the left supra orbital ridge. No other injuries on the body.

Internal Examination

The weight of the brain was 1100 g. There was cerebral oedema and congestion of vessels. There was atheroma formation in the circle of Willis.

An acute subdural hematoma with a volume of 210 ml was present on the right side. No membrane formation was seen. The right hemisphere was depressed. There was a midline shift towards the left. There was necrosis of the uncal region of the hippocampal gyrus. Haemorrhage was seen along the imprint of the free margin of the tentorium cerebelli which extended from the uncal region to the posterior aspect of the hemisphere. On coronal sections of the hemispheres there was a 5 mm area of haemorrhage in the basal ganglia region on the right side and necrosis of the hippocampal gyrus on the right side. Transverse sectioning of the brain stem showed haemorrhages which were multiple and peripheral in the pontine region. In the midbrain region a large single haemorrhage which was mainly central was present.

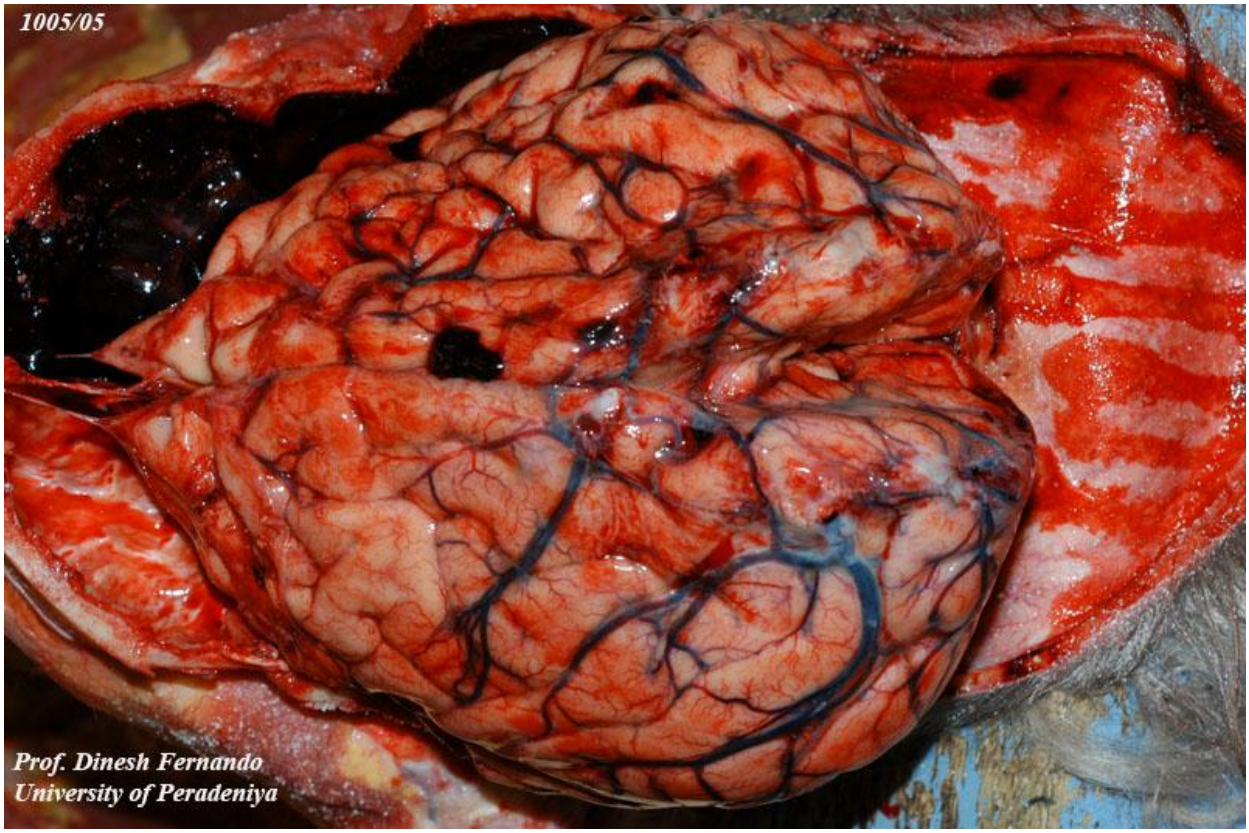


Figure 1: Acute subdural haematoma. Note: congestion of blood vessels

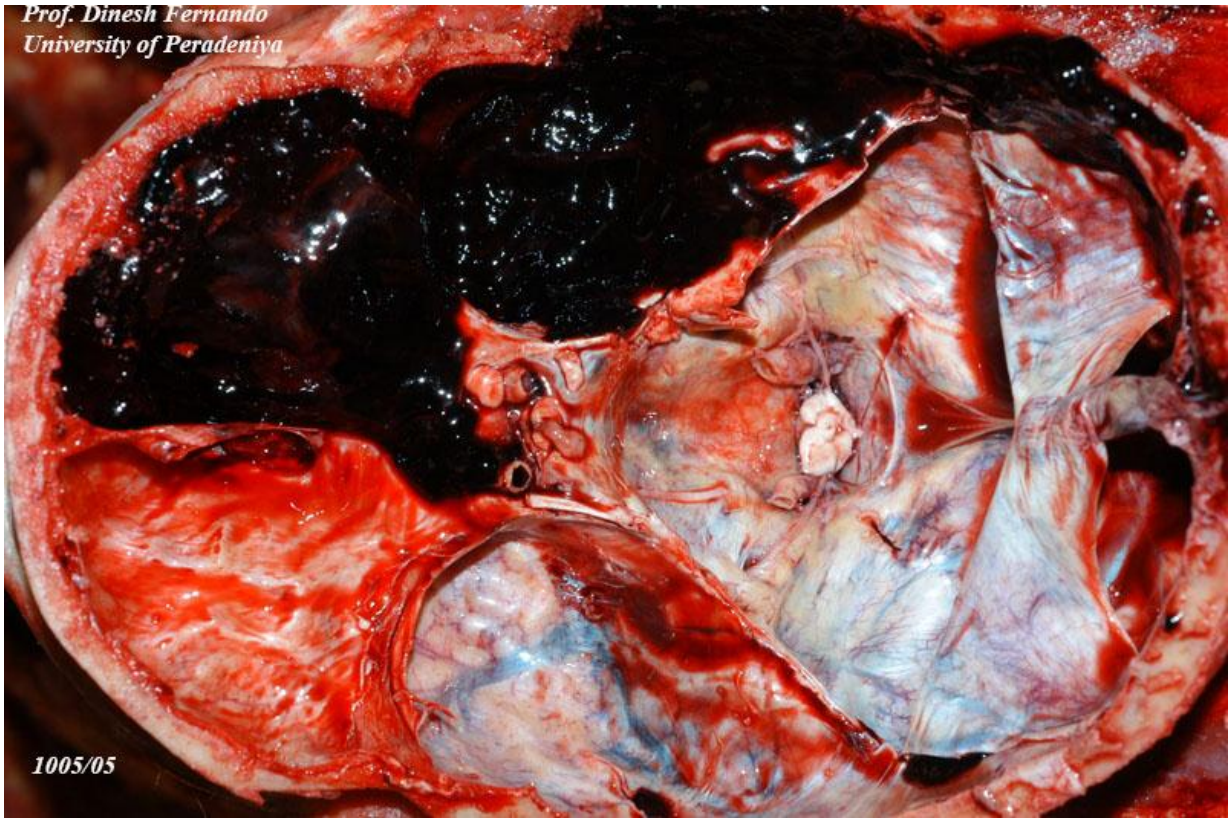


Figure 2: Acute subdural haematoma after removal of the brain

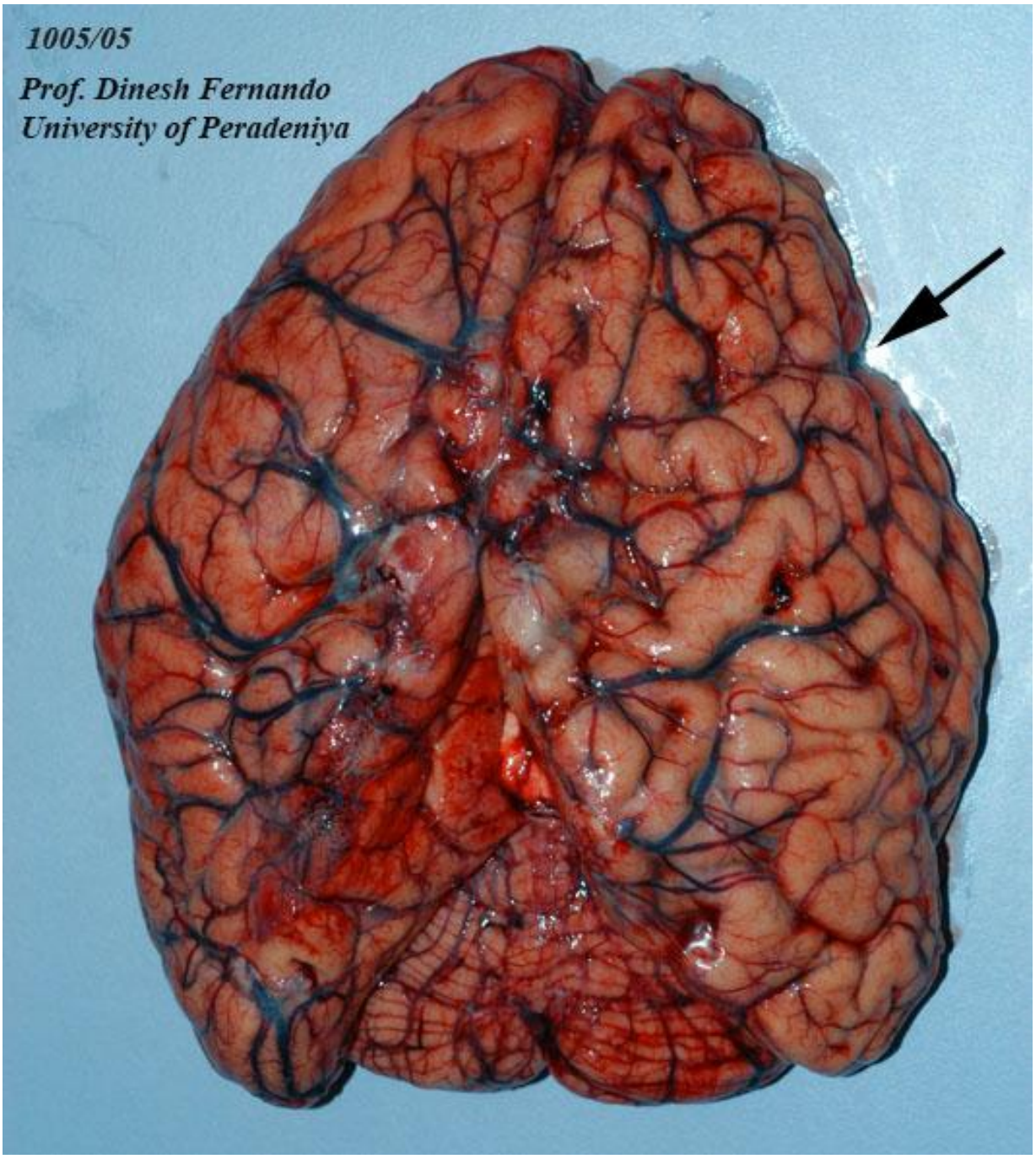
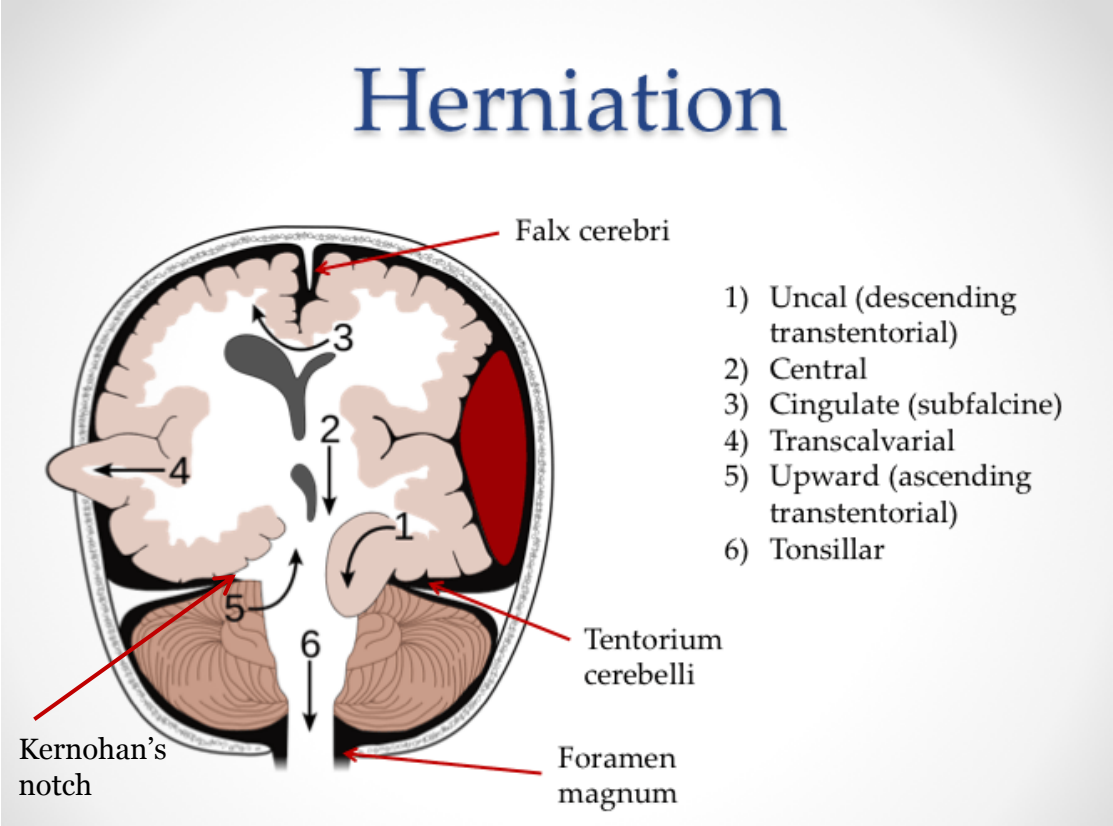
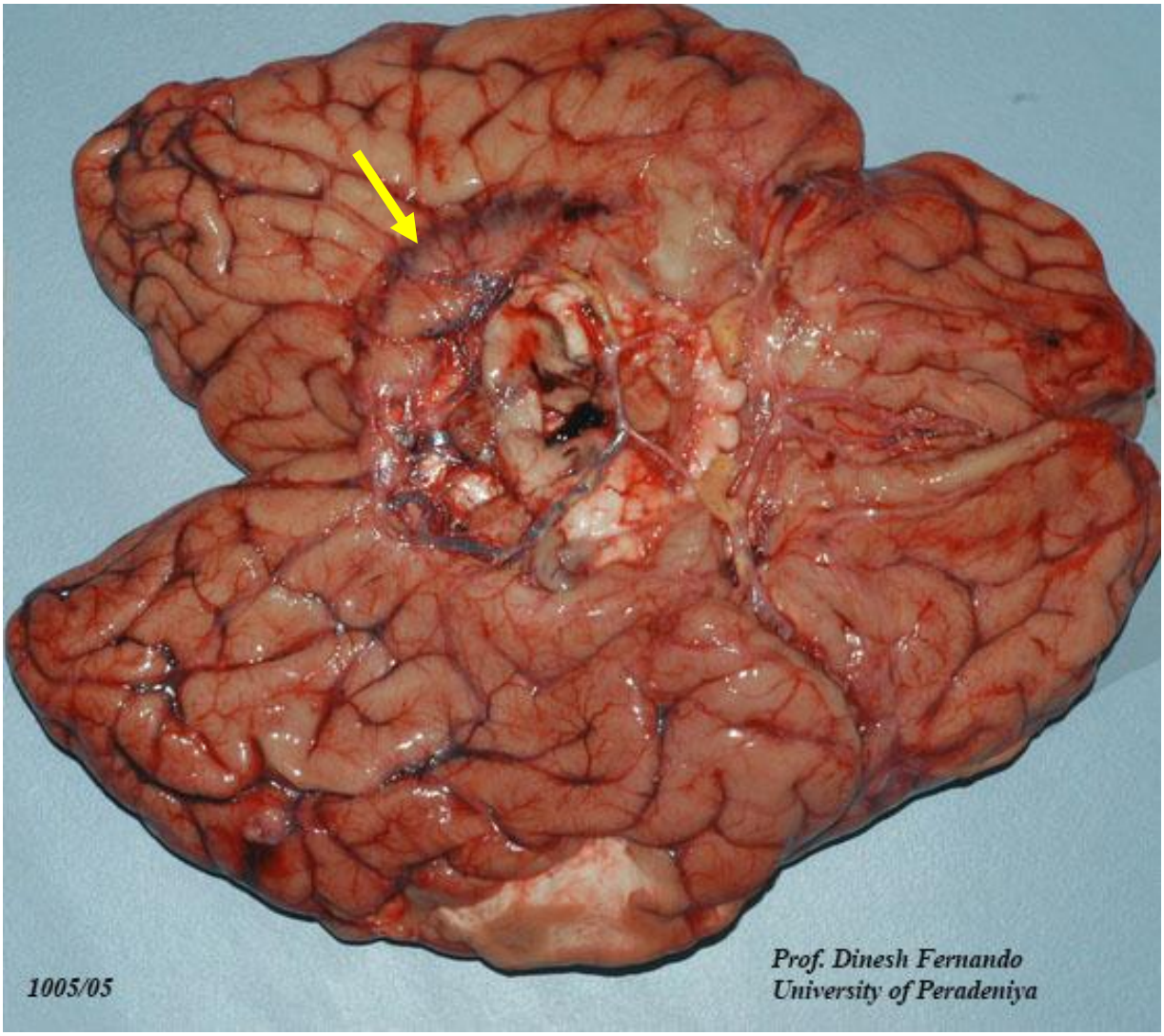


Figure 3: Cerebral oedema. Note: compression of the right cerebral hemisphere associated with midline shift to the left (arrow)

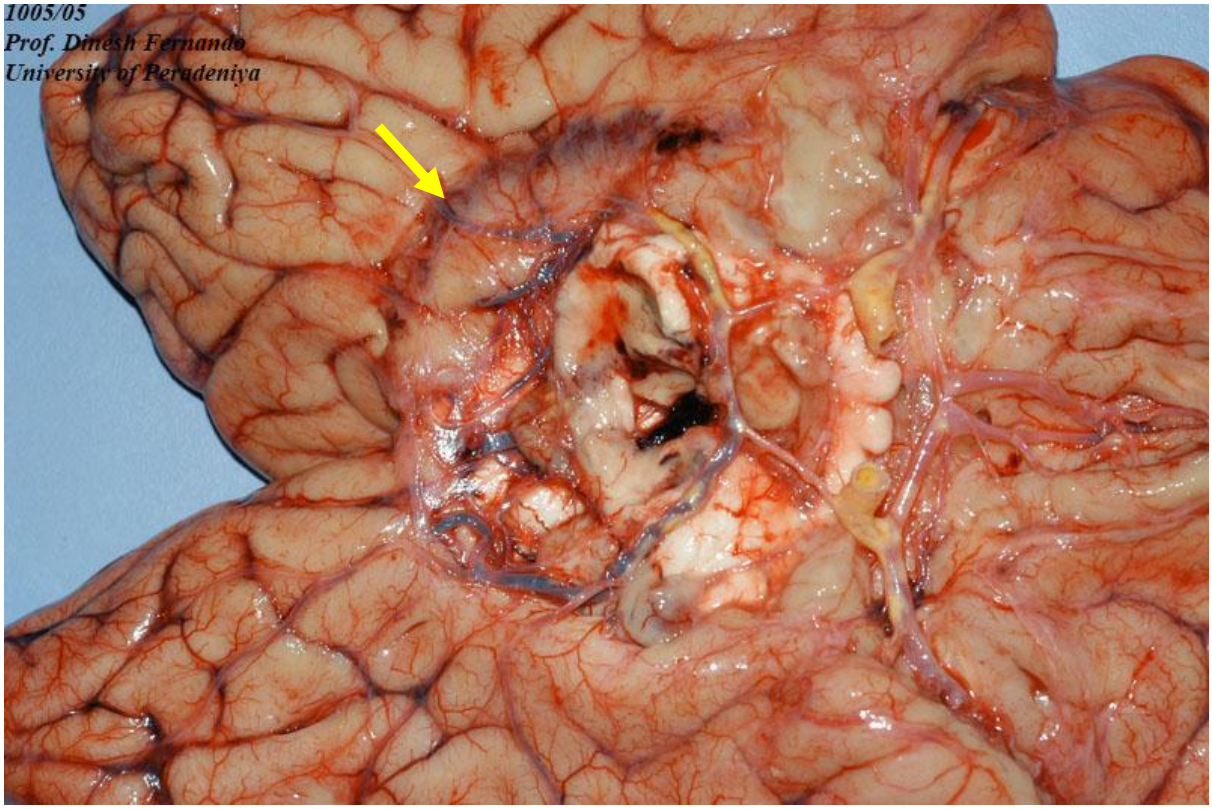


(Source: Wheeler J. *Significance of pupillary dilatation in head trauma*. [updated 26 Feb 2015] Available from: https://scghed.com/2015/02/cme-260215-significance-of-pupillary-dilatation-in-head-trauma/ Accessed 1st July 2020)

Figure 4: Schematic representation of Kernohan's notch. Demonstrated here are a subdural hematoma and uncal herniation on the same side. Notching of the midbrain is seen on the opposite side (Kernohan's notch). This damages the contralateral pyramidal tract fibres.



(a)



(b)

Figure 5(a, & b): Base of brain showing herniation of the uncus. Note: the haematoma caused by the free margin of the tentorium cerebelli on the hemisphere (arrow)

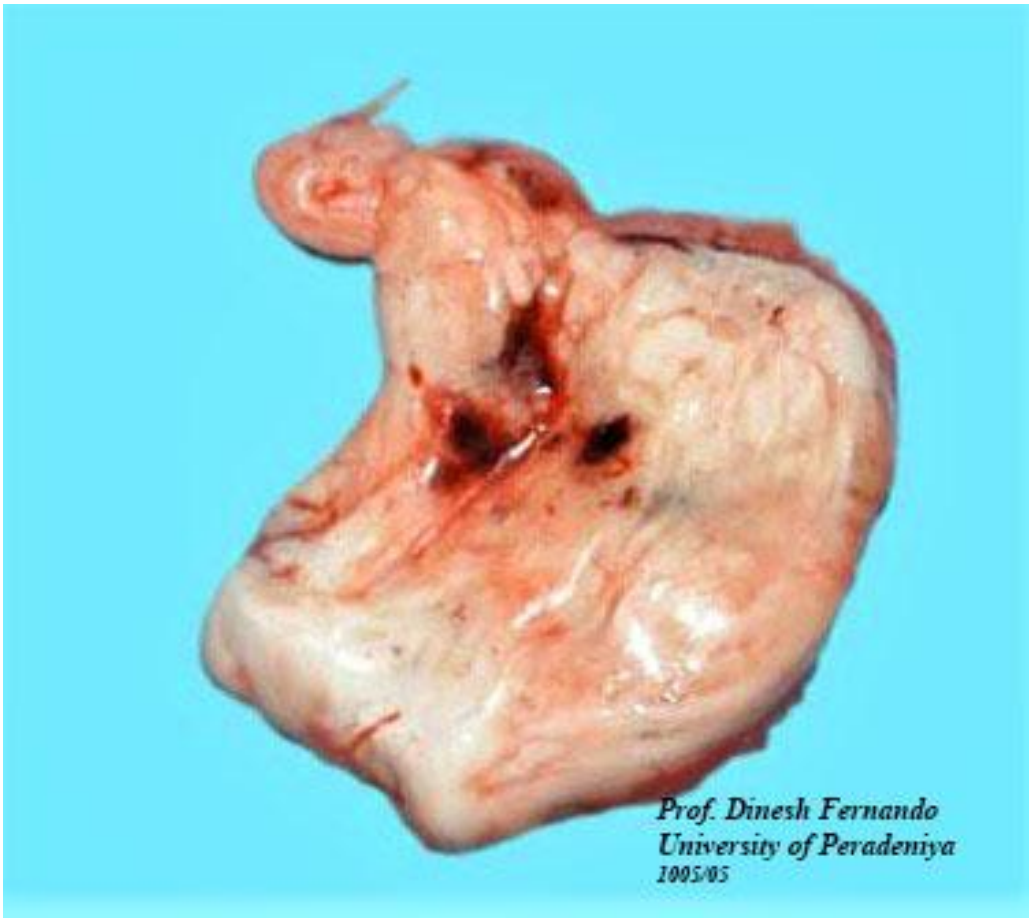


Figure 6: Brain stem haemorrhage

Microscopic Examination

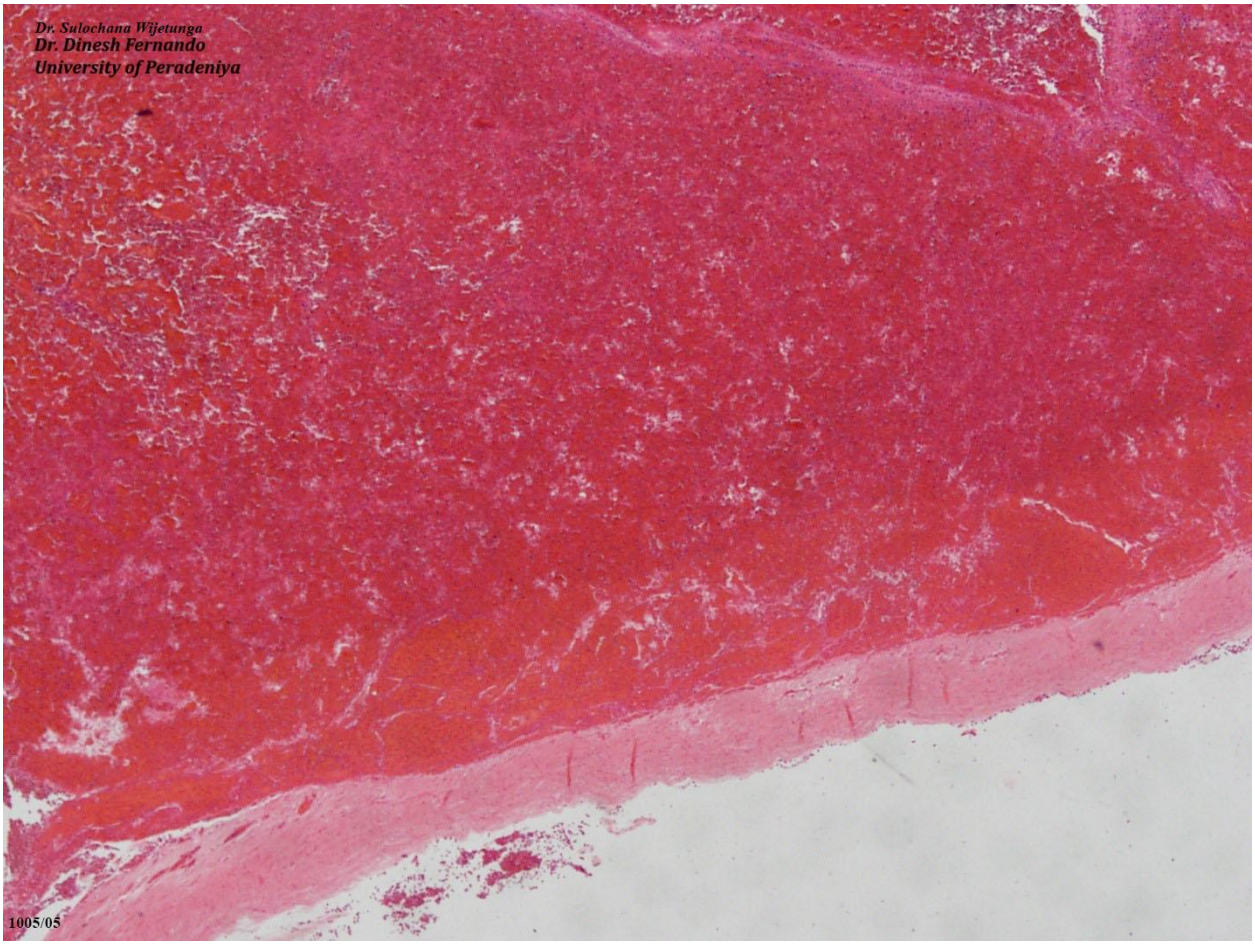


Figure 7: Acute subdural hematoma. Blood collects between the dura and the arachnoid membrane. Sometimes arachnoid membrane may also be torn due to trauma.

Cause of death

Acute subdural haematoma

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SCALDS IN A PERSON WITH ALZHEIMER'S DISEASE

Alzheimer's disease (AD) is the commonest cause of dementia in the elderly. It is a neurodegenerative disease caused by the accumulation of the proteins, beta amyloid and tau, in specific regions of the brain. This leads to insidious impairment of higher cognitive functions, which manifests as progressive memory impairment, deficit in visuo spatial orientation, language and personality over time. Grossly the brain shows a variable degree of cortical atrophy with widening of the cerebral sulci. Two pathological hallmarks of AD seen mainly in the end stage of the illness are neuritic (senile) plaques and neurofibrillary tangles. Both plaques and tangles contribute to progressive neural dysfunction with an initiation of an inflammatory response from microglia and astrocytes.

'Scald' is a feature of superficial (first degree) burns mostly due to tissue damage from hot liquids, usually by hot water. Erythema and blistering are the general features of scalds, but charring of the skin is also found when the liquid is extremely hot, such as with molten metal. Scalding burns generally occur on exposed skin, as even one layer of clothing can be sufficient to protect the body.

History

A 79-year-old-female in an advanced stage of Alzheimer's disease had filled a bath tub full of very hot water and got into it. She was unable to get out on her own due to having several hip replacement surgeries. When her husband heard her crying for help, he saw that she was sitting in the bath tub with both her legs draped over the sides of it.

External Examination

Approximately 50 % of the body surface area had scalds. It involved the entirety of the upper and lower back and both buttocks. The lateral aspects of the chest with extension to the anterior aspect of the chest, abdomen and upper thigh were scalded as was the entire posterior aspect of left upper limb and posteromedial aspect of right upper limb.

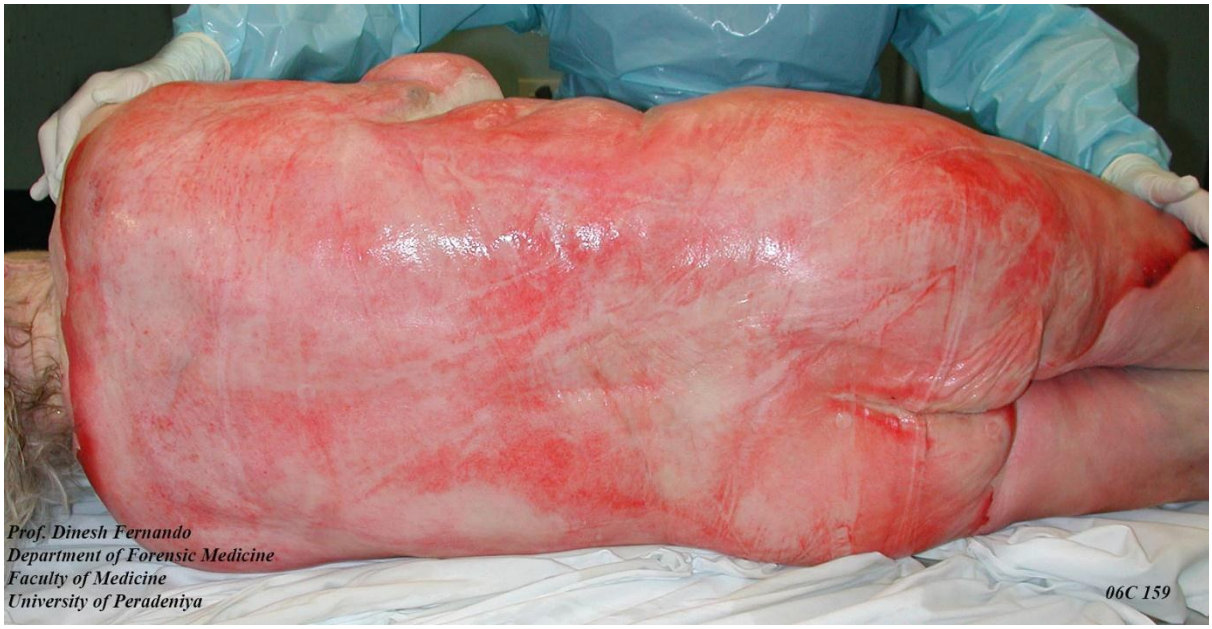


Figure 1: Scalds involving entire posterior aspect of the body. Note sharp margin on thigh

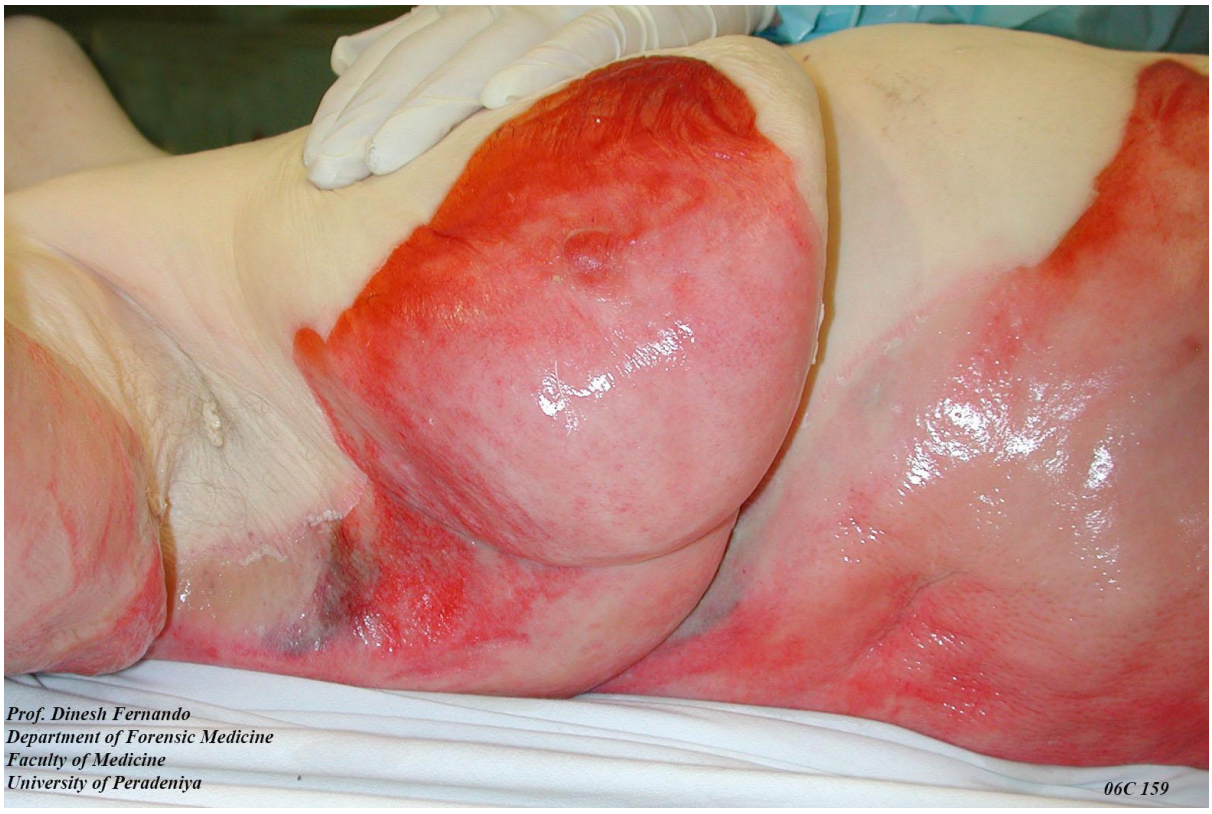


Figure 2: Scalds involving lateral aspect of the body. Note sparing of axilla

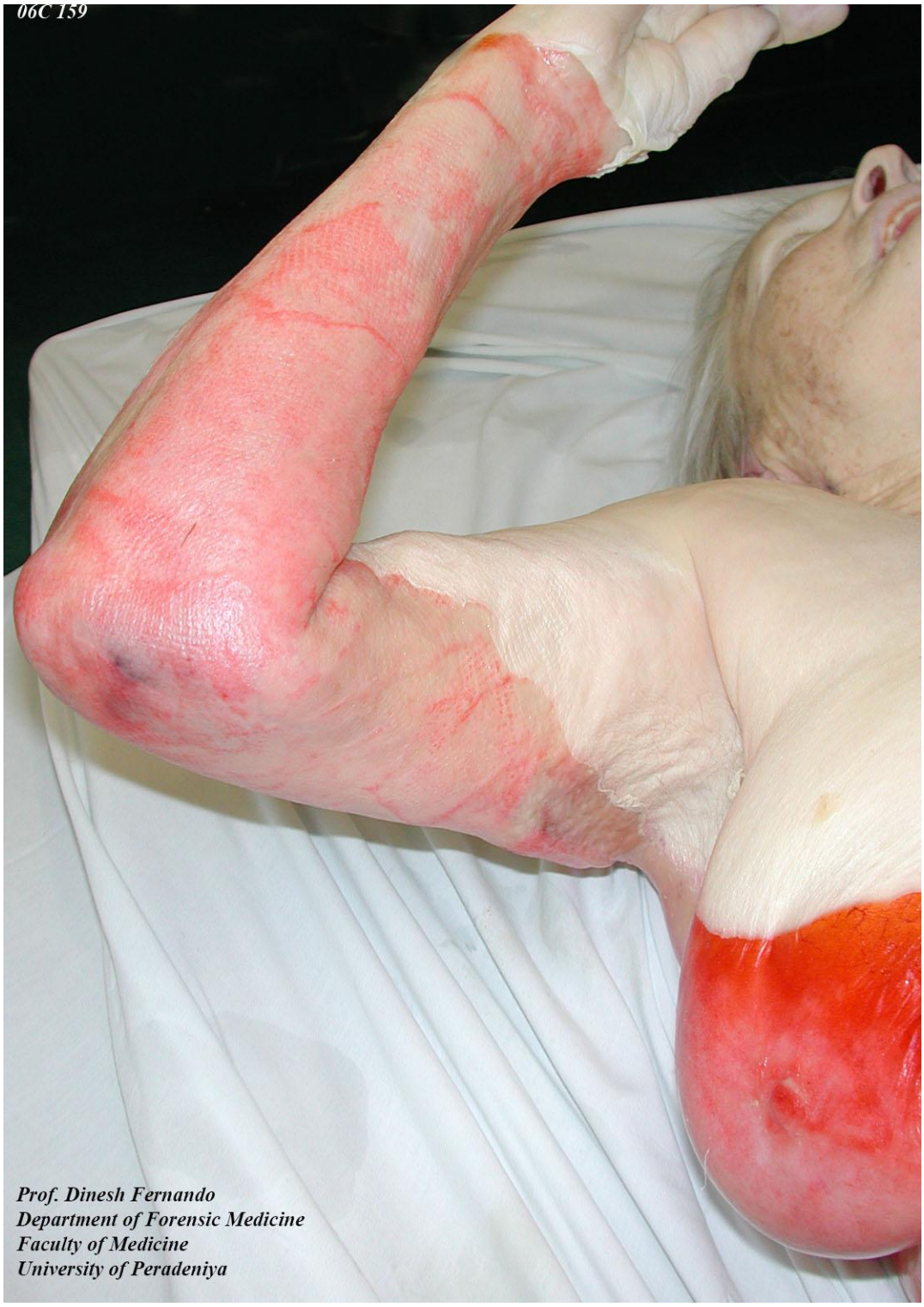


Figure 3: Scald of upper limb and wrist. Note sharp margin and sparing of axilla

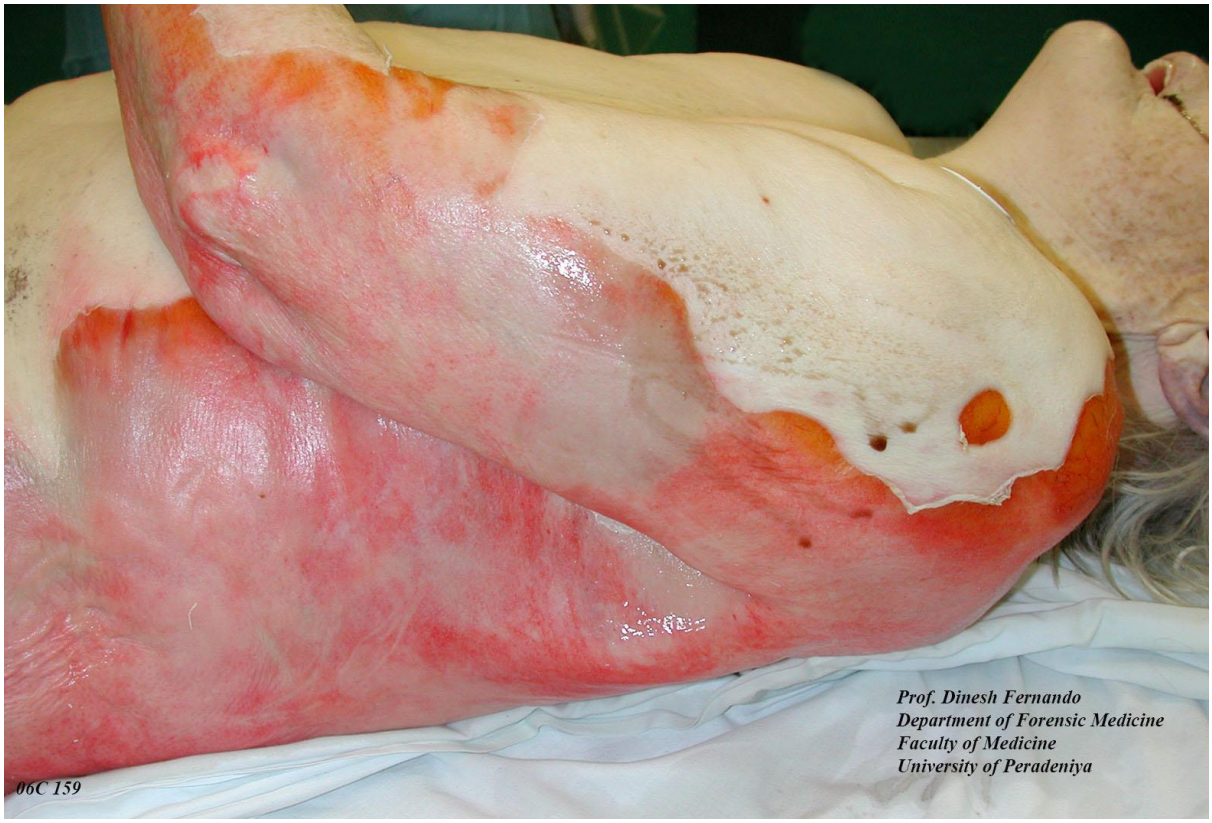


Figure 4: Note circular burn due to splashing on the outer aspect of the left arm

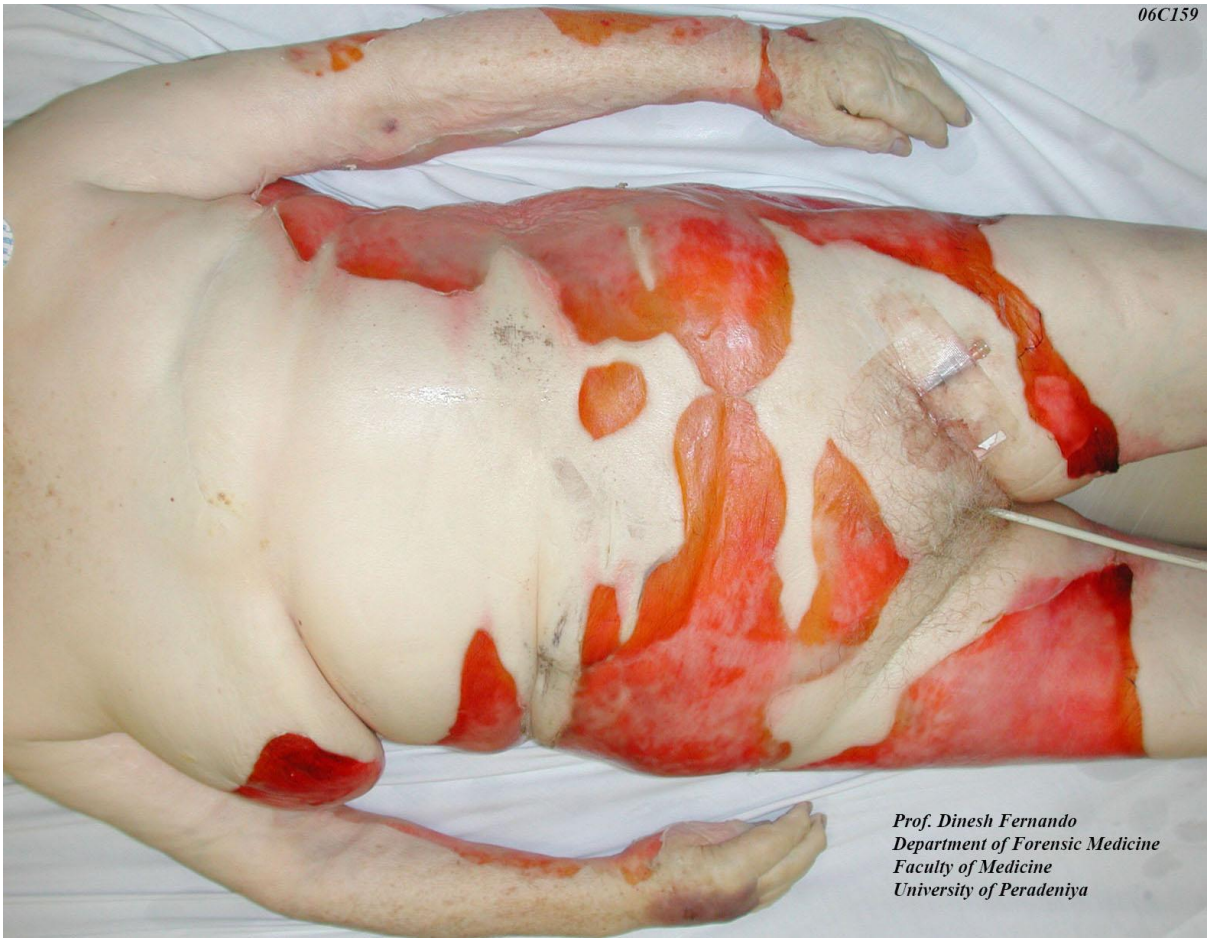


Figure 5: Displaced water extending over the anterior aspect of the body. Note the sharp margins and erythematous base

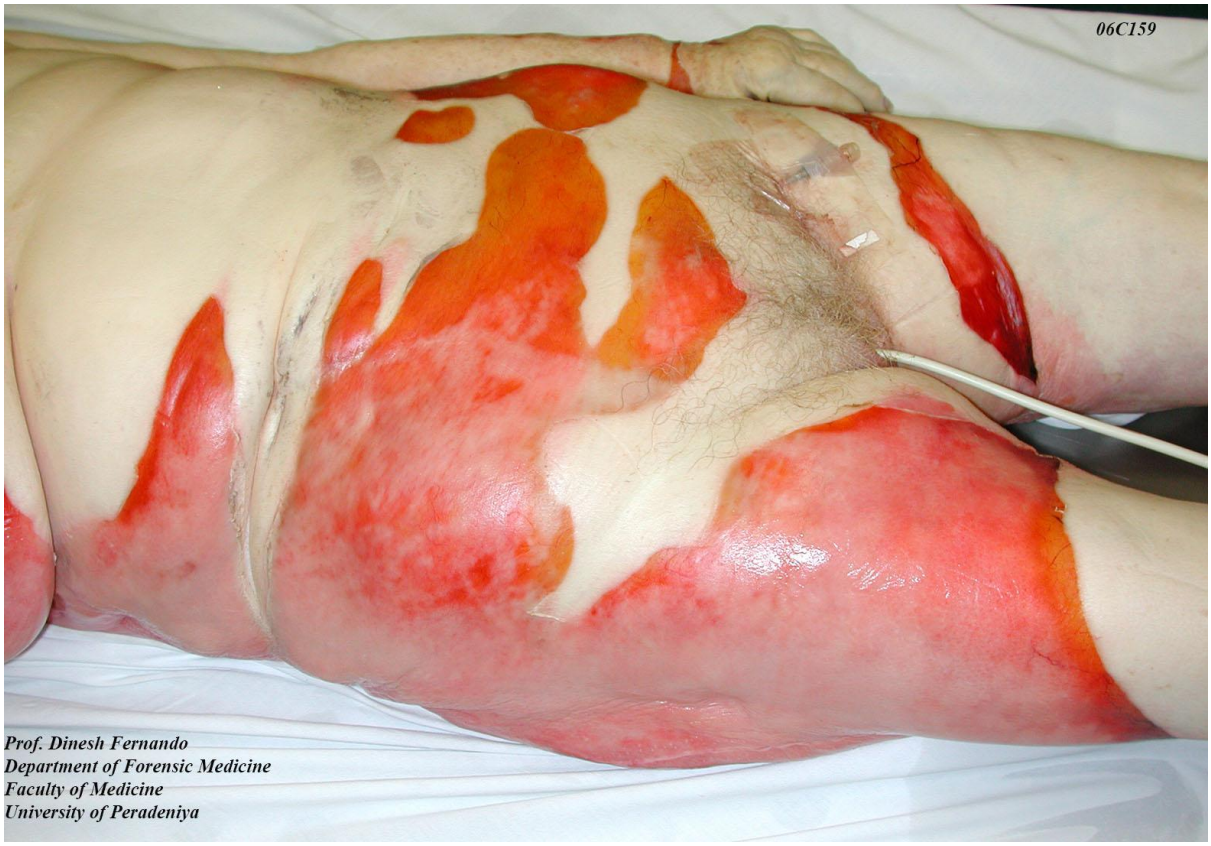
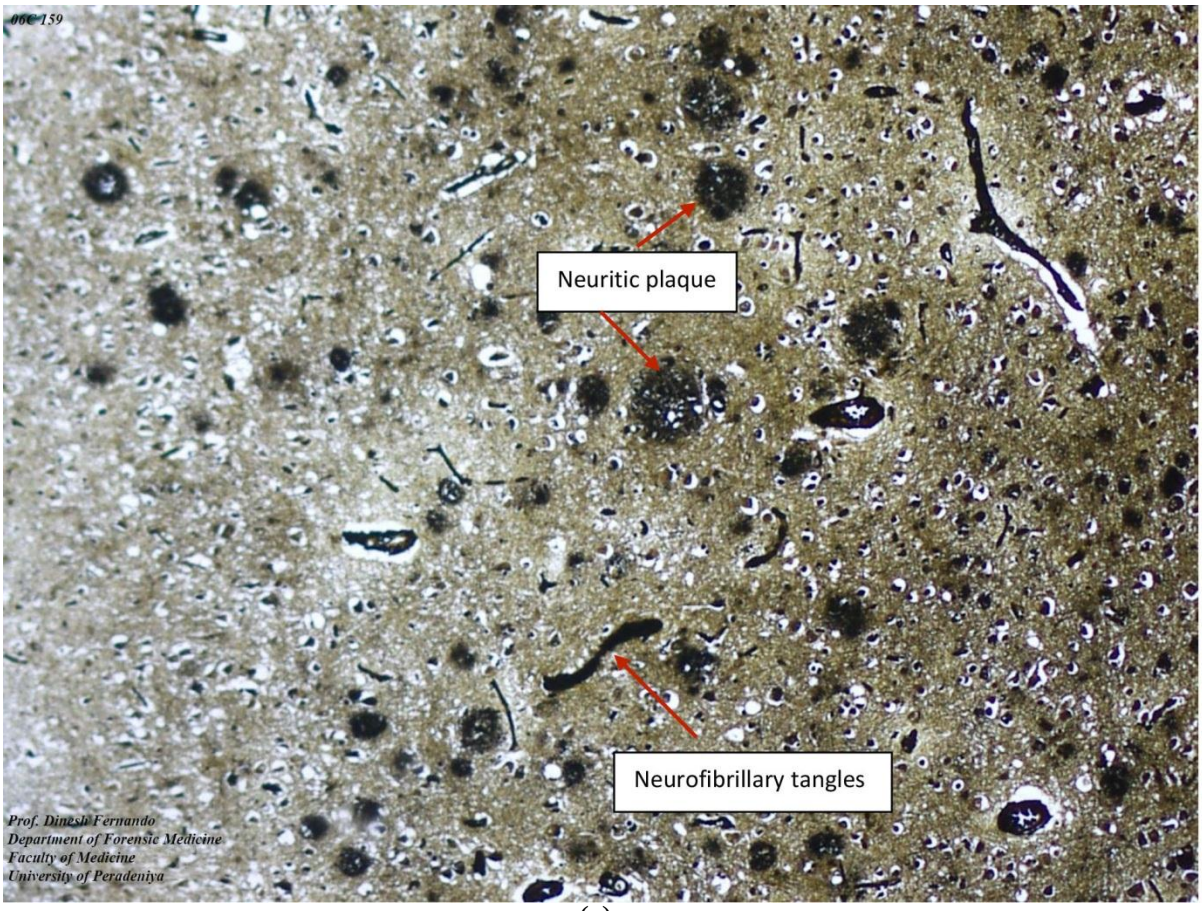
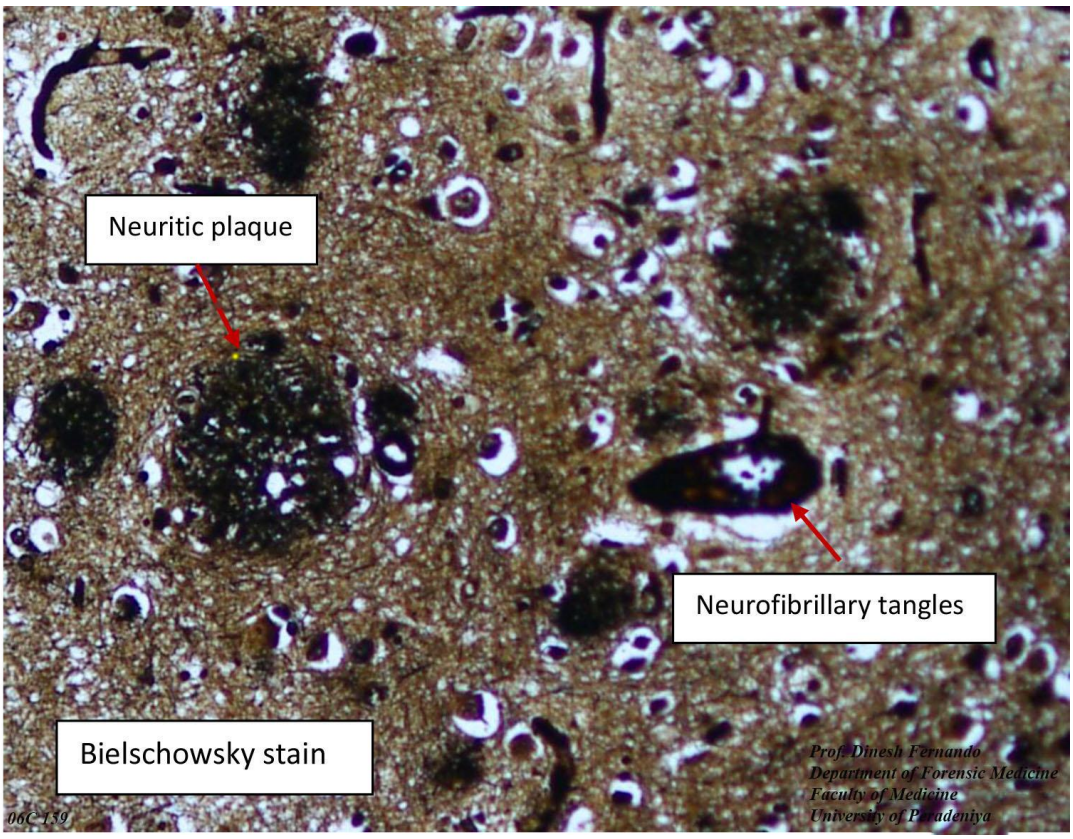


Figure 6: Note sparing of skin folds and inguinal region due to flexion of hip

Microscopic Examination



(a)



(b)

Figure 7(a, & b): Neurofibrillary tangles and neuritic plaques (Bielschowsky stain)

- Demonstration of neurofibrillary tangles and neuritic plaques in brain sections are required for confirmation of Alzheimer's disease at autopsy. Although these structures can be seen with routine haematoxylin and eosin stain, they are clearly demonstrated with Bielschowsky stain (a silver stain).

- Neurofibrillary tangles and neuritic plaques can also be seen as a senile change and in other degenerative diseases of the brain; however, these are characteristic features of Alzheimer's disease.

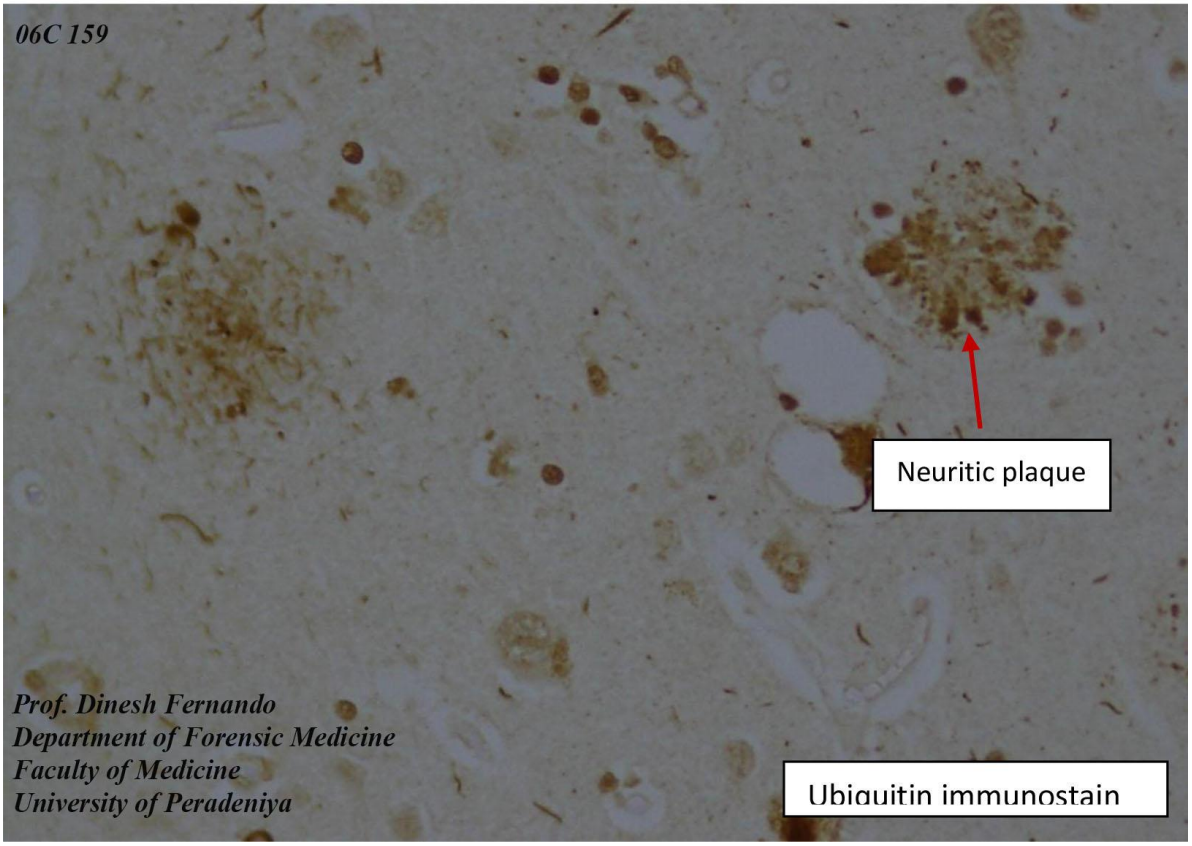


Figure 8: Neuritic plaque Stained with Ubiquitin immunostain

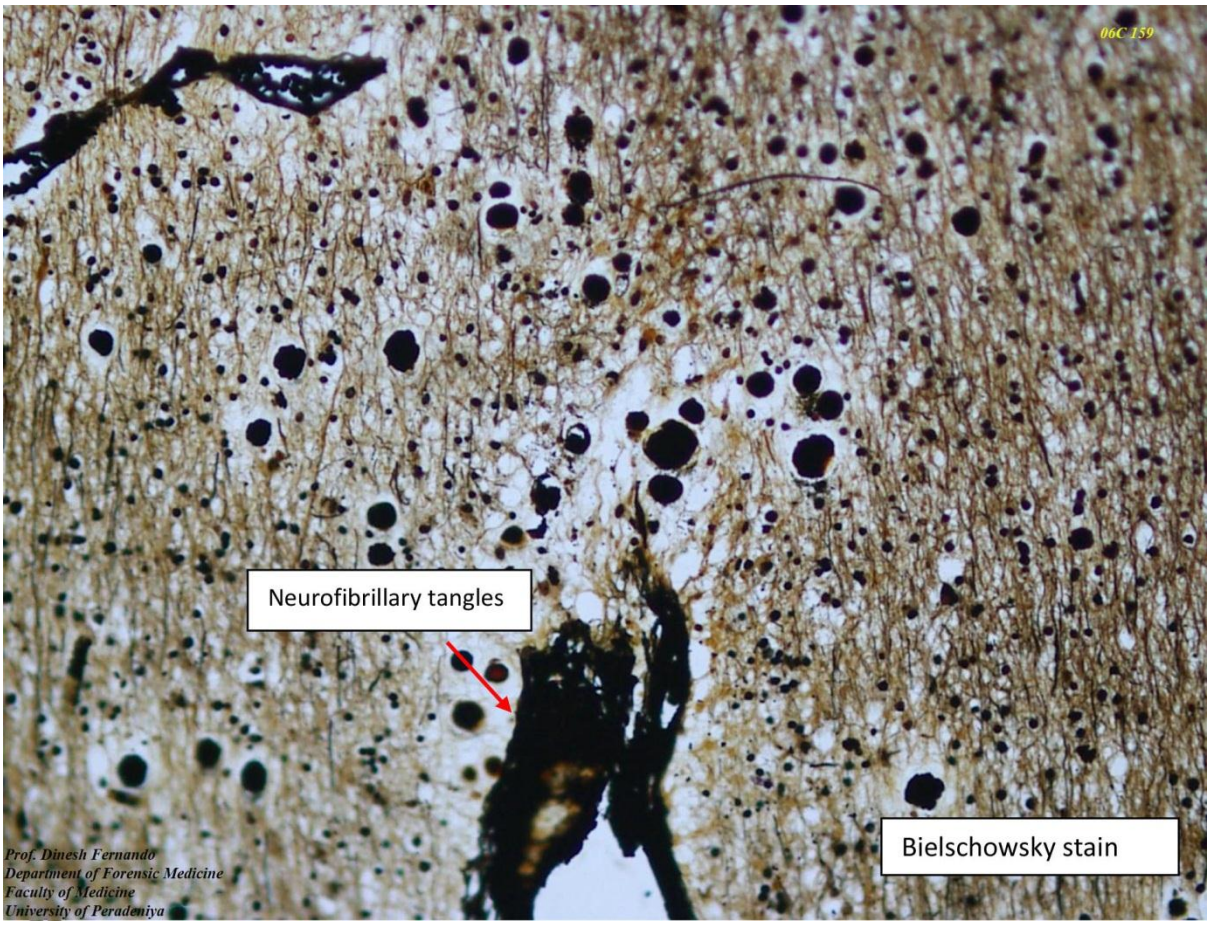


Figure 9: Neuritic plaques are degenerated neural structures deposited around small amyloid deposits.

- Neurofibrillary tangles are formed by accumulation of abnormally processed tau proteins inside neurons. The affected neurons eventually die and the insoluble proteins persist.

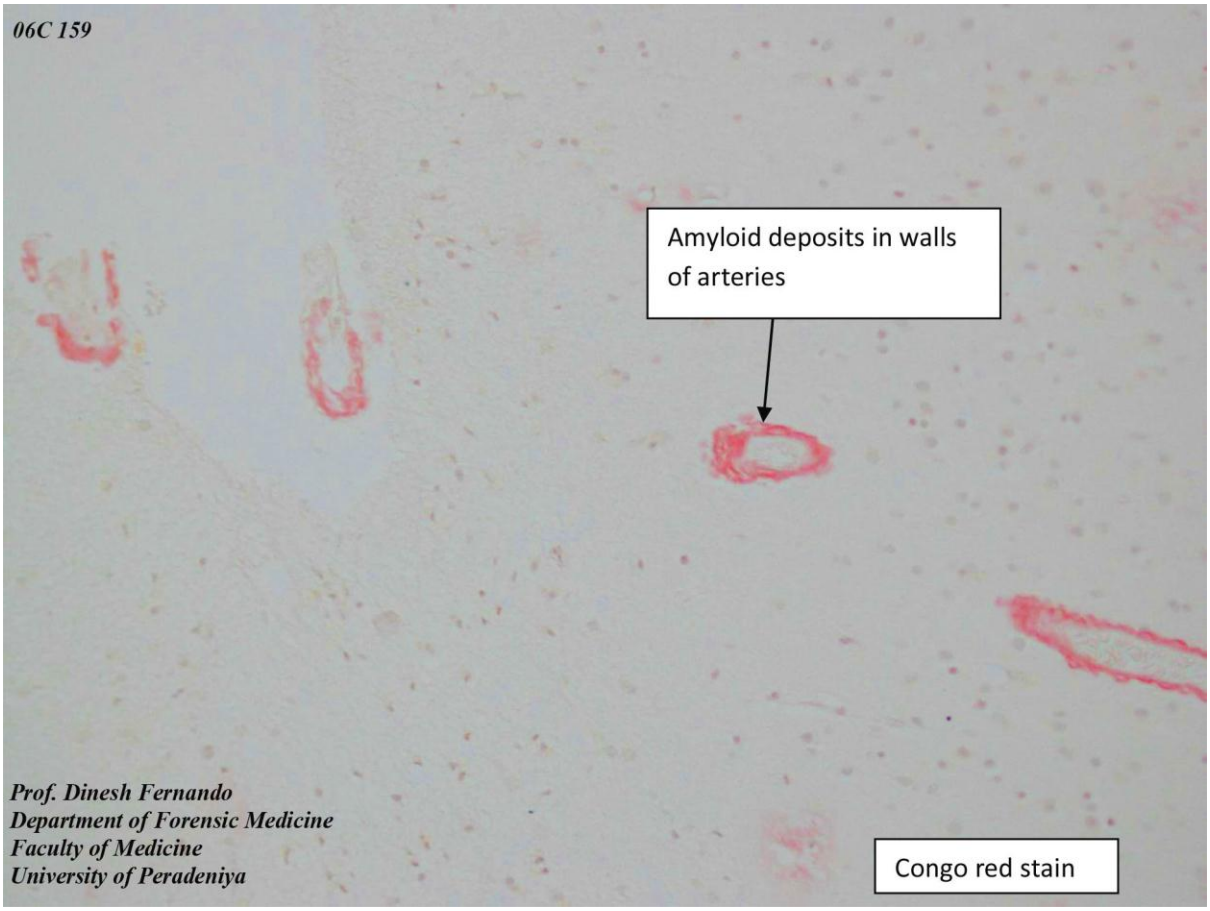


Figure 10: Amyloid deposits in walls of arteries stained with Congo red stain

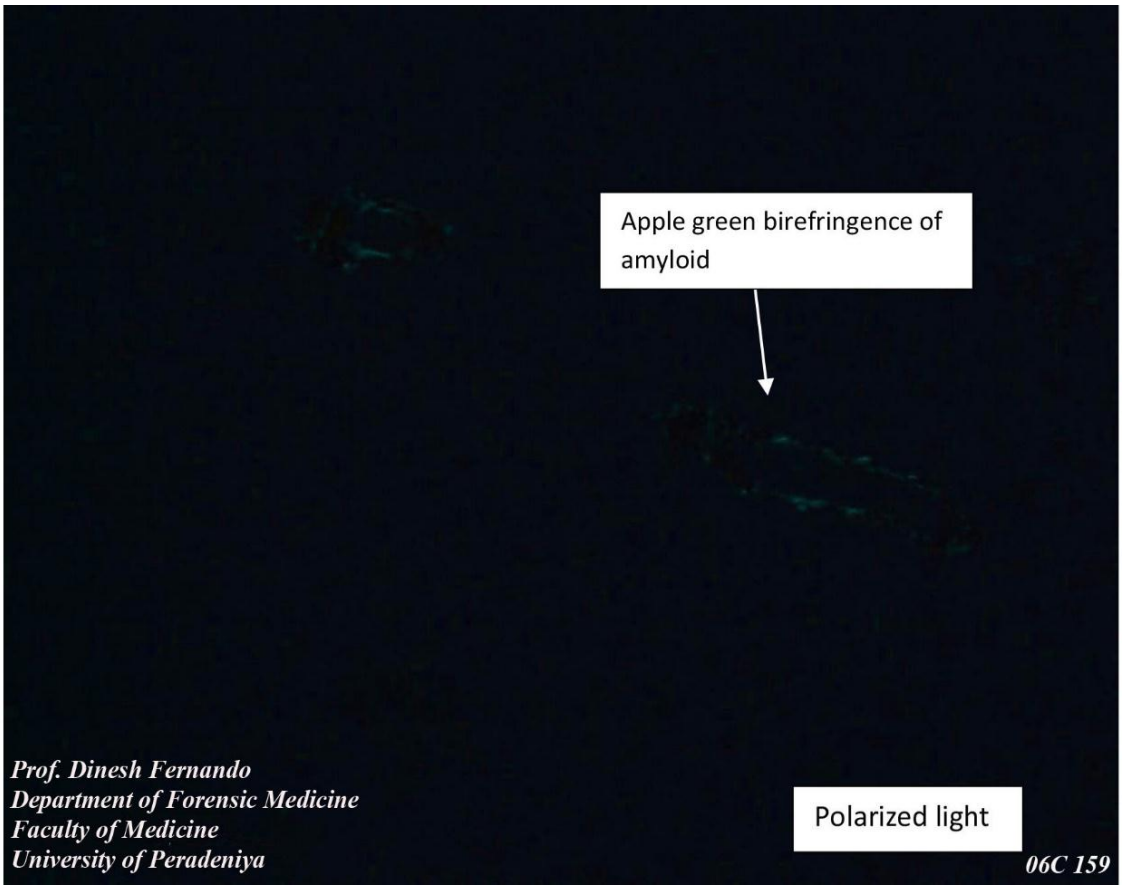


Figure 11: Amyloid angiopathy visualized under polarized light

- Amyloid angiopathy is also a common finding in Alzheimer's disease.
- Amyloid deposits in the vessel walls can be demonstrated by Congo red stained sections visualized under polarized stain. Amyloid shows an apple green birefringence with polarized light.

Cause of death

Scalds involving over 50 % of the body in a person with advanced Alzheimer's disease

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CEREBRAL INFARCTION

The brain is a highly aerobic organ and receives 15% of the resting cardiac output and consumes 20% of total body oxygen: therefore, a constant supply of blood is required, which is maintained by autoregulation of cerebral vascular resistance. The brain can be deprived of oxygen by hypoxia or ischaemia, which may be, either, transient or permanent. There are two types of acute ischaemic injury; global cerebral ischaemia and focal cerebral ischaemia. Global cerebral ischaemia occurs when there is a general reduction of cerebral perfusion as in shock, cardiac arrest, profound hypoglycaemia etc. Focal cerebral ischaemia is the cessation of blood flow to a localized area of the brain, either due to large vessel or small vessel disease.

Infarcts can be divided into two broad groups based on their macroscopic and radiologic appearances; non-haemorrhagic infarcts (resulting from acute vascular occlusions) and haemorrhagic infarcts (resulting from reperfusion of ischemic tissue, either through collaterals or after dissolution of emboli).

Occlusion of the vessels causes brain ischaemia which is followed by infarction. The infarcted region is surrounded by a swollen area which is known as "ischaemic penumbra". Though it is structurally intact, functions are affected. Due to the hypoxia to the brain tissue, a fall in cellular ATP with the release of glutamate leads to a release of free radicals. These alterations lead to inflammatory damage, necrosis and apoptotic cell death, finally leading to neuronal death.

Embolic infarctions are commoner than thrombosis. Cardiac mural thrombi are the major source of emboli, with valvular diseases and atrial fibrillation being predisposing factors. Thromboemboli arise from atheromatous plaques, mainly, within the carotid arteries or aortic arch. Emboli of venous origin can enter the arterial circulation through defects in the cardiac circulation. Emboli tend to lodge mainly in branching points or in stenotic areas of the vessels. Thrombotic occlusions mainly occur near atherosclerotic sites; commonly seen near the carotid bifurcation, the origin of the middle cerebral artery, and at either end of the basilar artery. The territory of the middle cerebral artery – a direct extension of the internal carotid artery – is the most frequently affected area due to embolic infarction.

'Stroke' is the collection of the well-recognized cluster of neurological signs and symptoms of cerebral infarction. The manifestations vary depending on the infarct site and extent. When the insult is mild, there may be only a transient post ischaemic confusional state, with eventual, complete recovery. In severe global cerebral ischemia, widespread neuronal death occurs, irrespective of regional vulnerability. In some cases, it may lead to brain death, including evidence of diffuse cortical injury. Cerebral infarction is typically caused by infarction in the internal capsule, following thromboembolism in the lenticulostriate branch of the middle cerebral artery.

In global cerebral ischemia, the brain is swollen, with wide gyri and narrowed sulci with poor demarcation in between. The irreversible ischemic injury is categorized into three stages according to the histopathological changes which occur with time. Early changes occur within 12 to 24 hours from the injury. It includes acute neuronal cell change (red neurons) and neutrophilic infiltration. Subacute changes occur from 24 hours to 2 weeks, with tissue necrosis, influx of macrophages, vascular proliferation, and reactive gliosis. Gliosis is a change in glial cells in response to brain injury causing

proliferation or hypertrophy of several different types of glial cells, including astrocytes, microglia, and oligodendrocytes. The third stage is the "repair" which is seen after 2 weeks. It is characterized by removal of all necrotic tissue; thus, loss of the organized structure of the brain tissue is seen. Neuronal loss and gliosis in the neocortex typically are uneven, while some layers are preserved, making a pattern termed as pseudolaminar necrosis.

Border zone or watershed infarcts are wedge shaped areas of infarction that occur in regions at the most distal portions of arterial territories. The border zone between the anterior and the middle cerebral artery distributions is at the greatest risk. Damage to this region produces a band of necrosis over the cerebral convexity.

During the first 6 hours of a non-haemorrhagic infarct, no macroscopic changes are seen. But after 48 hours, the tissue becomes pale, soft, and swollen. The brain turns gelatinous and friable from 2 days to 10 days. The boundary between normal and abnormal tissue becomes more distinct, due to the oedema resolving in the adjacent viable tissue. Liquefaction starts from day 10 to week 3, leaving a fluid-filled cavity lined by dark grey tissue. This gradually expands as dead tissue is resorbed with time.

In the cerebral cortex, the cavity is separated from the meninges and subarachnoid space by a gliotic layer of tissue. This layer consists of a dense tissue of glial fibres with new capillaries and perivascular connective tissues. The pia and arachnoid are not affected and do not contribute to the healing process.

The macroscopy and microscopy of a haemorrhagic infarct is the same as of the non-haemorrhagic infarcts, with the addition of blood extravasation to the brain tissue, while intracerebral haematomas are observed in extensive haemorrhage.

History

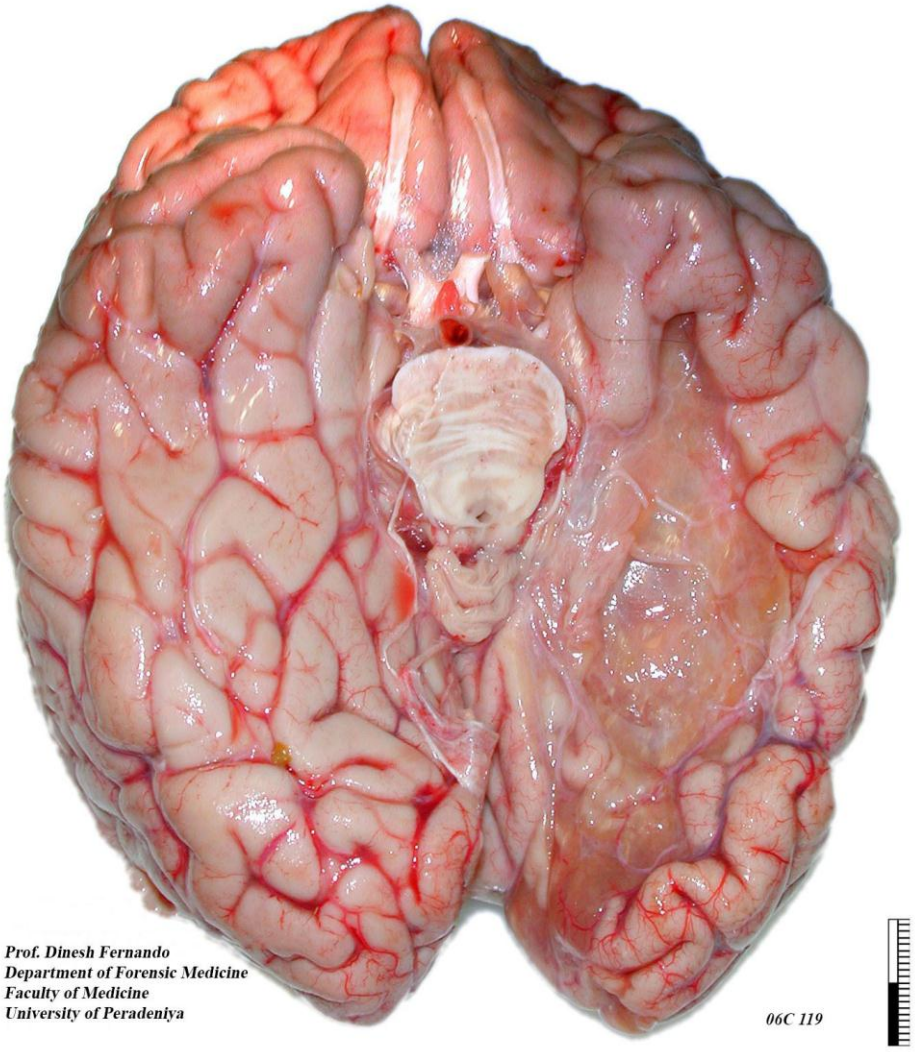
A 77-year-old male who had several strokes over the past six years and had pancreatitis for the past four years presented to the local hospital with a productive cough of a few days and severe abdominal pain, especially in the right upper quadrant. He had vomited bile. Investigations showed raised amylase. He was tachypnoeic with a respiratory rate of 36. The blood pressure had been 120/60 & GCS was 13/15. His condition had deteriorated and he had transferred to a tertiary care hospital ICU on the evening prior to his death. During transfer in the ambulance he had increasing respiratory distress and became more tachypnoeic with saturation being in the low to mid 90s. He had been ventilated in the ICU for 19 hours. Gradually, his condition worsened and treatment was subsequently withdrawn.

Internal Examination

Central Nervous System: A gelatinous area was present on the inferior aspect of the left occipital lobe close to the midline. Multiple atheromatous plaques were present in the Circle of Willis which had dilated vessels. Multiple sections of the cerebral hemispheres revealed an area of old necrosis in the inferior left occipital lobe.

Endocrine system: The thyroid was unremarkable. The pancreas appeared haemorrhagic but macroscopic fibrosis or saponification was not seen.

In order to demonstrate saponification, images from a different case are given. In addition, liver necrosis is also depicted. For images see 'Acute Pancreatitis' in Endocrine system.



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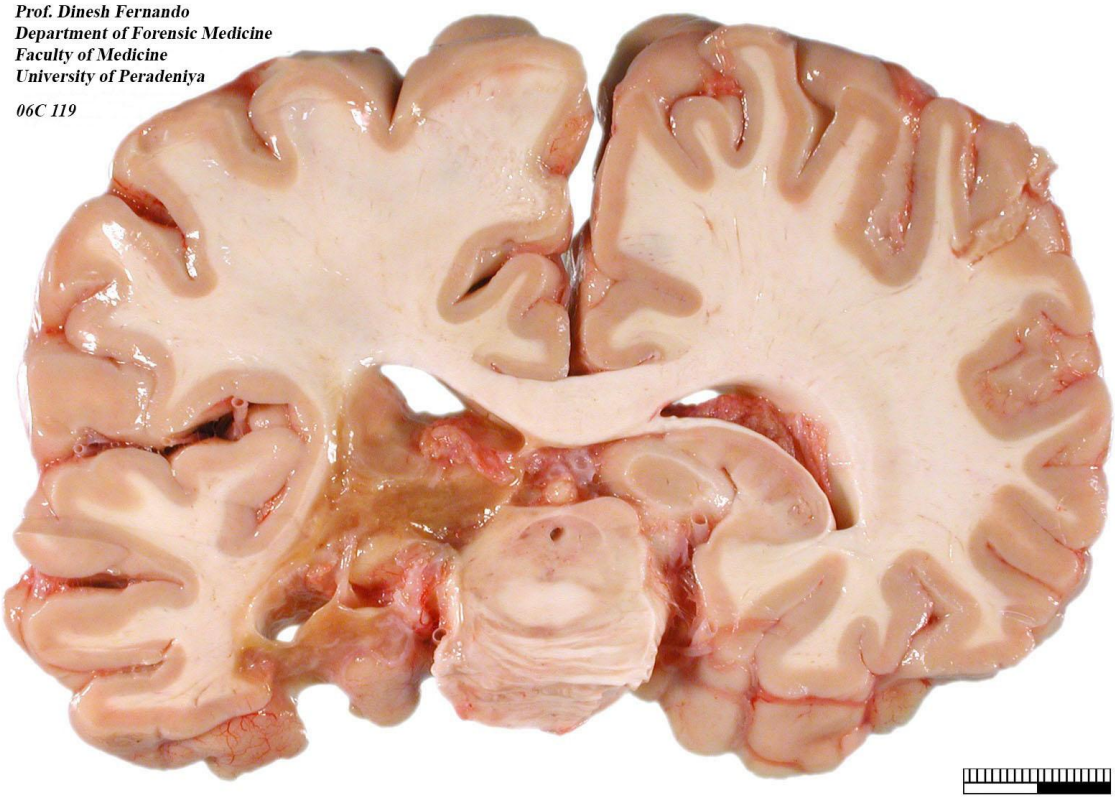
Prof. Dinesh Fernando
Department of Forensic Medicine
Faculty of Medicine
University of Peradeniya
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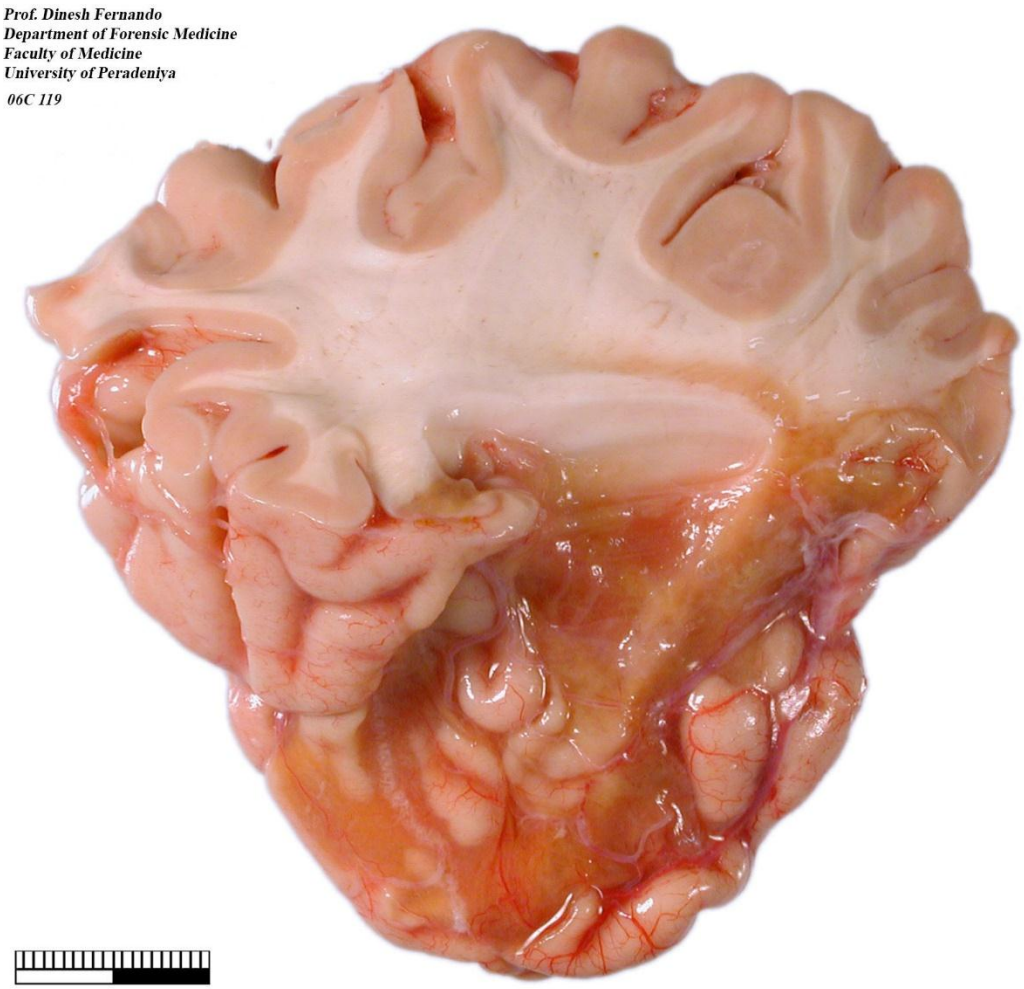
(b)



(c)

Figure 1(a,b,&c) : Large old Cerebral infarction

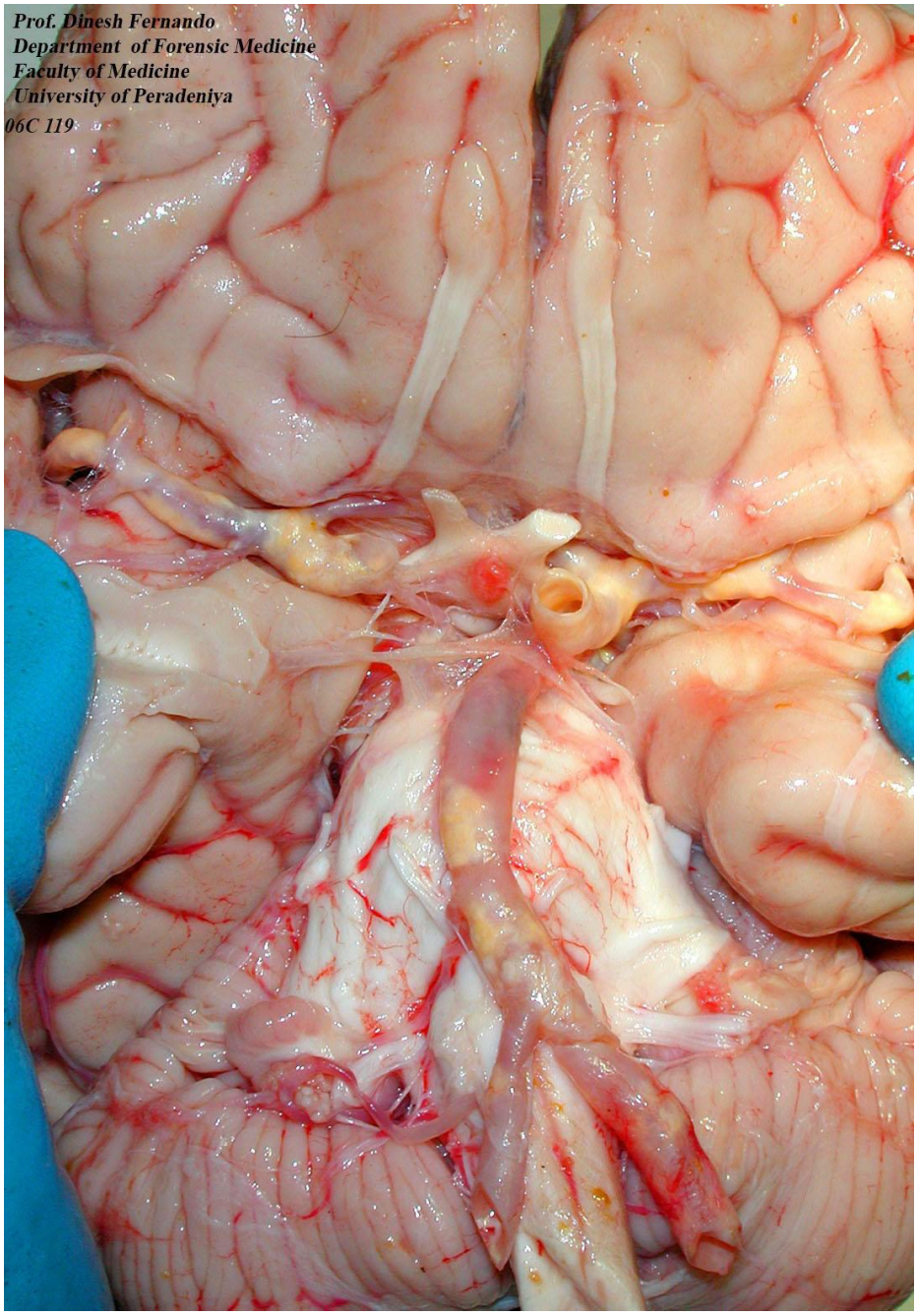


Figure 2: Atheromatous plaques in the circle of Willis

Cause of death

Acute pancreatitis in a person with previous cerebral infarction

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BERRY ANEURYSM

Berry aneurysms are the most common type of intracranial aneurysms and are the major cause of non-traumatic subarachnoid haemorrhage. Rupture of berry aneurysms is a relatively common cause of sudden collapse and rapid death. They are located at the branching points of cerebral arteries in the circle of Willis, with approximately 90% found in the anterior circulation. Of them, 40% are seen in anterior cerebral artery and anterior communicating artery and 25% each is seen in the internal carotid artery and middle cerebral artery. The balance 10% is seen in the vertebro basilar artery. Internal carotid artery aneurysms are mostly seen among females and anterior communicating artery aneurysms are more commonly seen in males. Chances of intra cranial haemorrhage is more in middle cerebral artery aneurysms and intra ventricular haemorrhage is more in anterior cerebral, anterior communicating and vertebro basilar artery aneurysms. The prevalence of aneurysms has been reported as 3 - 4% of all routine autopsies. Though they can be found at any age, they increase in frequency with age.

In early development, a defect in the formation of the media gives rise to a weakness of the vessel wall mainly at branching points. Persistence of incomplete involuted embryonic arteries with residual medial weakness is thought to be the reason for aneurysms which are away from the point of bifurcation. Aneurysms are thin walled protrusions of an artery. The muscular wall and internal elastic laminar are absent beyond the neck of an aneurysm Therefore, the sac is lined only by thickened hyalinised intima, while the adventitia continues over the sac of the aneurysm.

The most common type of cerebral aneurysm is called a saccular, or berry aneurysm. It looks like a “berry” with a narrow stem. Two other types of cerebral aneurysms are fusiform and dissecting aneurysms. A fusiform aneurysm bulges out on all sides circumferentially. Fusiform aneurysms are generally associated with atherosclerosis. A dissecting aneurysm may result from a tear in the inner layer of the artery wall, causing blood to leak into the layers. This may cause a ballooning out on one side of the artery wall or it may block off or obstruct blood flow through the artery. Multiplicity of aneurysms is quite common (around 30%).

There are several risk factors for berry aneurysms; genetic factors like, positive family history, female gender, arterio-venous malformations, polycystic kidney disease, Ehlers-Danlos syndrome, bicuspid aortic valve etc and other factors like hypertension, cigarette smoking, heavy alcohol and caffeine intake. Atherosclerosis plays a secondary role which leads to focal destruction and weakening of the vessels walls.

A cause of medico-legal problems is the association of trauma and sub arachnoid haemorrhage (SAH), either from a ruptured pre-existing ‘berry’ aneurysm or a traumatic aneurysm occurring after blunt, penetrating or iatrogenic head trauma. Traumatic intracerebral aneurysms are rare. The reported incidence is less than 1 per cent, and they are more commonly seen in children. They are caused by direct injury to the vessel wall or by acceleration induced shear forces and more than half of them are associated with skull fracture.

Berry aneurysms usually rupture at the apex. The haemorrhage occurs mainly into the subarachnoid space, while some may also occur into the substance of the brain. Minor leakage from the aneurysm may precede rupture. In such cases, the patient often complains of headache for days or weeks prior to rupture. Patients with spontaneous rupture of a berry aneurysm presents with a sudden excruciating headache which is often described as 'thunder clap headache' with neck stiffness and vomiting. Examination findings include meningism with focal neurological signs. Symptoms may resolve or may progress over hours or days to coma and death. Death is due to generalized vasospasm triggered by the subarachnoid haemorrhage, with resultant ischemic injury to the brain.

At autopsy, typically a fresh subarachnoid bleed with a ruptured aneurysm can be seen. The densest haemorrhage is seen over the base of the brain towards the ventral side, especially in the basal cisterns. Lesser amounts of blood is seen laterally and dorsally. Large pools of blood on the ventral surface of the brain often make it difficult to locate the aneurysm. Fresh bleed appears in bright red colour and if it lasts a week or so, a brownish tinge will appear as the haemoglobin undergoes changes.

History

A 48 year old male soldier was at home on leave and was last seen by his wife at 6.45 a.m. when she left for work. When she returned at 4.00 p.m. the same day, the diseased was found dead sitting at his computer table.

Internal examination

Central Nervous System

A thin SAH was seen over both convexities of the brain and predominantly in the base. A ruptured berry aneurysm measuring approximate 2-3 mm in diameter at the root of the right internal carotid artery was seen. A non-ruptured berry aneurysm was found at the basilar apex (bifurcation of the basilar artery).

Macroscopic examination

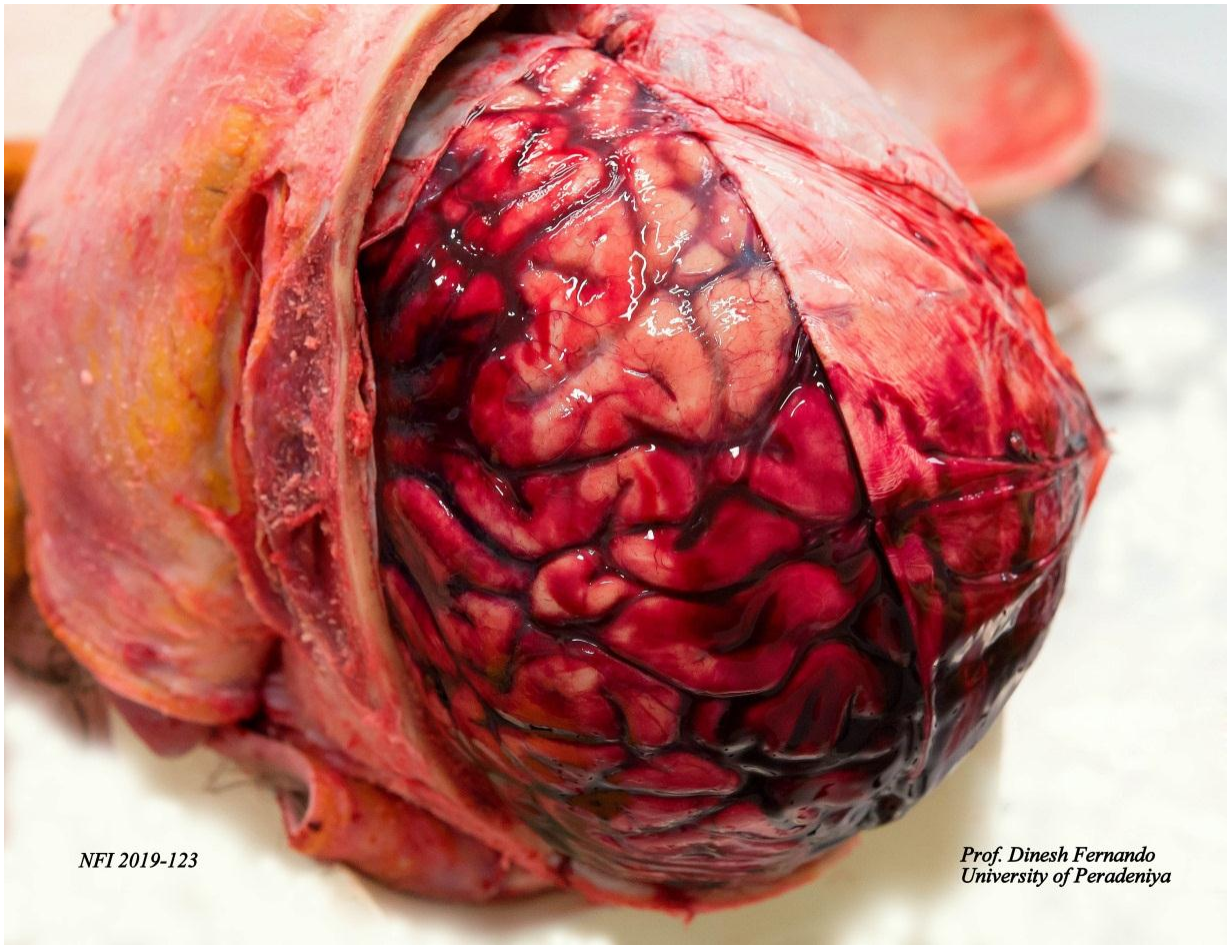


Figure 1: Sub arachnoid haemorrhage in an adult brain with part of the dura everted

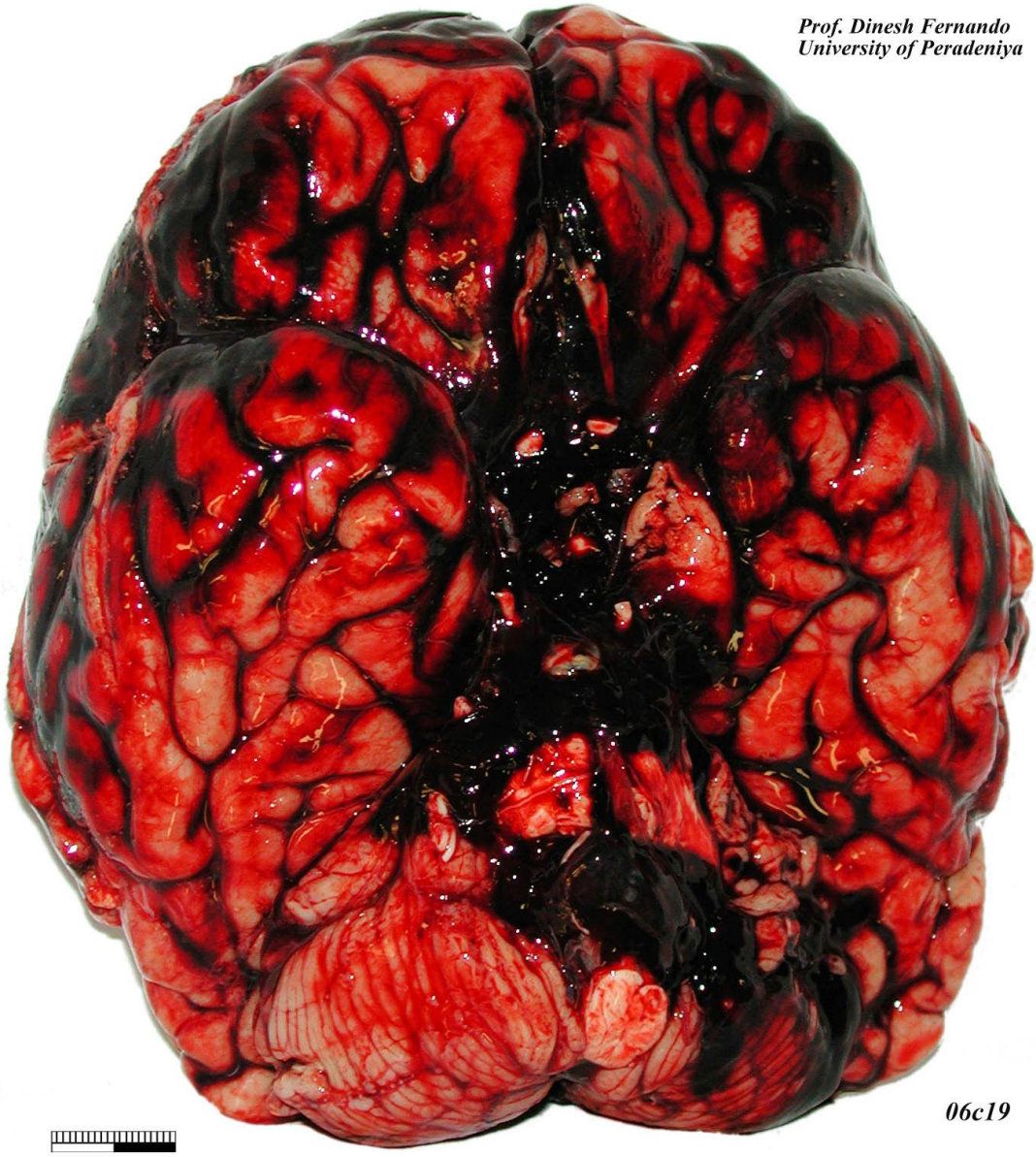
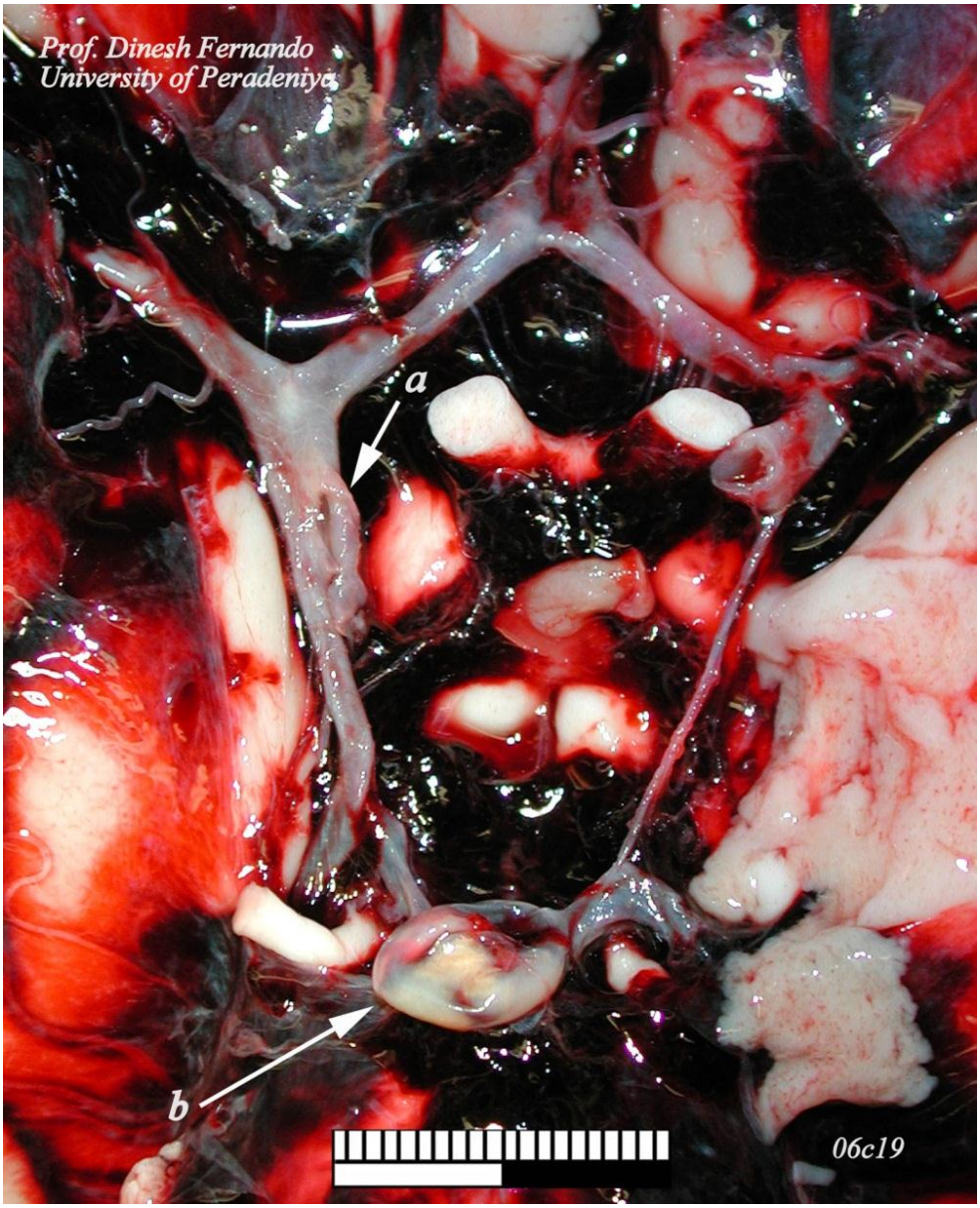


Figure 2: Sub arachnoid haemorrhage seen predominantly at the base of the brain



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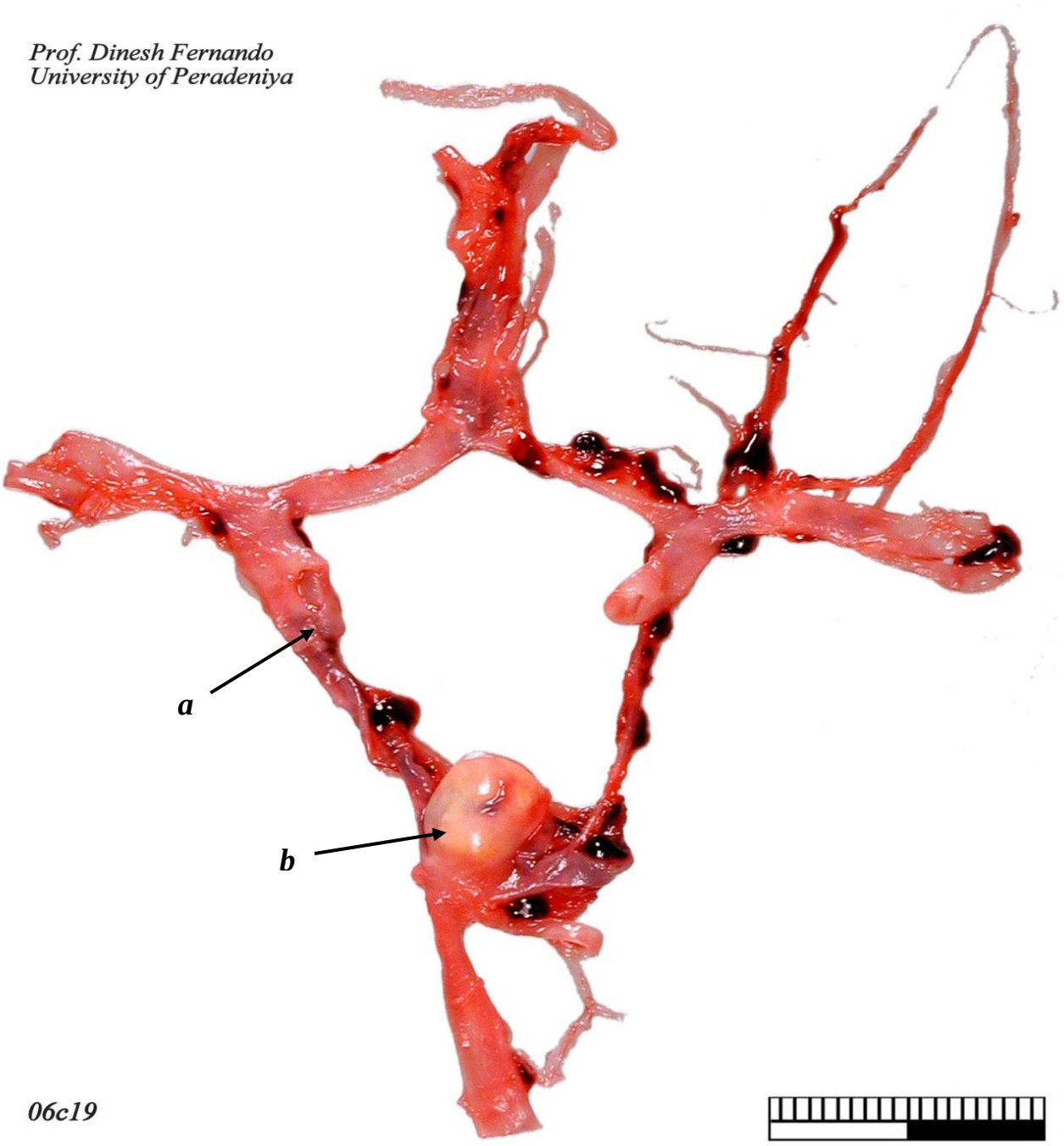
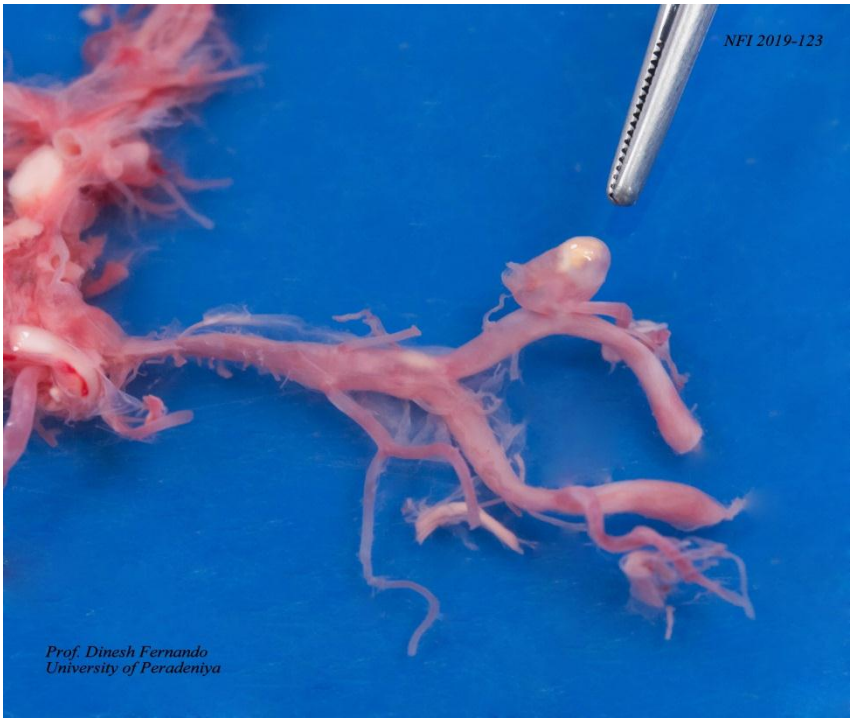


(b)

Figure 3(a, b): Multiple aneurysms in the circle of Willis. Arrow ‘a’ root of the internal carotid artery and arrow ‘b’ bifurcation of the basilar artery (basilar apex)

(a)



(b)

Figure 4(a, b): A non-ruptured vertebral artery aneurysm, which is very rare, from another case

Microscopic Examination

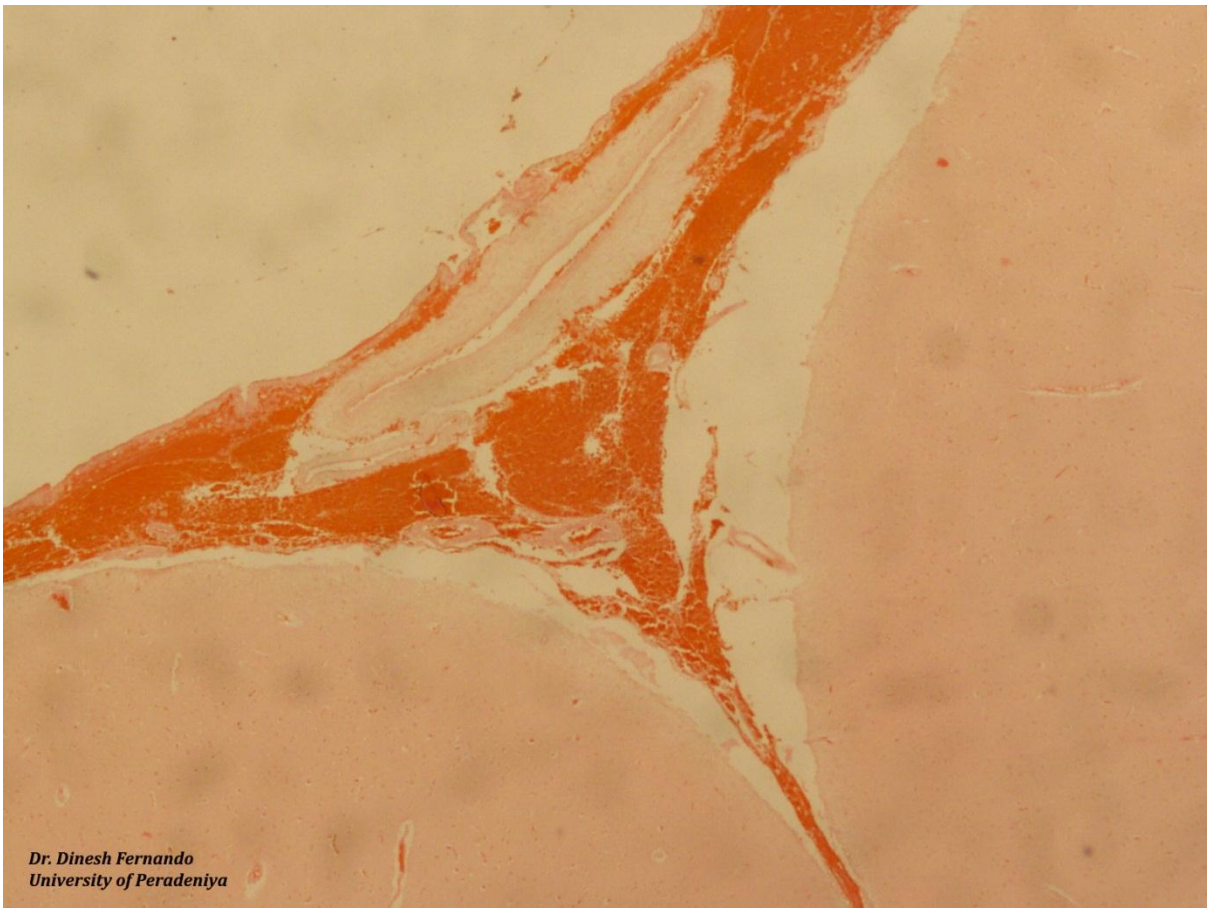


Figure 5: Sub Arachnoid Haemorrhage

Cause of death

Sub Arachnoid Haemorrhage due to ruptured berry aneurysm

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COLOR ATLAS OF

FORENSIC PATHOLOGY

RESPIRATORY SYSTEM

BRONCHOPNEUMONIA

Pneumonia is defined as inflammation of the substance of the lung. Commonest causative organism is bacteria. Pneumonia presents as an acute illness with symptoms such as productive cough with purulent sputum, shortness of breath and fever along with physical signs compatible with consolidation. There are two anatomic patterns called bronchopneumonia and lobar pneumonia. In bronchopneumonia multi lobar patchy consolidation frequently involving bilateral and basal lung parenchyma is seen. The initial infection starts within the bronchi and bronchioles (bronchocentric) and extends into the adjacent alveoli later. The adjacent areas of consolidation are usually hyperaemic and oedematous.

Lobar pneumonia shows uniform distribution of inflammation, usually in a single lobe, which is not bronchocentric. It evolves through four stages called congestion, red hepatisation, gray hepatisation, and resolution. The pleural reaction (fibrinous or fibrinopurulent pleuritis), which may leave fibrous thickening or permanent adhesions is more commonly seen in lobar pneumonia.

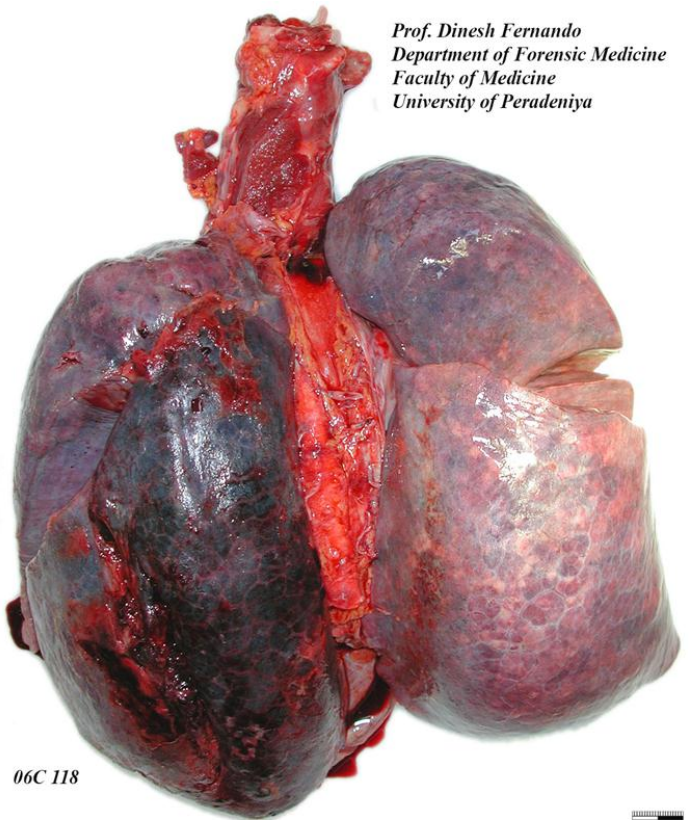
History

A 35-year-old male sustained a head injury with subdural haemorrhage following a fall from a height of 5 to 6 metres. He was in the ICU and had been ventilated. He was unconscious and died after 2 days without regaining consciousness.

Internal examination

Respiratory Tract: The left pleural cavity contained 100 ml and the right pleural cavity contained 50 ml of straw-coloured fluid. There were fresh adhesions between the right lung and the pleural cavity and fibrinous tags were present on the pleural surfaces, lower lobes of both lungs and between lobes. The larynx, trachea and mainstem bronchi had congested mucosal surfaces. The right and left lungs weighed 1,230 grams and 1,100 grams respectively. The pulmonary parenchyma manifested a variegated appearance and had pus like material exuding from the cut surfaces of the bronchi. Both lower lobes were firm to touch.

Macroscopic Examination



(a)

Prof. Dinesh Fernando
Department of Forensic Medicine
Faculty of Medicine
University of Peradeniya

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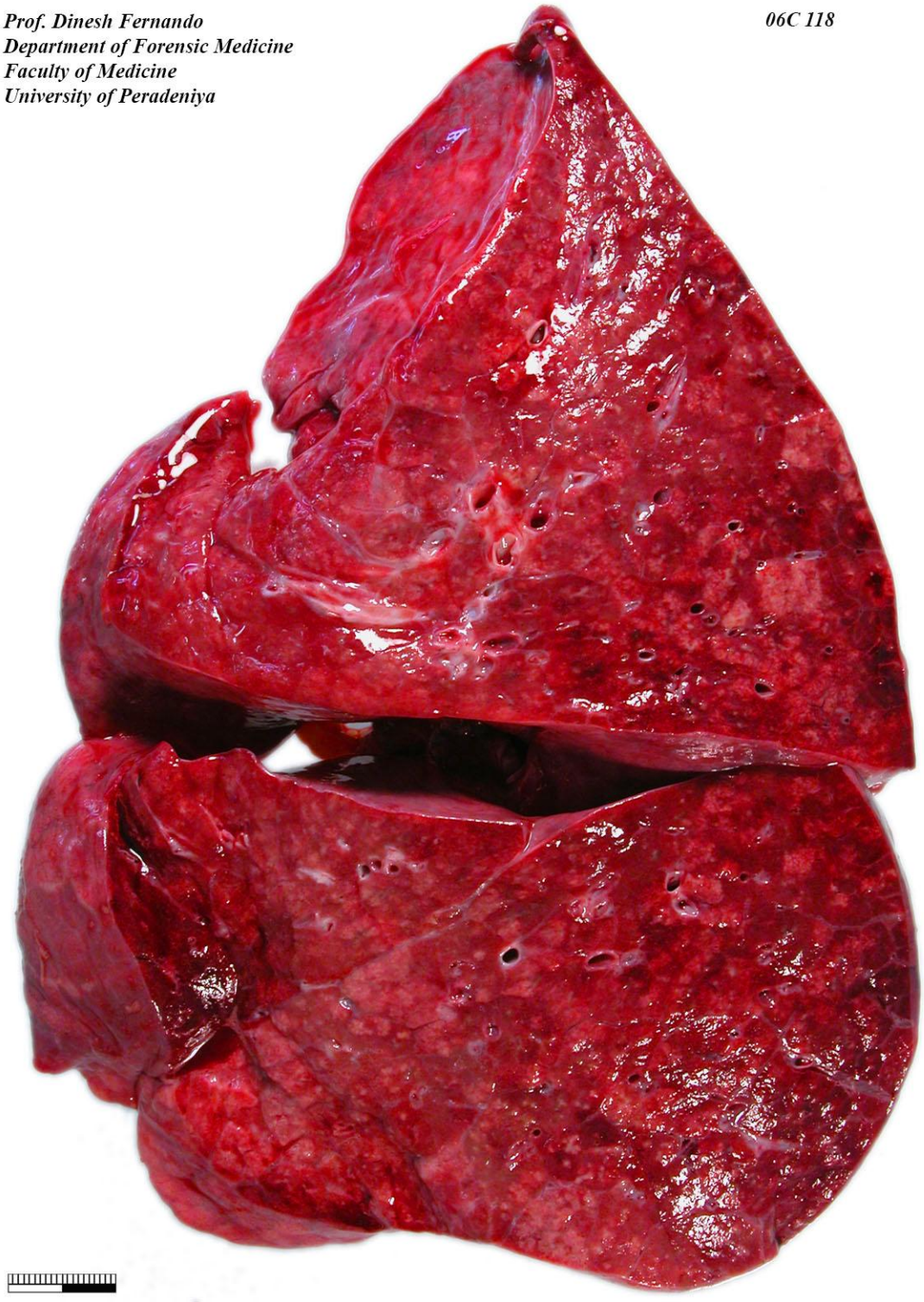

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(c)

Figure 1(a, b & c): Macroscopic appearance of the lungs

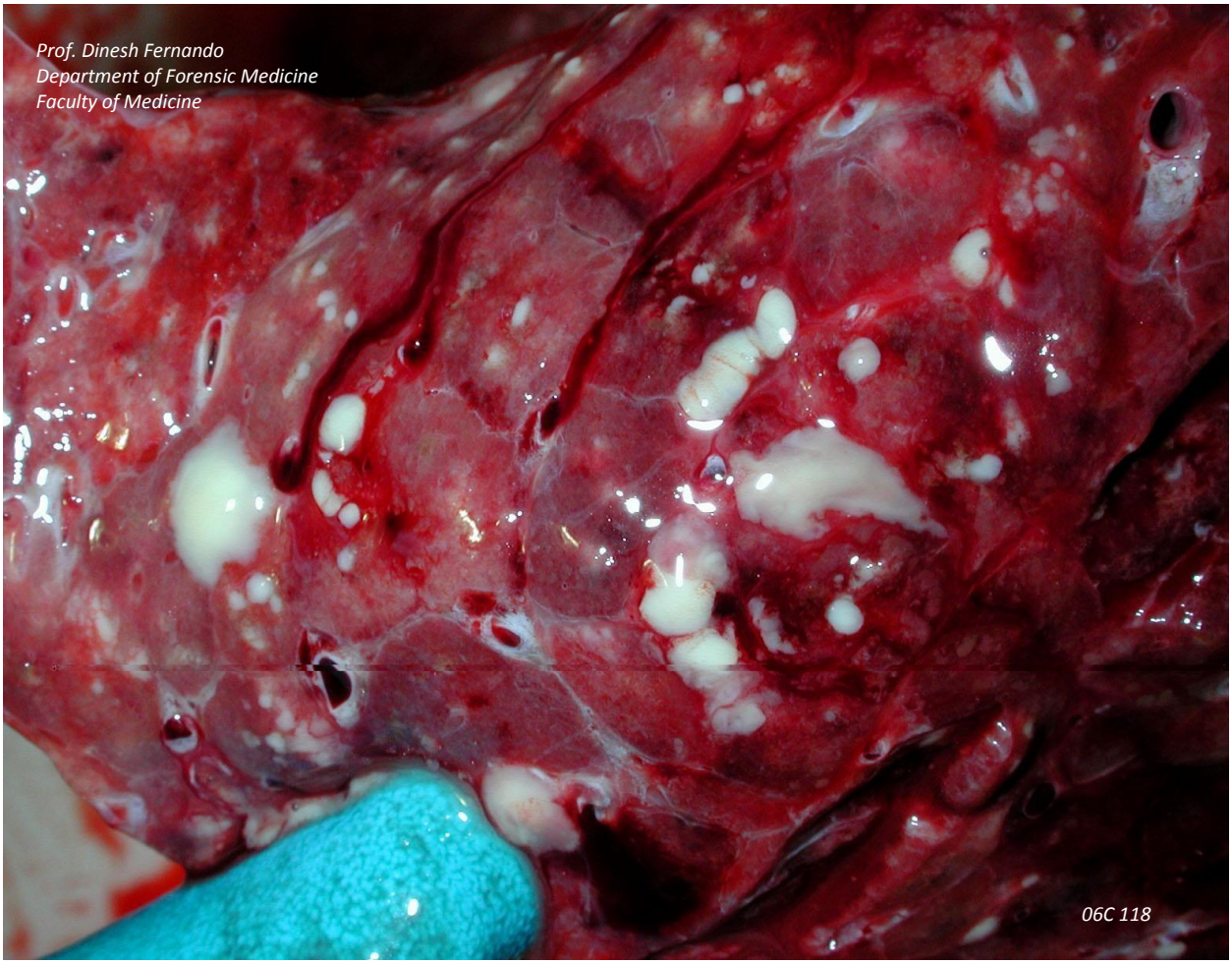

Figure 2: Congested parenchyma with pus-like material from bronchi

Microscopic examination

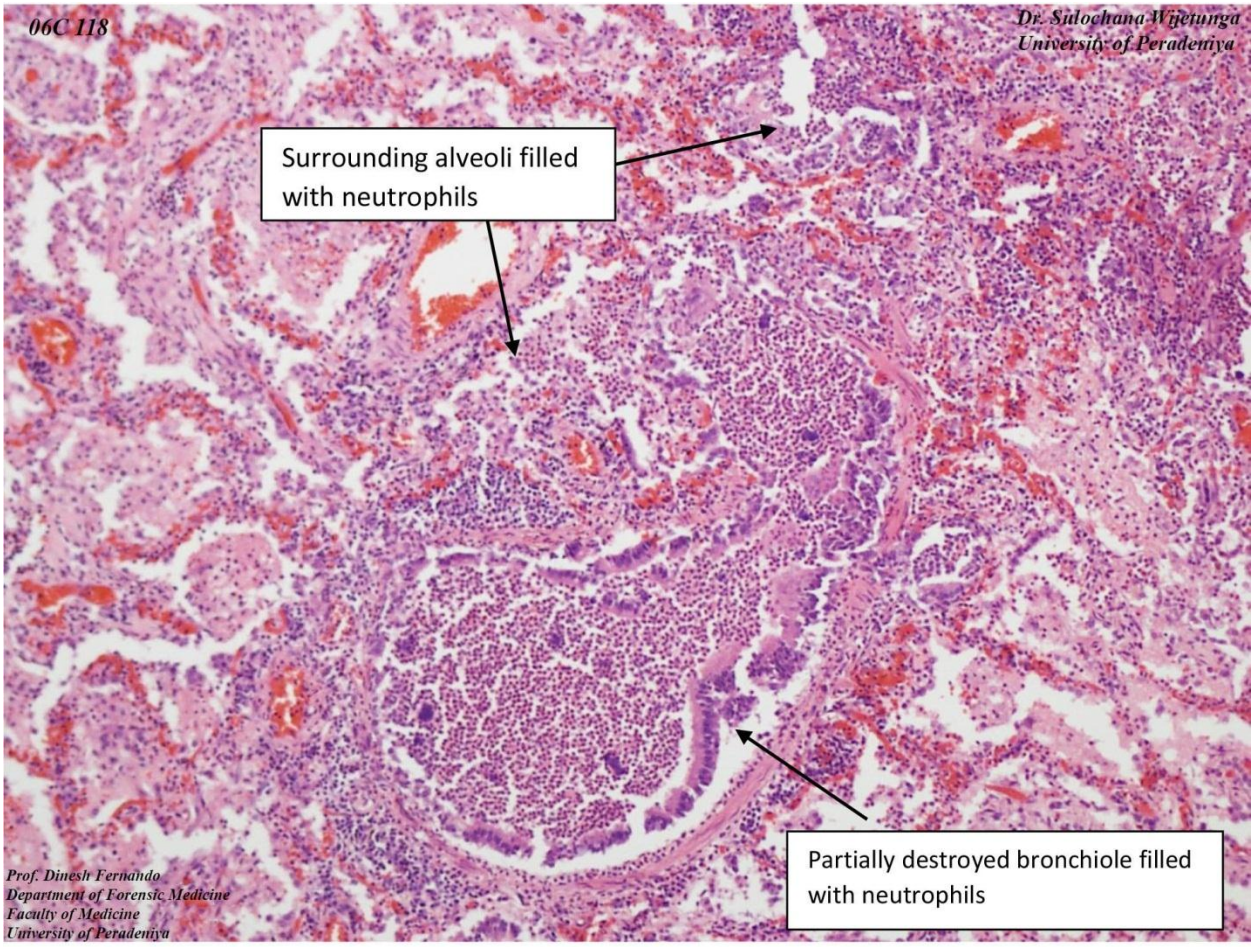

Figure 3: A bronchiole partially destroyed by a neutrophil infiltration and the surrounding alveoli filled with neutrophils

Bronchopneumonia is characterized by bronchocentric acute inflammation in the lung parenchyma. Bronchopneumonia has a patchy distribution. Whereas, lobar pneumonia shows uniform distribution of acute inflammation in the affected area and is not bronchocentric.

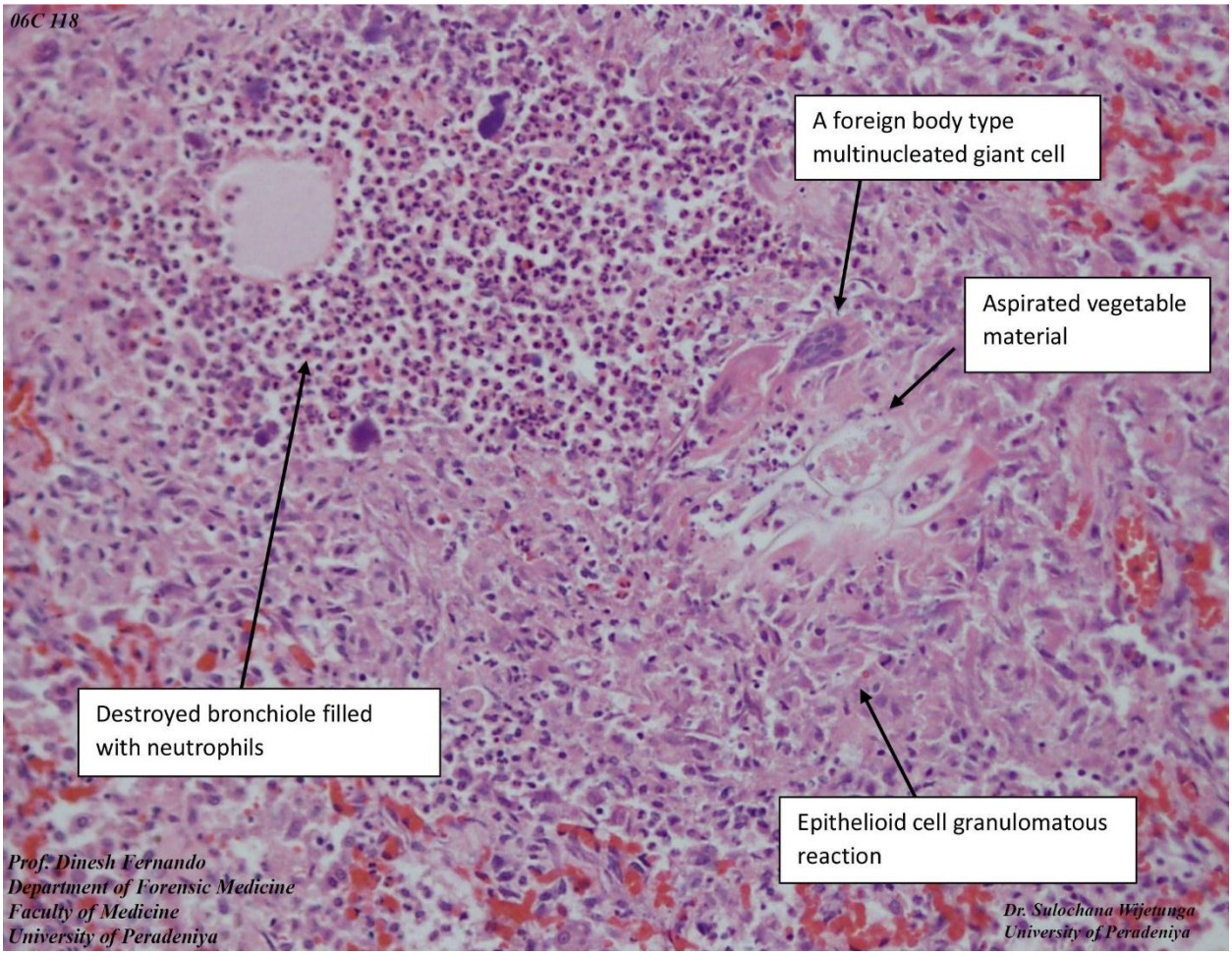

Figure 4: A completely destroyed bronchiole by the inflammation

- Bronchiolar lumen is filled with neutrophils. The wall is destroyed by a foreign body type granulomatous reaction to aspirated material.

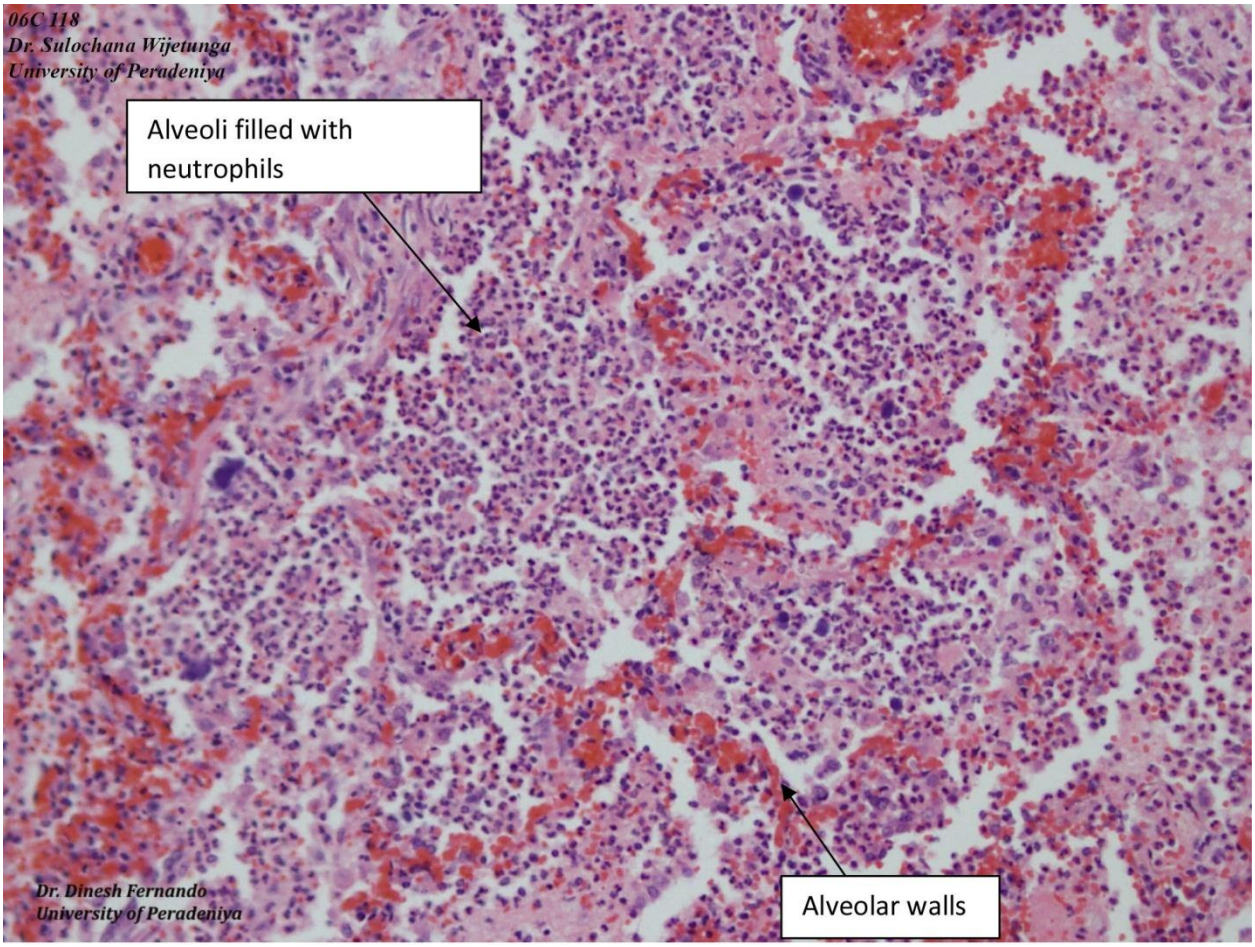

Figure 5: The alveoli surrounding the inflamed bronchiole (high power)

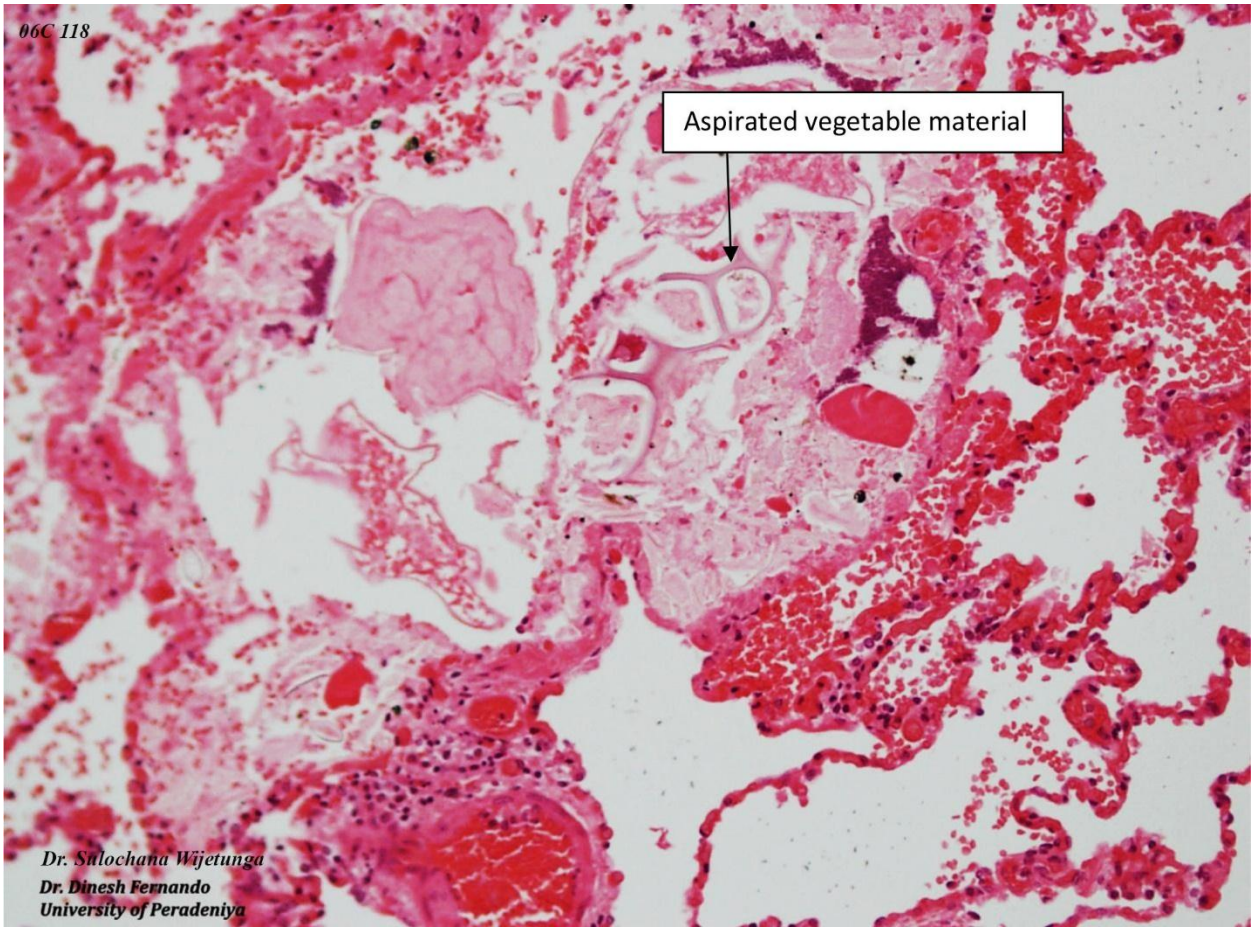

Figure 6: Aspirated vegetable material in the lung parenchyma. The bronchopneumonia in the diseased is an aspiration pneumonia.

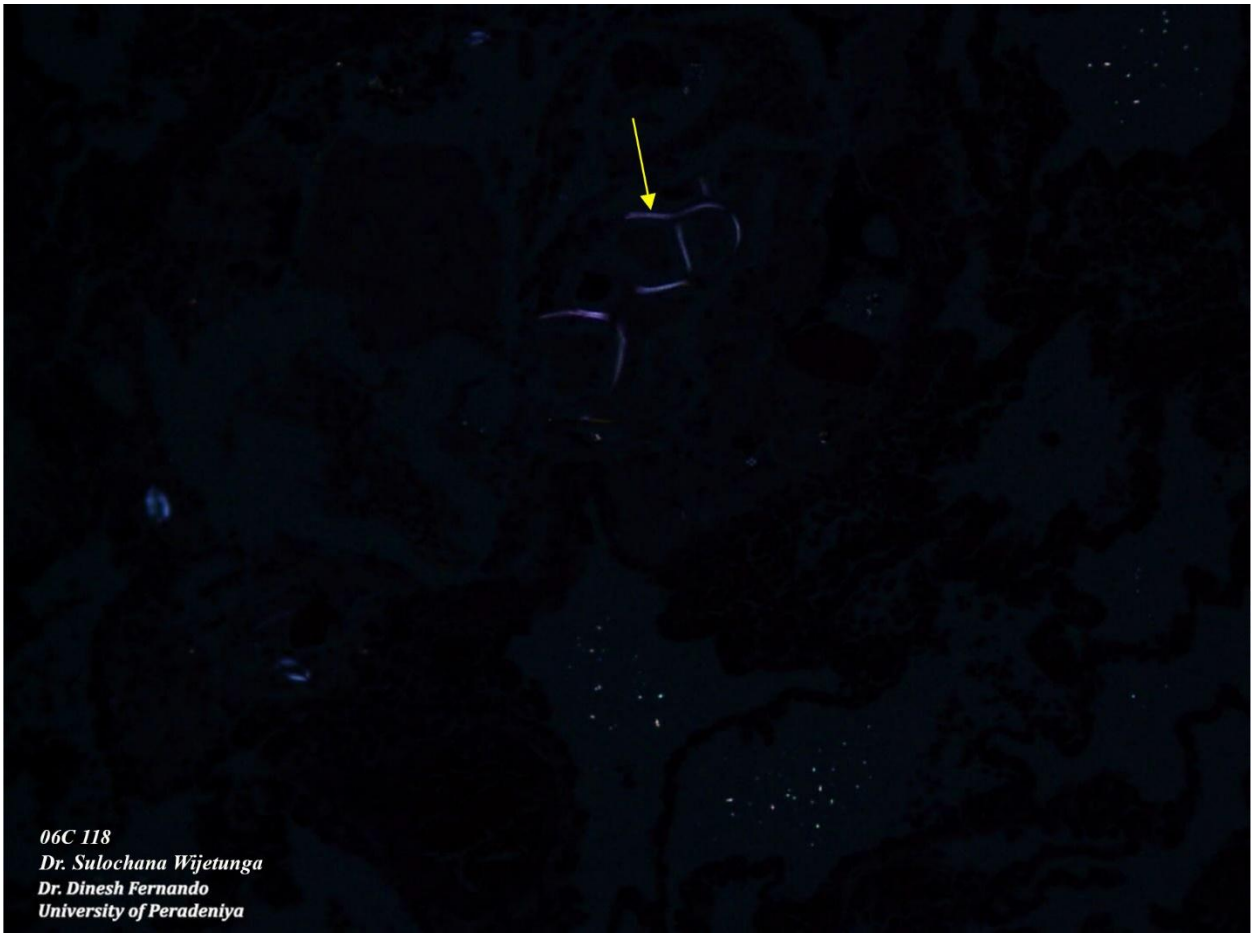

Figure 7: Aspirated vegetable material highlighted by polarized light

Cause of death

Bronchopneumonia due to prolonged unconsciousness due to head injury sustained from a fall from a height.

In order to demonstrate the aspects of Lobar pneumonia lung, microscopic images of a different case are illustrated as follows.

Lobar Pneumonia

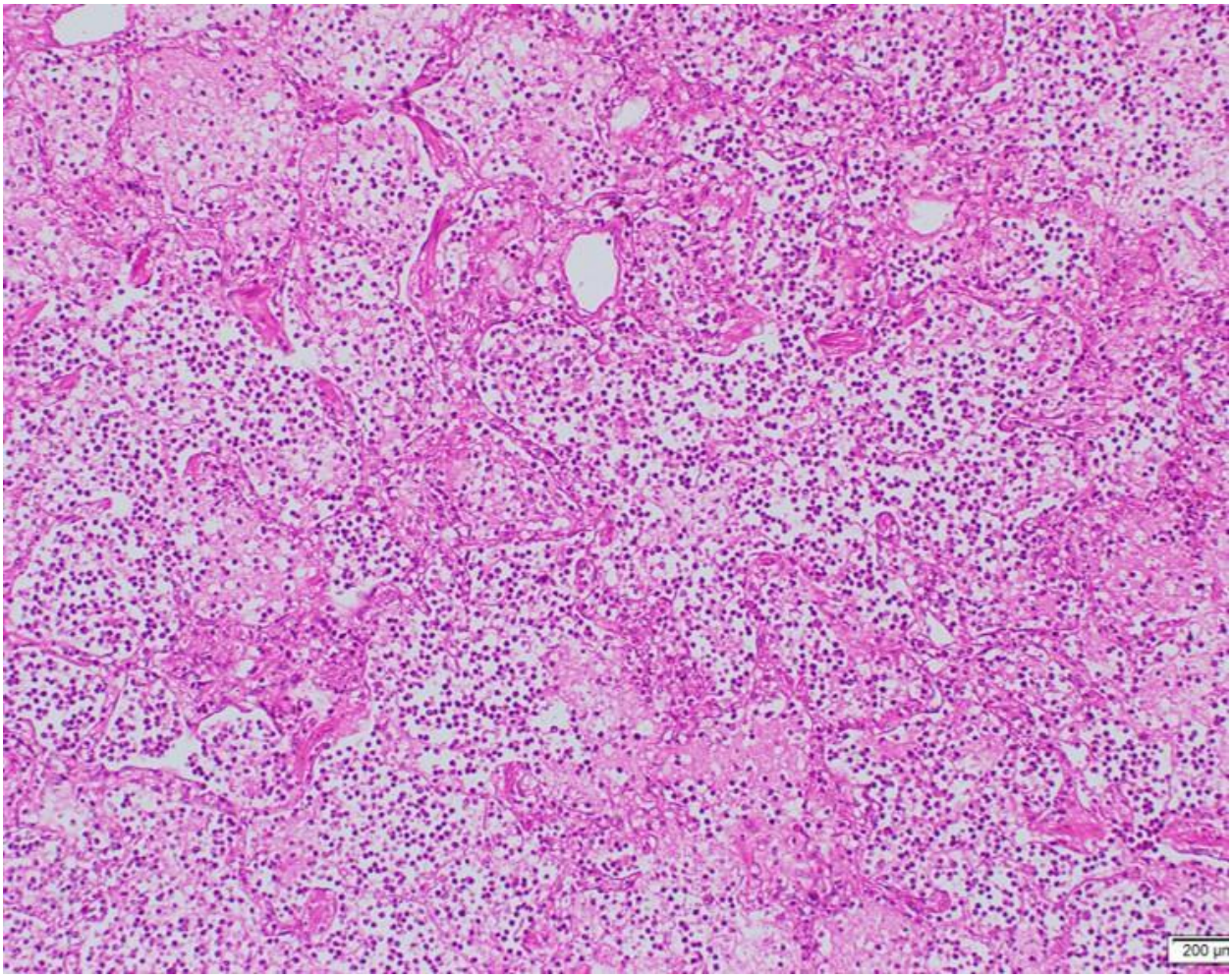

Figure 1: Diffuse lobar pneumonia

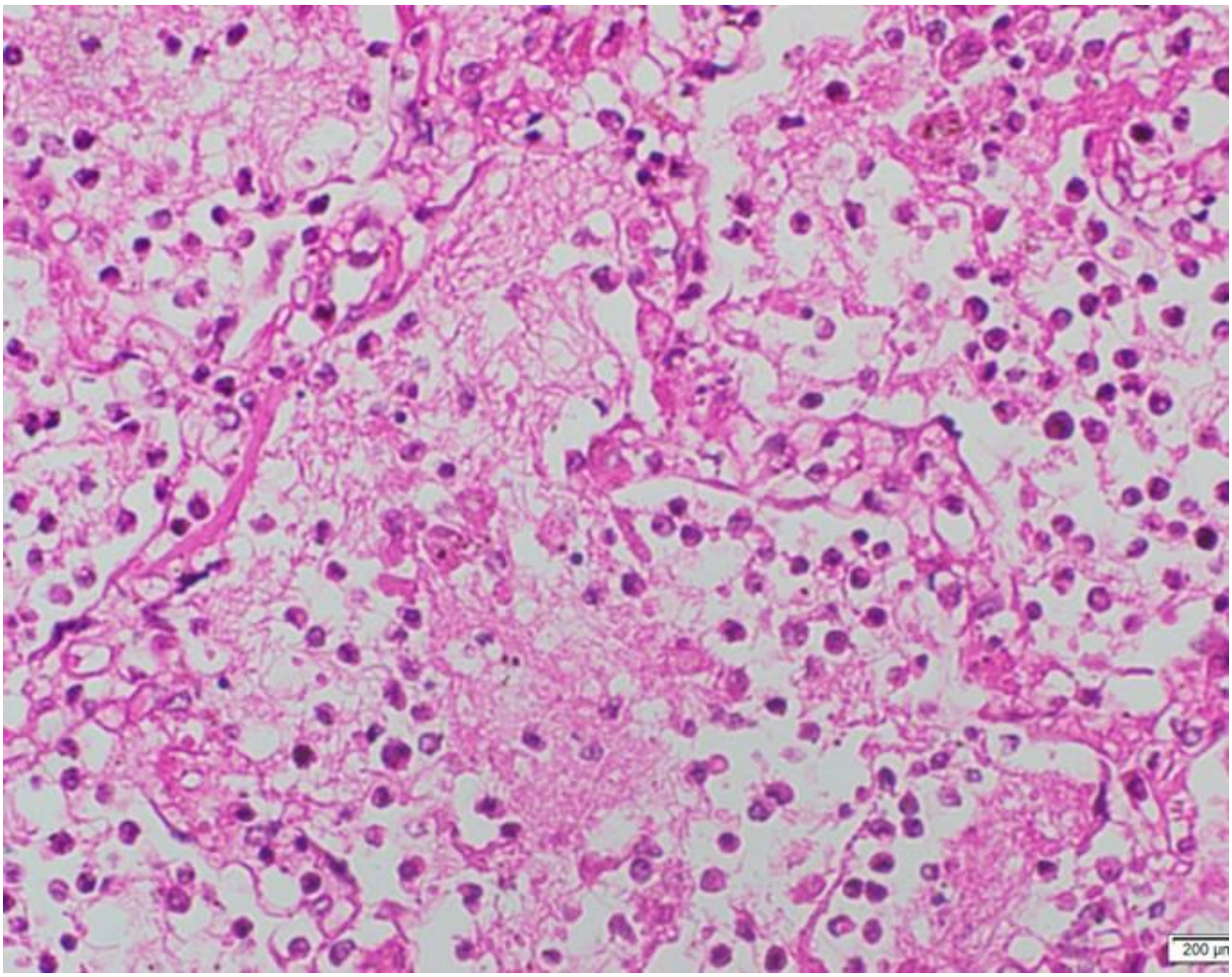

Figure 2: Diffuse lobar pneumonia (High power)

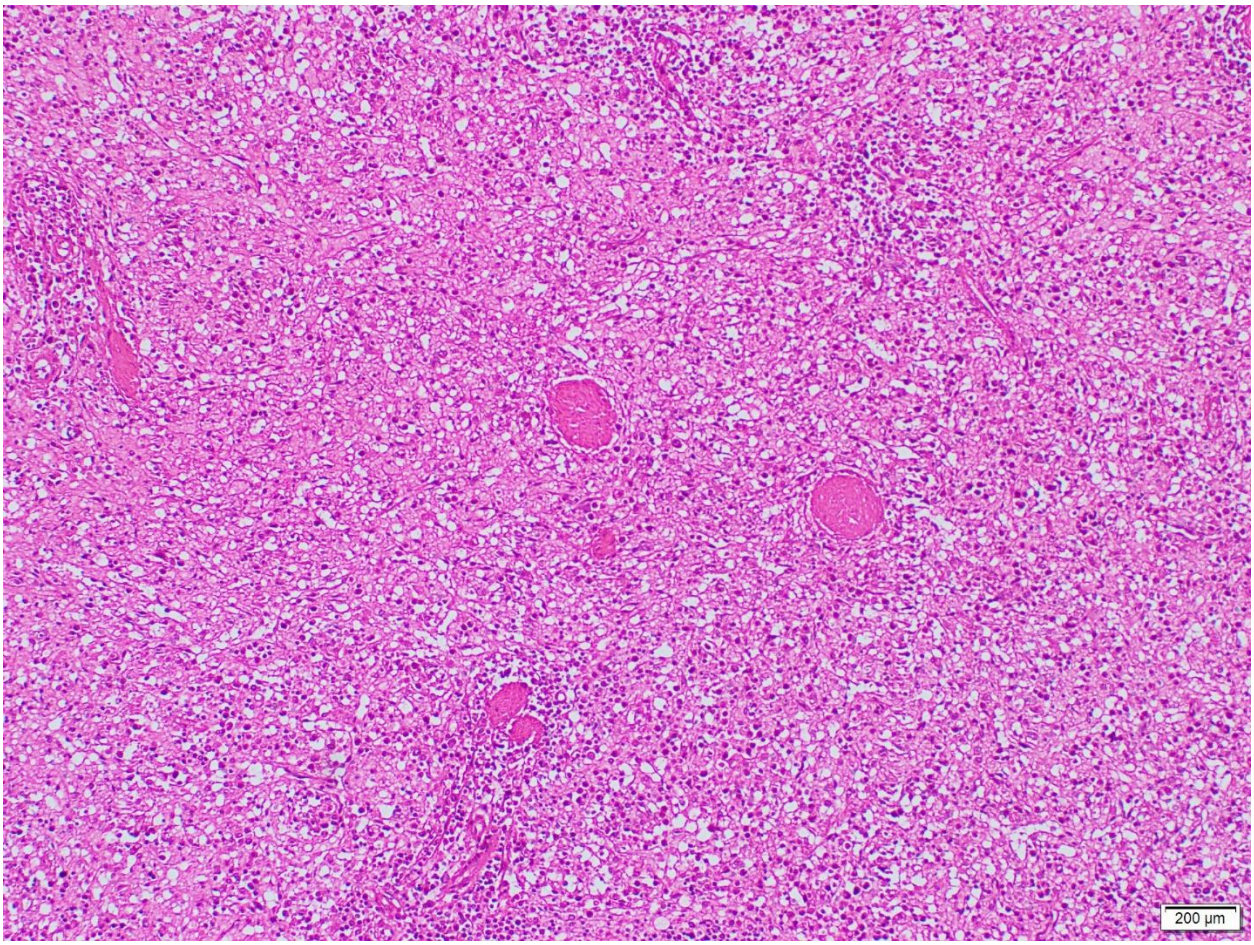

Figure 3: Thrombi lung

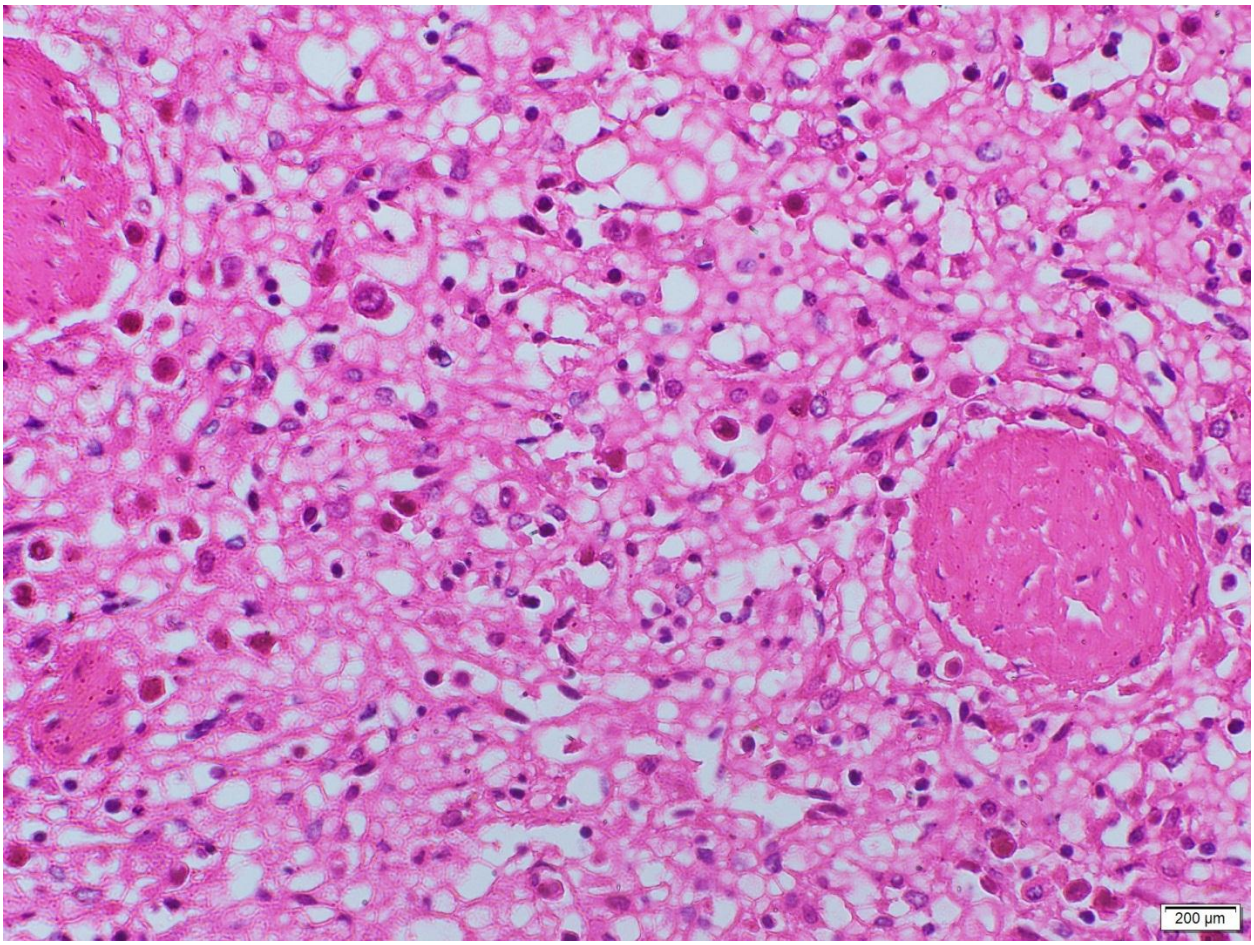

Figure 4: Thrombi lung (high power)

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.
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STAPHYLOCOCCAL PNEUMONIA

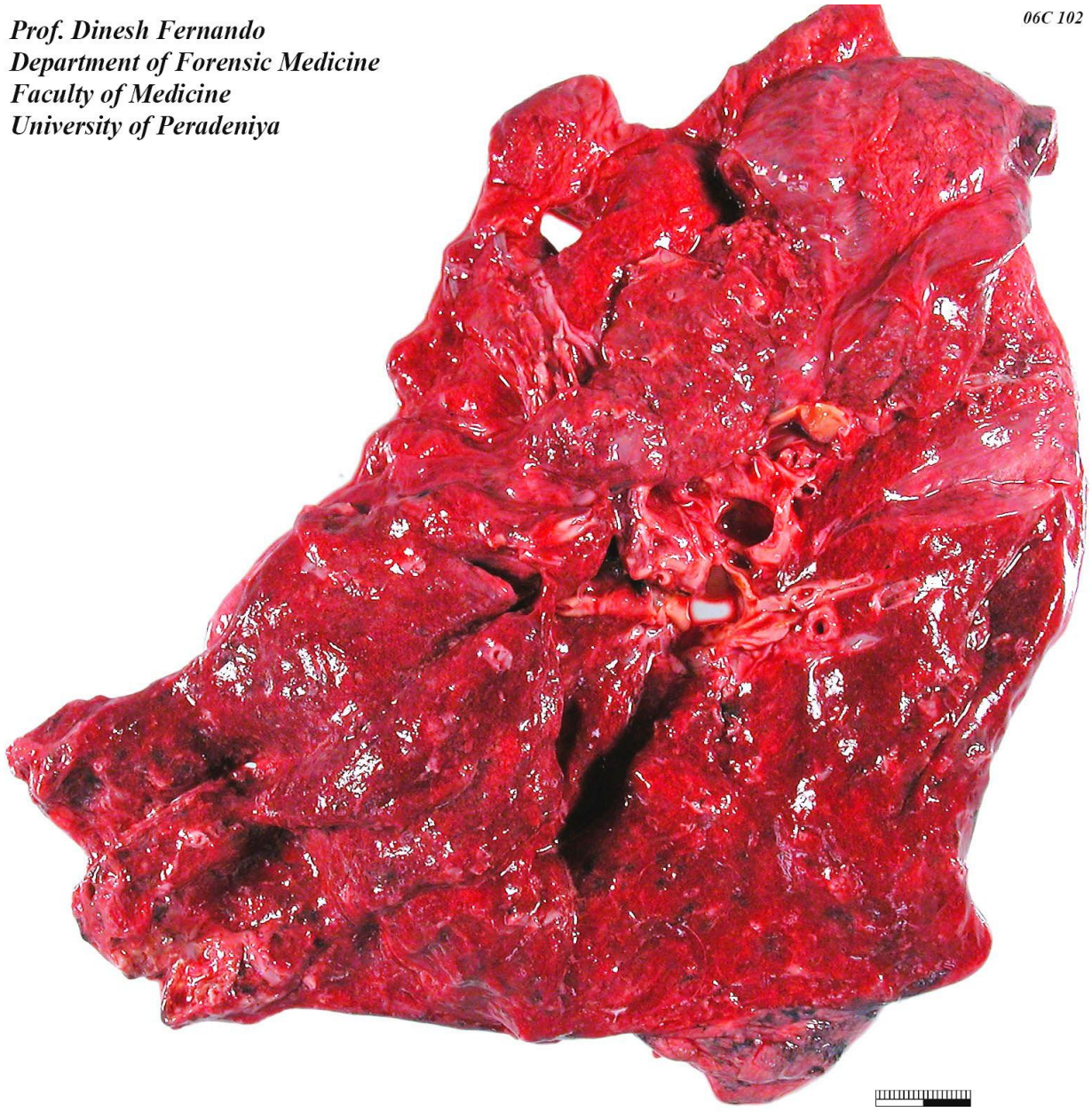
Staphylococcus aureus is an important cause of secondary bacterial pneumonia in children and healthy adults following viral respiratory infections. Those with diabetes mellitus, head trauma, and patients in intensive care units are more susceptible. Methicillin-sensitive strains are much commoner, but an increasing incidence of methicillin-resistant disease is now observed. It is associated with many complications such as necrotizing cavitating lesions, empyema with bilateral infiltrates. Especially in injecting drug users, septic emboli containing staphylococci can gives rise to multiple abscesses in the lung.

History

A 67-year-old female who had a past history of asthma, diabetes and angina had been having a productive cough for several days. A day before her death she had been coughing up blood. She was found dead next to her bed lying on her right side. She was last seen alive approximately 12 hours previously.

Internal examination

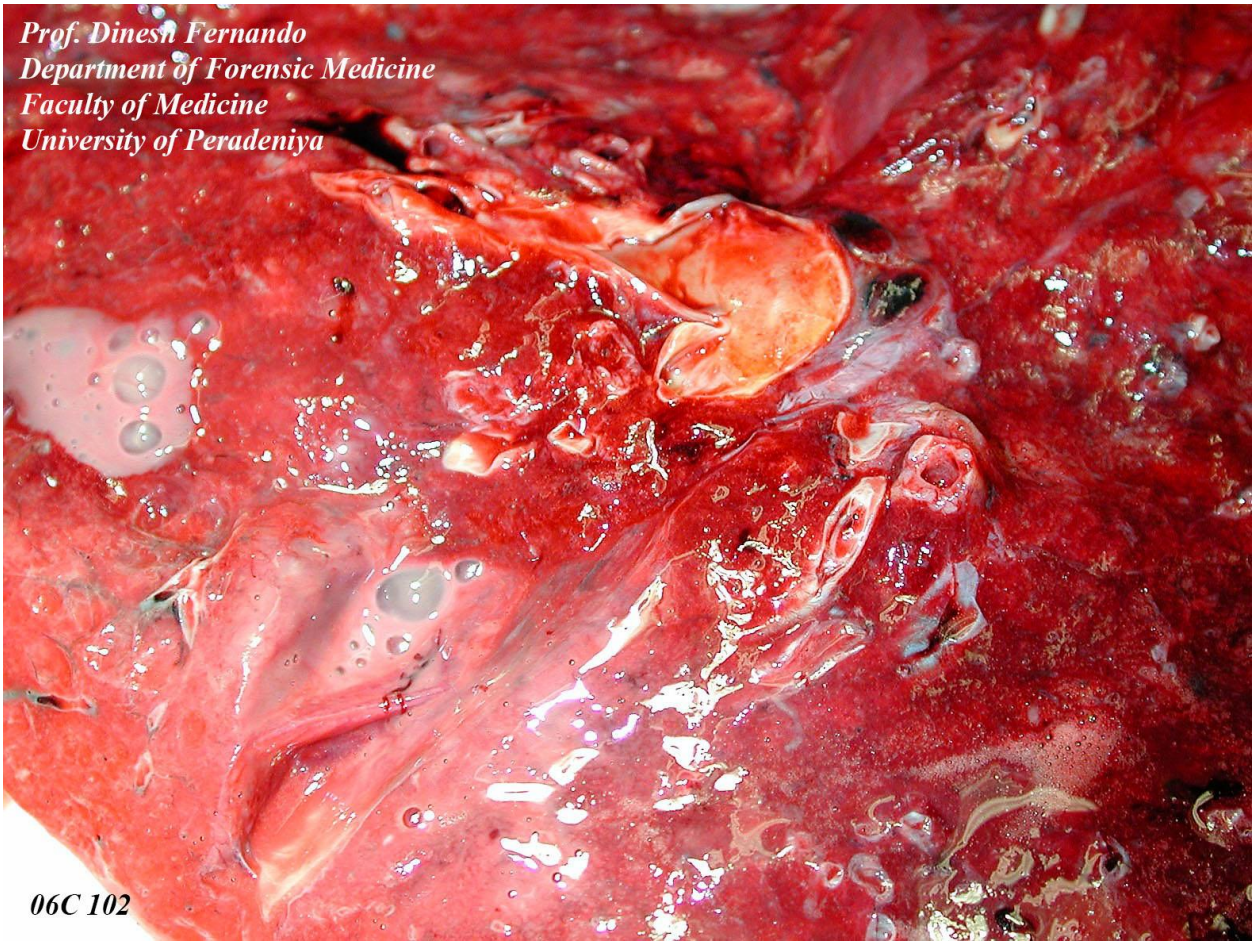
Respiratory Tract: 50 ml of straw-coloured fluid was present in the right pleural space and 100 ml was present in the left. The right lung was adherent to the chest wall. The larynx, trachea and mainstem bronchi had congested mucosal surfaces. The right and left lungs weighed 564 grams and 686 grams respectively. Multiple adhesions were present between the lobes of the right lung, the diaphragm and the chest wall. Multiple bullae were present on both lungs. A calcified circular mass measuring approximately 1cm in diameter was situated sub pleurally on the lower part of the right upper lobe. On cut section, a necrotic centre was present. The rest of the right upper lobe was necrotic and had pus-like material within dilated spaces. The cut section of the left lung revealed multiple dilated spaces within the parenchyma filled with pus-like material. The pulmonary arteries had atheroma. The bronchi were thick-walled and prominent. Gross pulmonary oedema was present.


Prof. Dinesh Fernando
Department of Forensic Medicine
Faculty of Medicine
University of Peradeniya
06C 102

(a)

(b)

Figure 1(a & b): Cut section of lung showing congestion, dilated spaces and pus-like material especially in the lower lobe


Prof. Dinesh Fernando
Department of Forensic Medicine
Faculty of Medicine
University of Peradeniya
06C 102

(a)

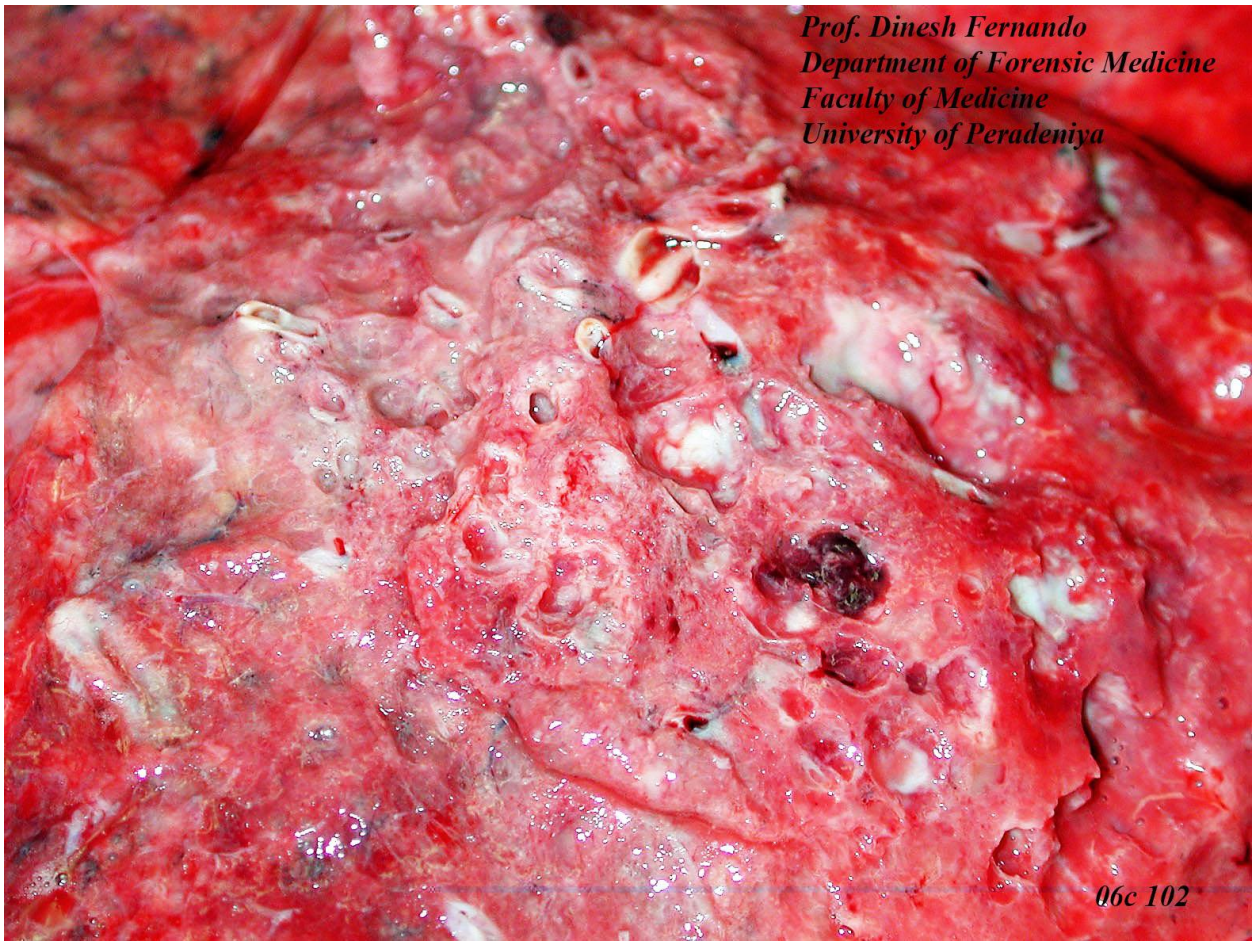


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Figure 2(a & b): Congested parenchyma with pus-like material from bronchi

Microscopic Examination

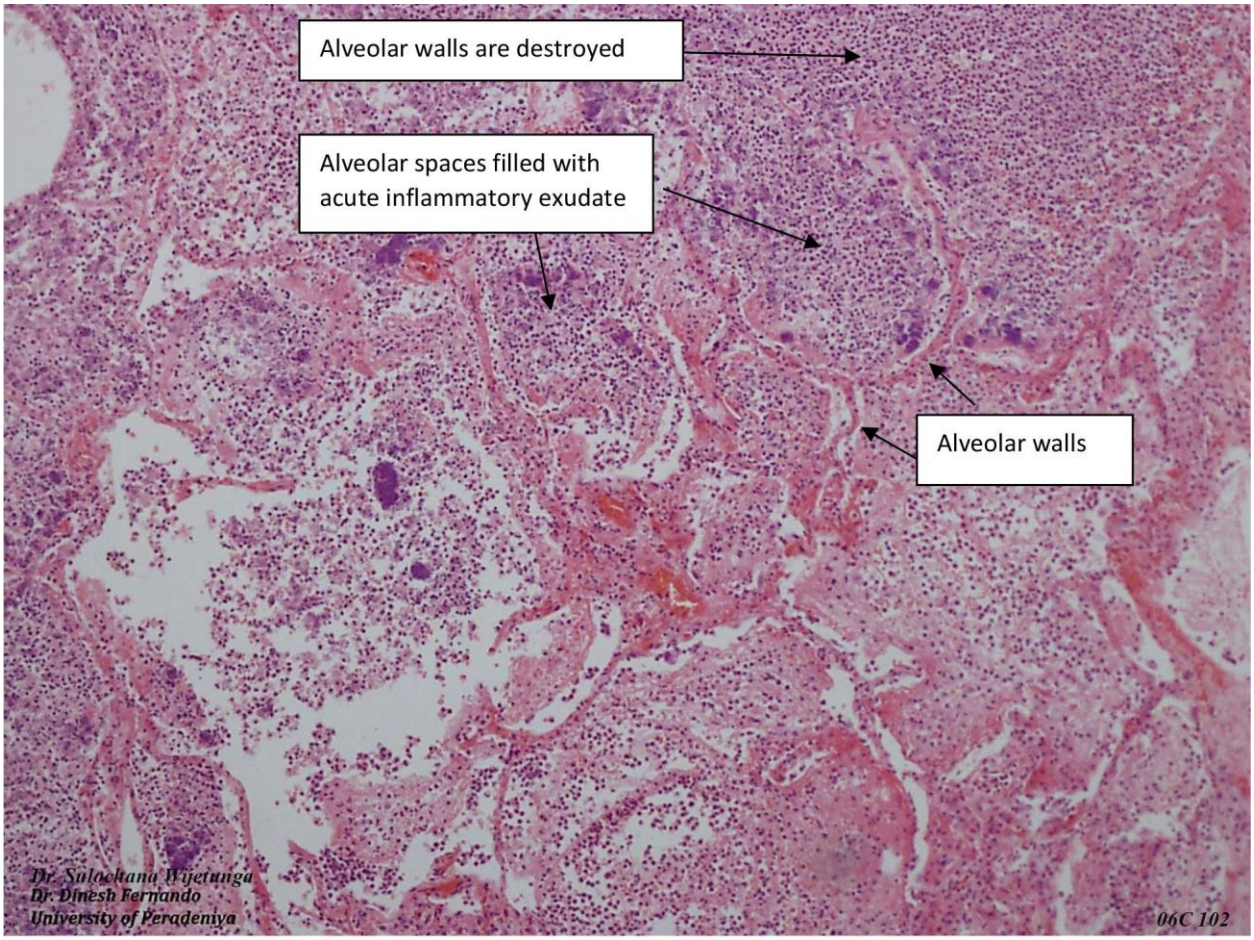

Figure 3: Alveoli filled with cellular acute inflammatory exudate (Low power view)

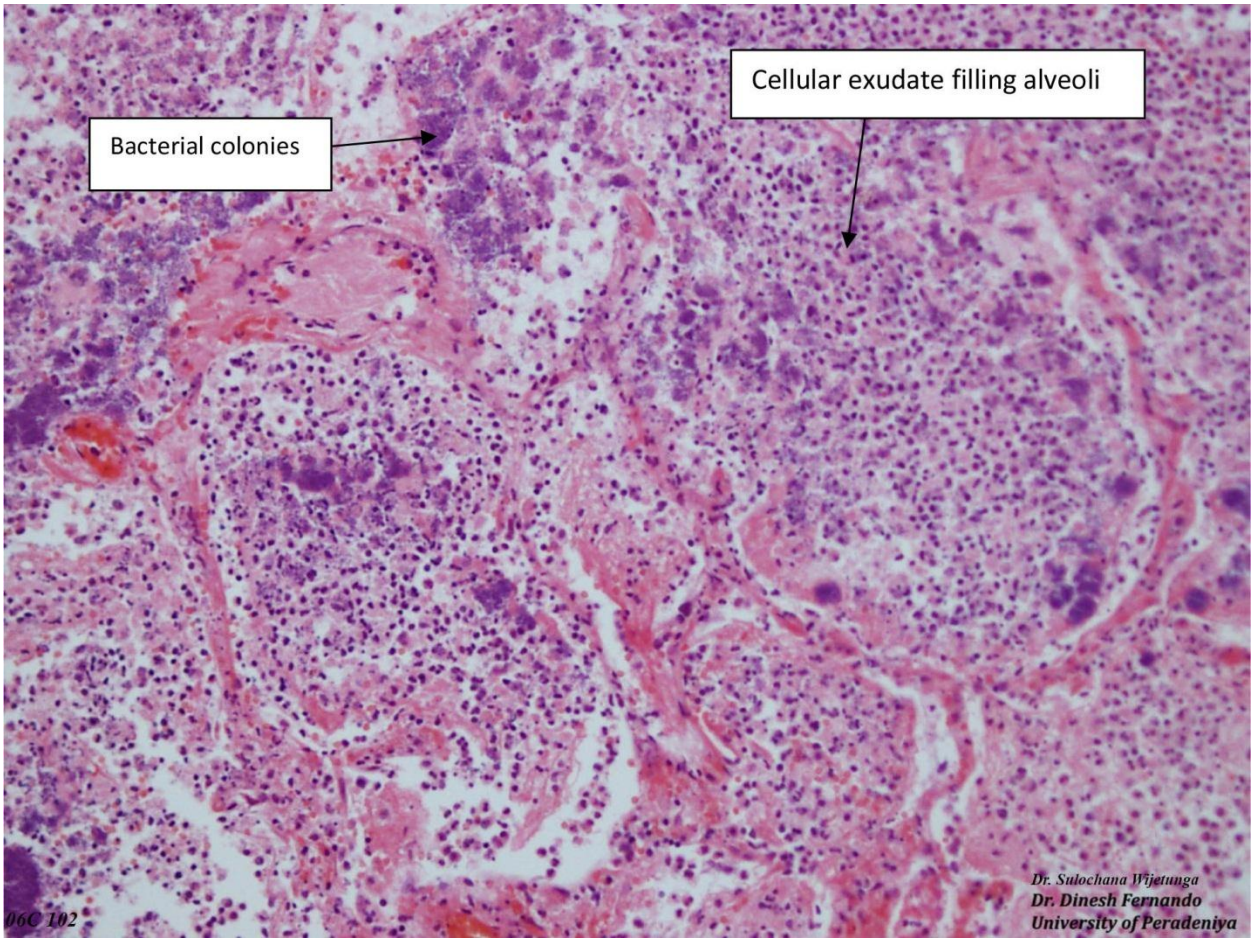

Figure 4: The alveolar cellular exudate mixed with bacterial colonies (High power view)

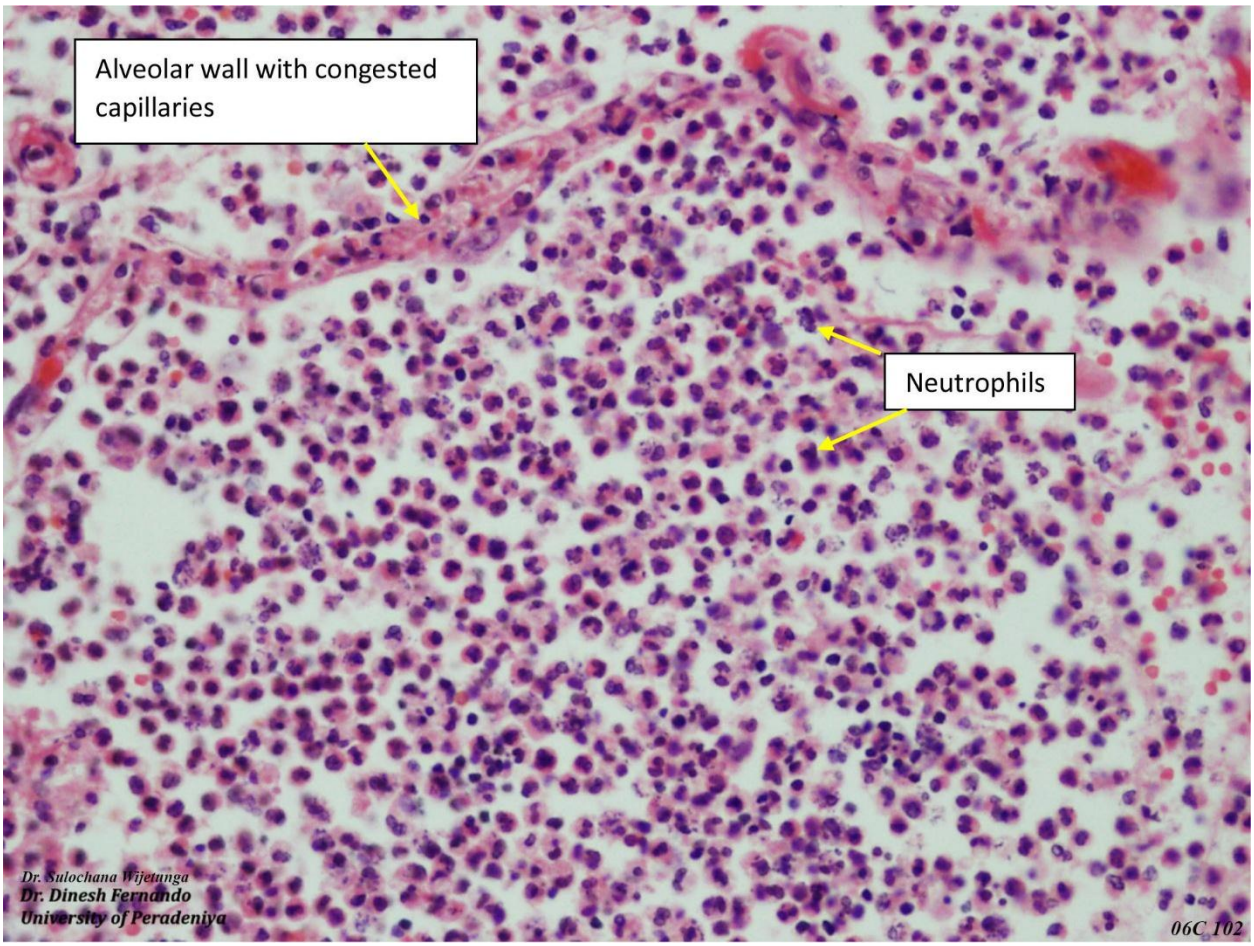

Figure 5: Abundance of neutrophils within alveoli. Neutrophils are recognized by their lobulated nuclei (High power view)

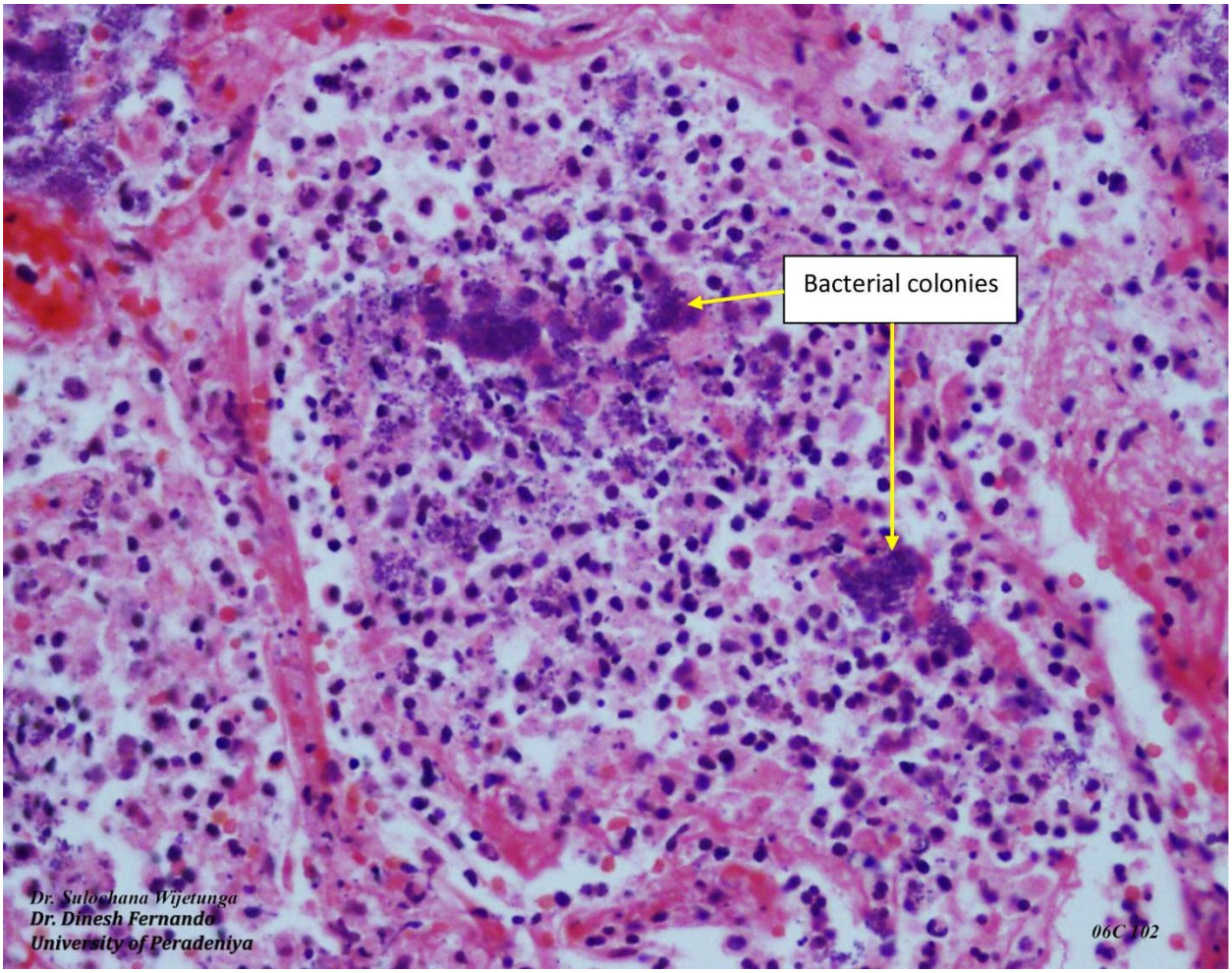

Figure 6: Bacterial colonies observed as densely basophilic powdery material (High power view)

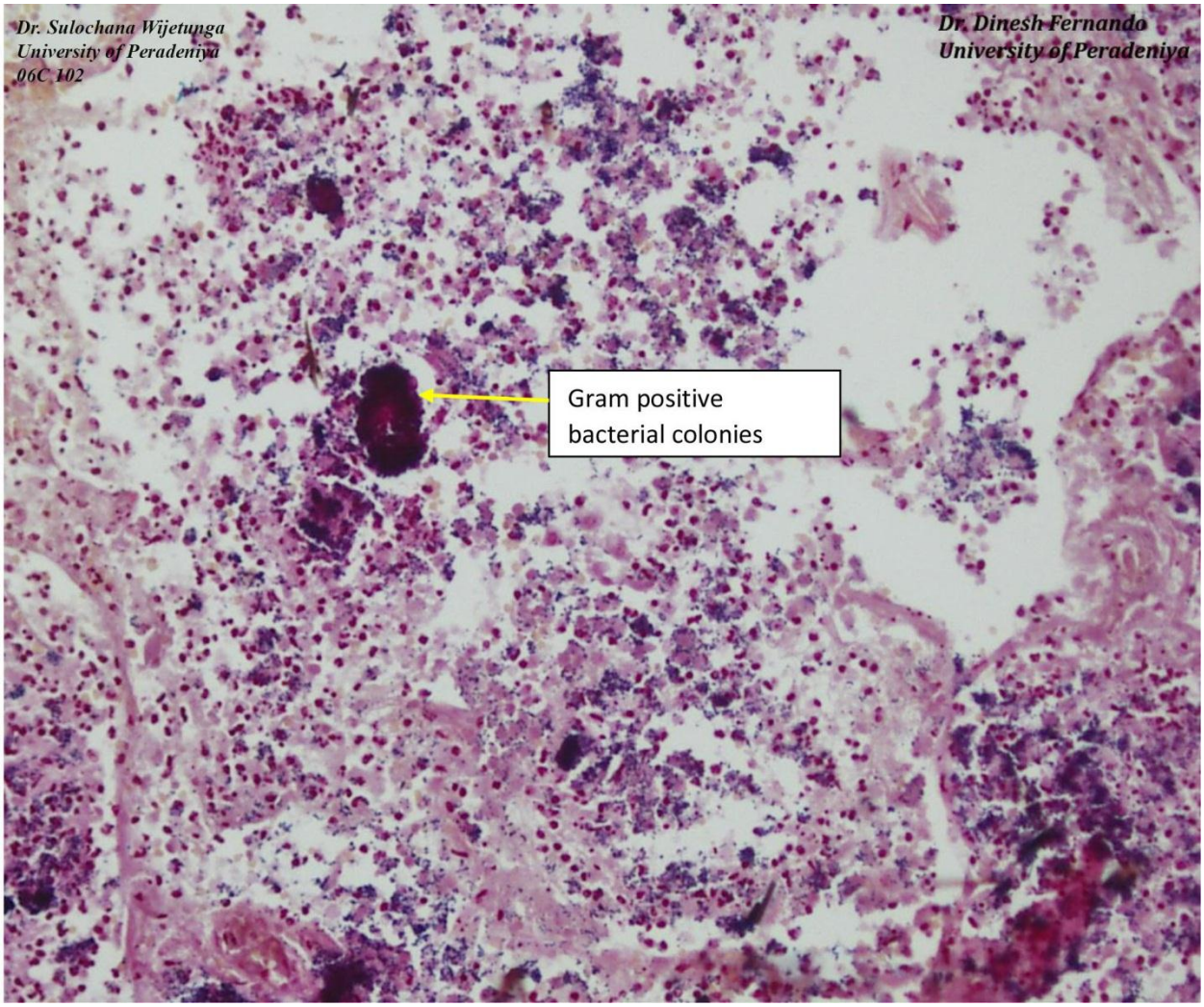

Figure 7: Gram positive bacterial colonies observed on gram stained sections

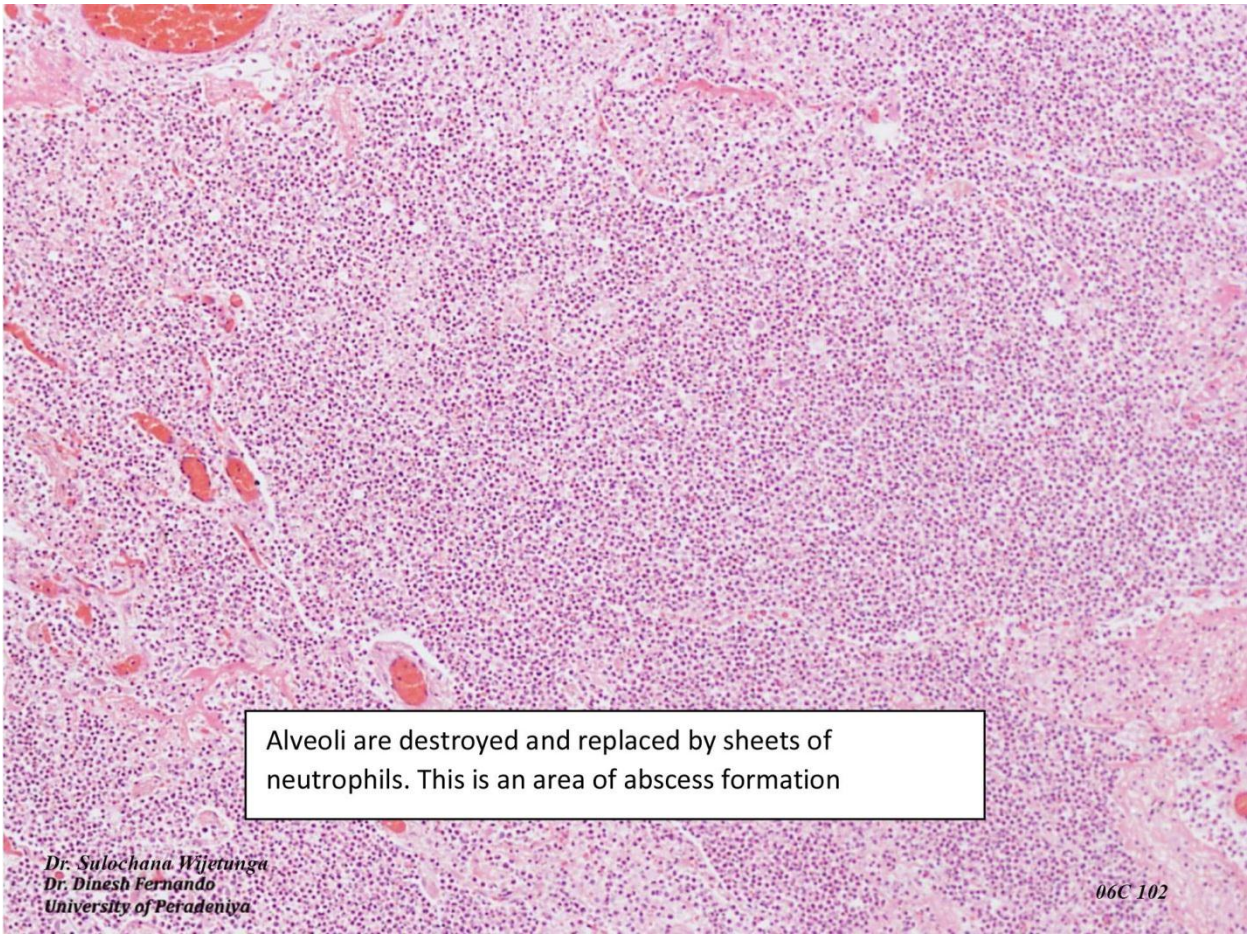

Figure 8: An area of abscess formation, which is a result of localized extensive tissue damage by acute inflammation

- Pneumonia is histologically characterized by presence of an acute inflammatory exudate in the lung parenchyma, namely within alveoli, with, or without bronchiolar involvement. The acute inflammatory exudates are predominantly composed of neutrophils with fibrin, cellular debris and oedema.

Cause of death

Staphylococcal pneumonia

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COLOR ATLAS OF

FORENSIC PATHOLOGY

ENDOCRINE SYSTEM

ADRENAL HAEMORRHAGE

Adrenal haemorrhages, which are frequently bilateral, leads to rapid destruction of the adrenals and precipitates acute adrenal insufficiency. The patient presents with symptoms of adrenal insufficiency, shock or in acute adrenal crisis.

Waterhouse-Friderichsen syndrome is a rare clinical syndrome with bilateral adrenal haemorrhages seen mainly in meningococcal or pseudomonas septicaemia in children, but also several other bacterial and viral infections can cause it. Other causes of bilateral adrenal haemorrhage include coagulation disorders (eg, antiphospholipid syndrome), adrenal vein thrombosis, adrenal metastases, traumatic shock, severe burns, abdominal surgery, and obstetric complications etc.

Pathophysiology of bilateral adrenal haemorrhage is still debatable. According to literature it is said to be a manifestation of stress response which leads to an increase serum adrenocorticotrophin (ACTH) followed by synthesis of cortisol and adrenaline. This will ultimately raise adrenal blood flow, which increases pressure within the vessels leading to rupture.

Unilateral adrenal haemorrhages are uncommon and it is most frequently seen in direct trauma causing traumatic adrenal rupture.The inner cortex and medulla are almost entirely replaced by haematomas.

History

A 35-year-old male sustained a head injury following a fall from a height of 5 to 6 metres.
The injuries sustained in the head included; a left sided subdural haematoma with extension into the subarachnoid space, associated with cortical contusion, oedema and midline shift.

The upper pole of the right kidney was lacerated.

A craniotomy for the subdural haematoma and decompression was done and a large amount of blood (approximately 500mls) was drained. He was ventilated and died after 2 days without regaining consciousness.

Internal examination

Endocrine system: The left adrenal was of normal size with thin yellow cortex and grey tan medulla. The right adrenal had a large haemorrhage and the haemorrhage plus adrenal gland weighed 38 grams.

Central Nervous System: The brain was herniating out of the craniectomy site. An organising extradural haematoma which measured approximately 25mls was present on the left temporo-parietal region in relation to the craniectomy site. Another extradural haematoma which measured approximately 50mls was present in the right occipital area and was composed of relatively fresh blood.

Macroscopic Examination

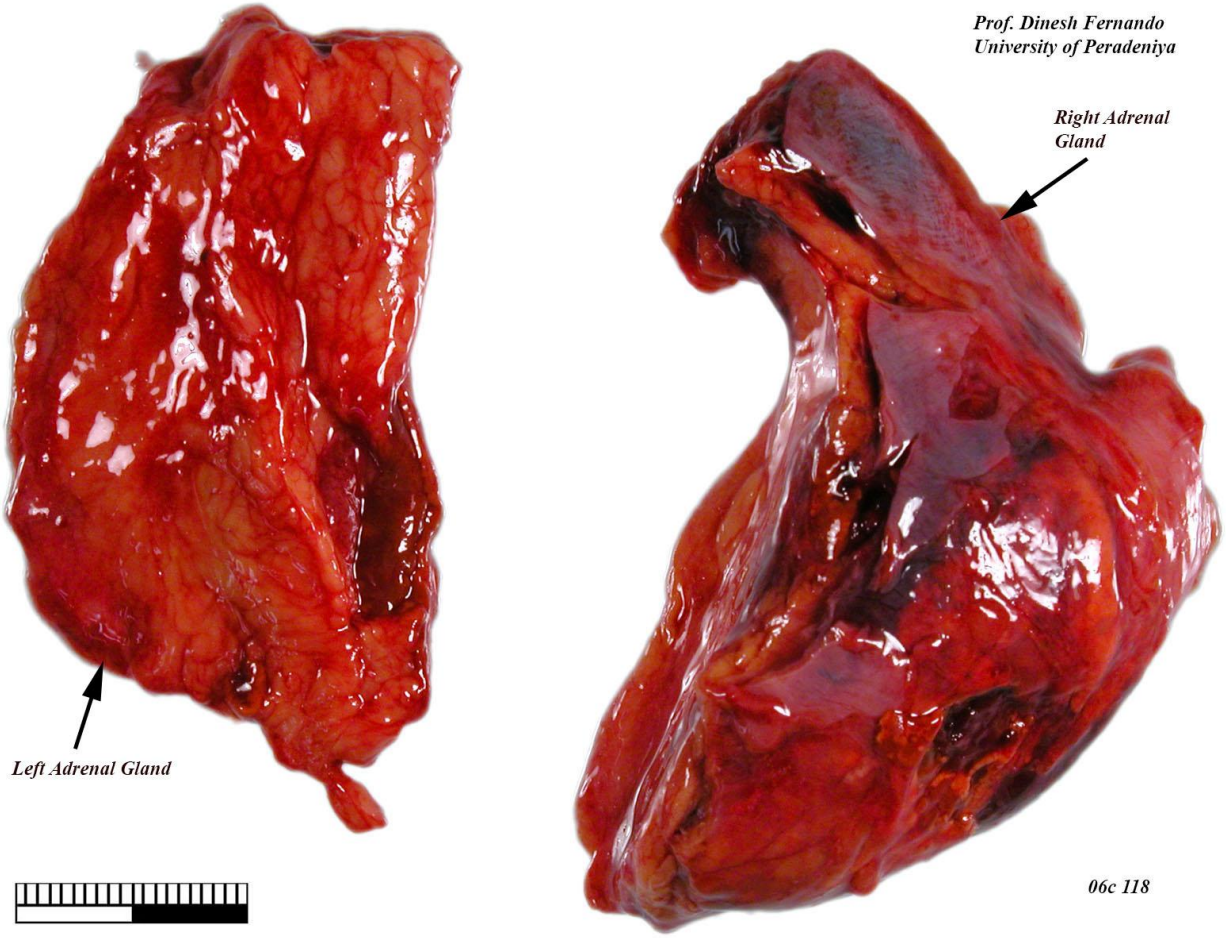
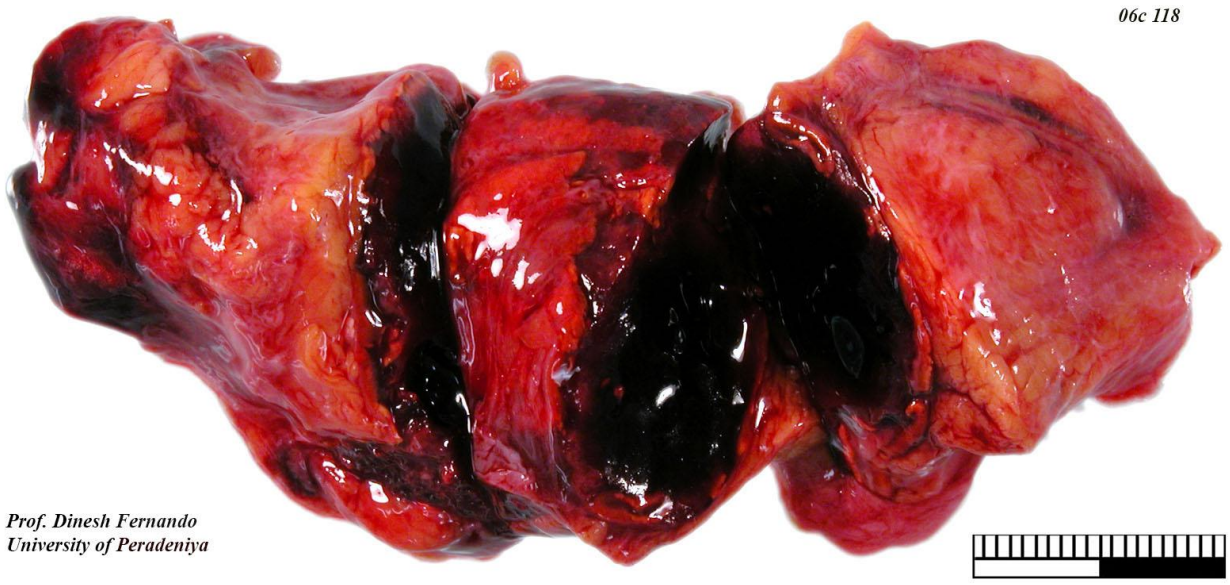


Figure 1: Left and right adrenal glands



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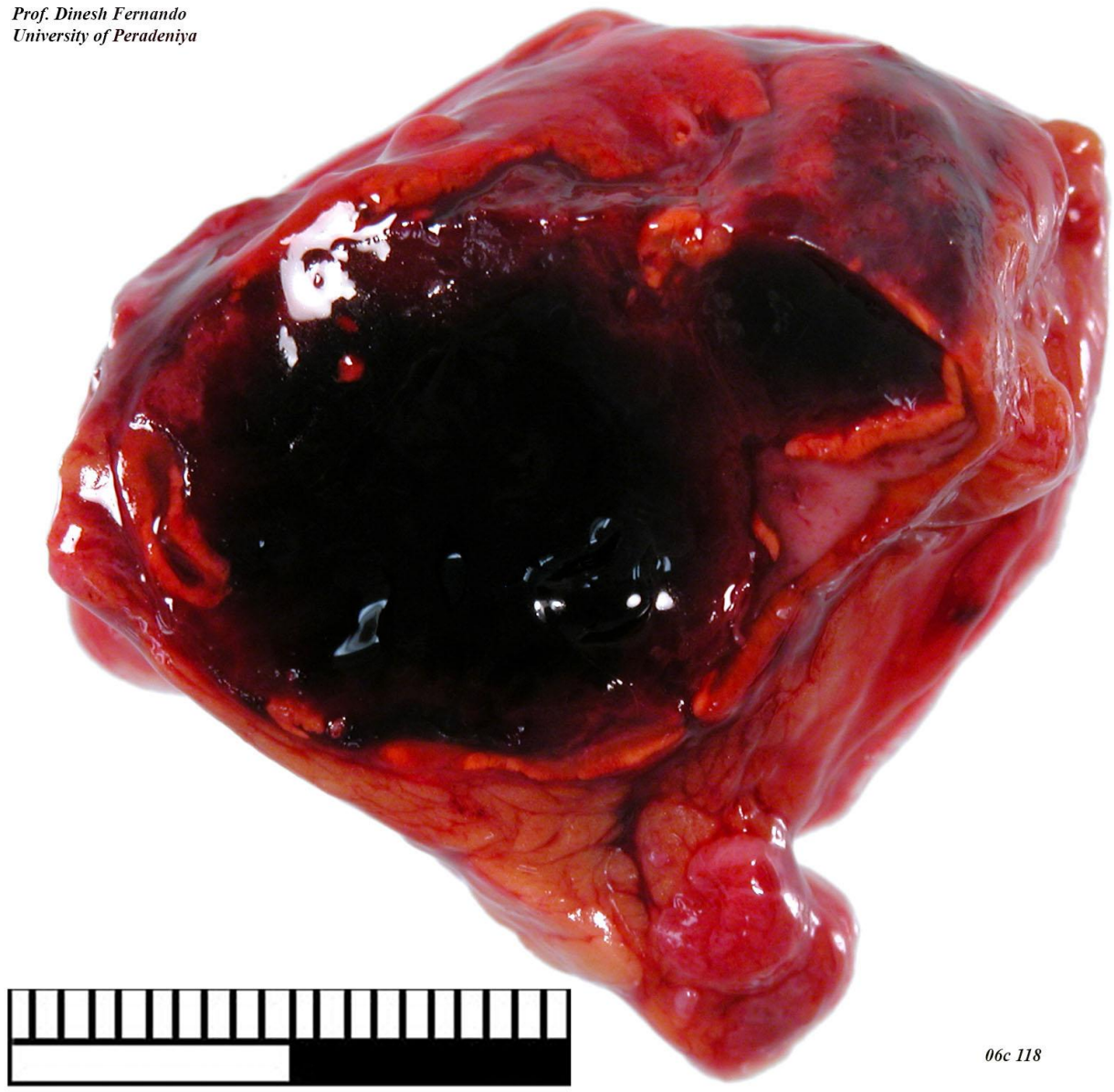

(b)

Figure 2(a & b): Haemorrhage within the right adrenal gland

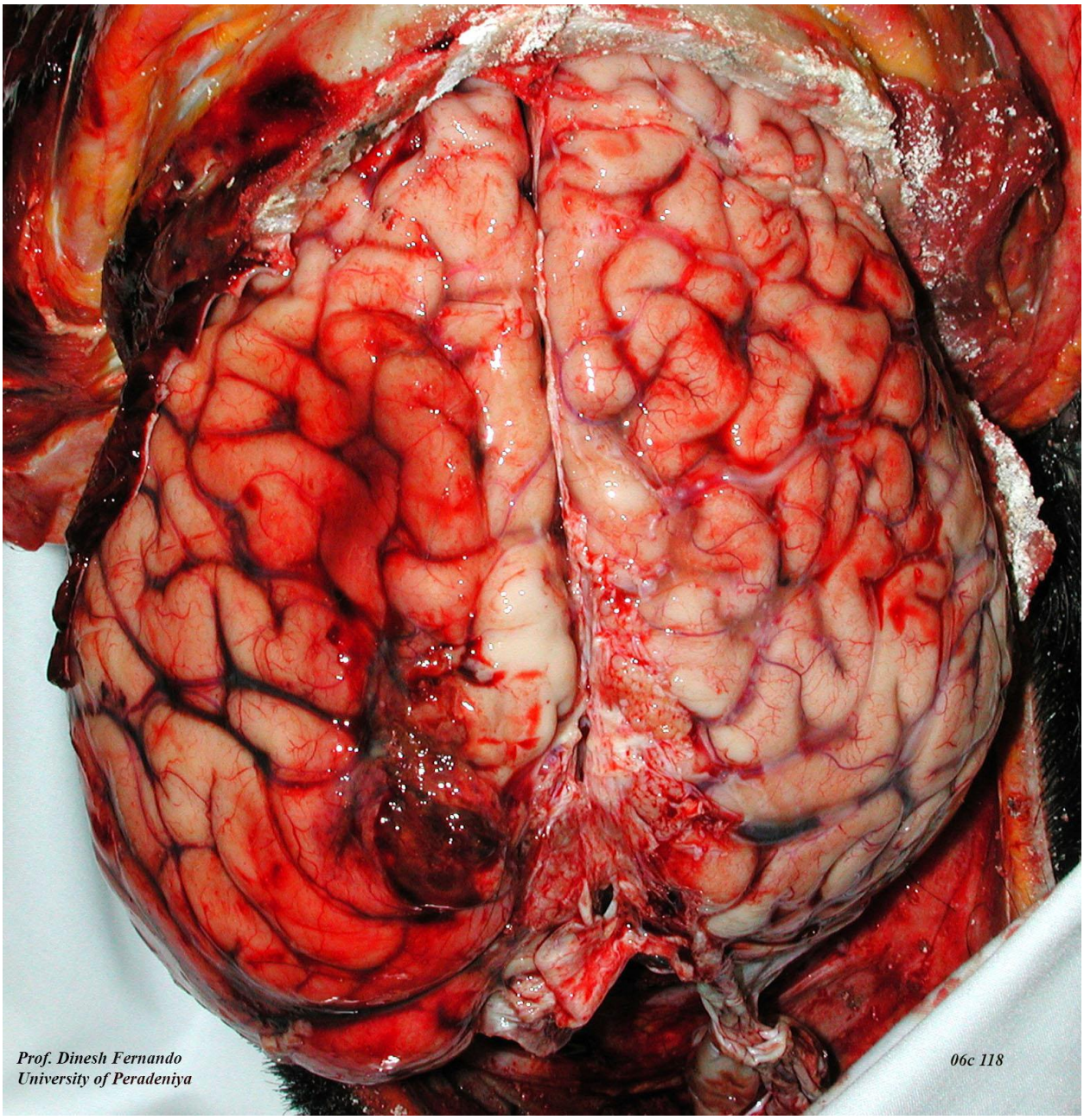

Figure 3: Oedema of left cerebral hemisphere

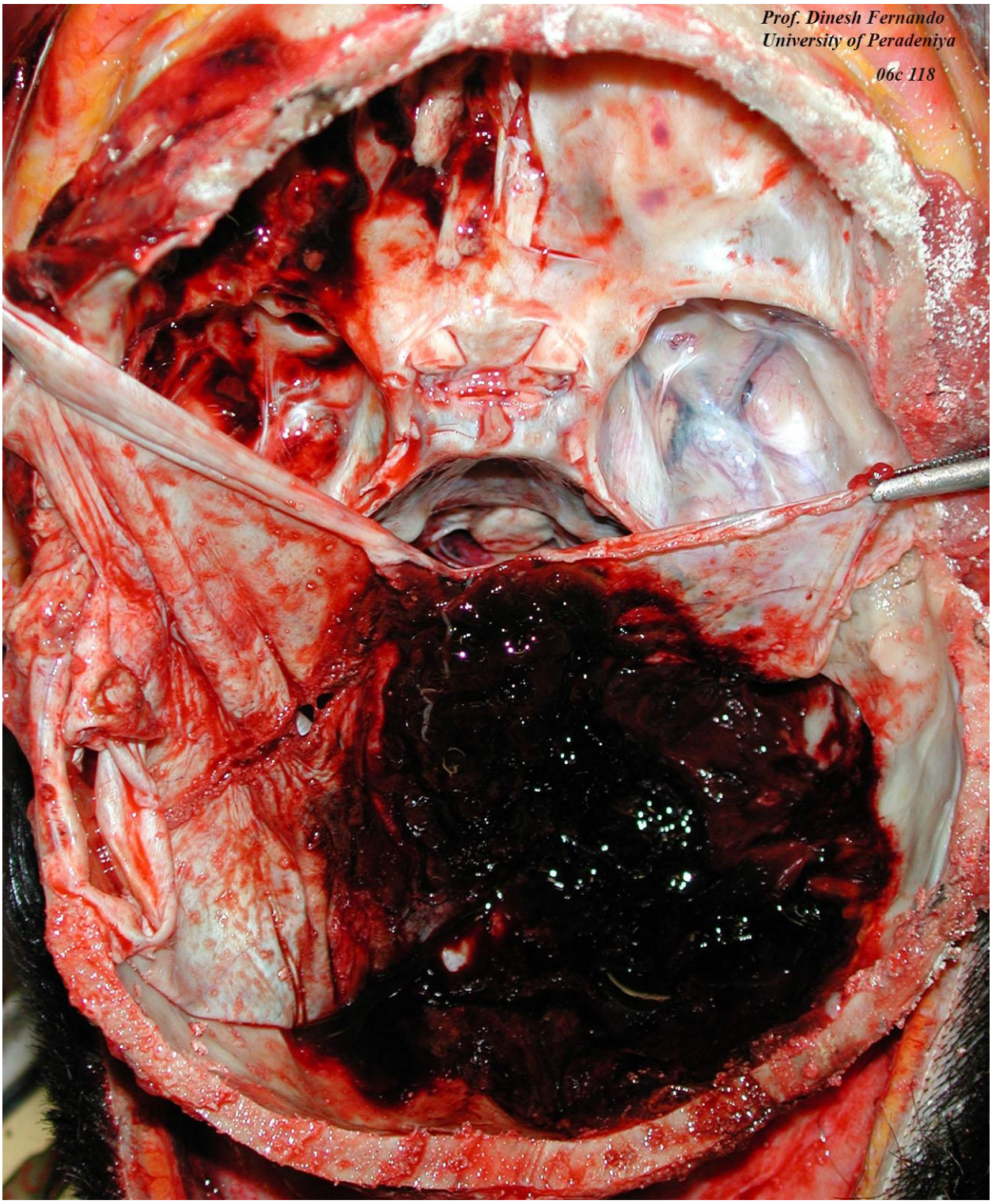

Figure 4: Extradural haematoma in occipital area

Figure 5: Dura with extradural haematoma

Microscopic Examination

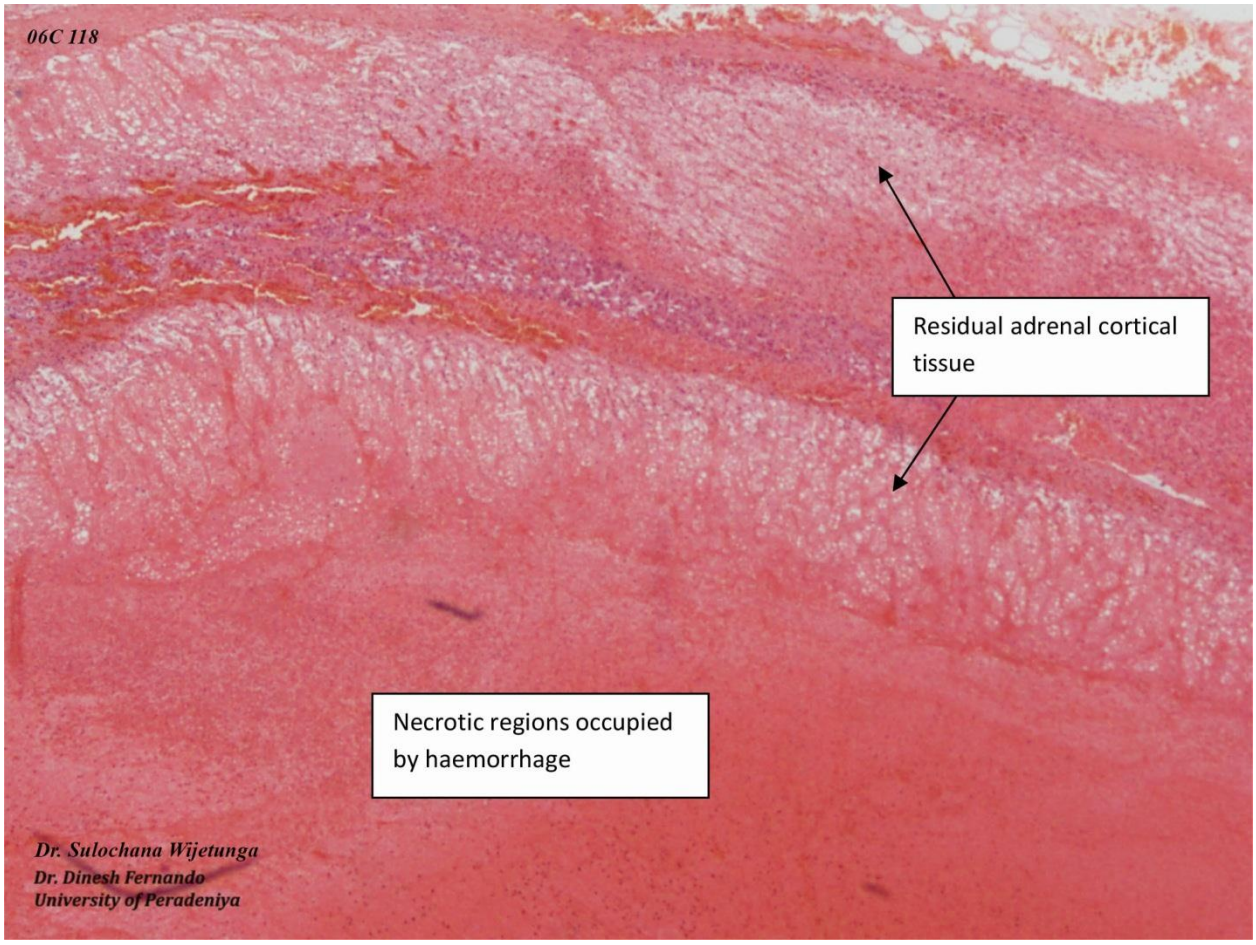

Figure 6: There is some residual viable cortical tissue

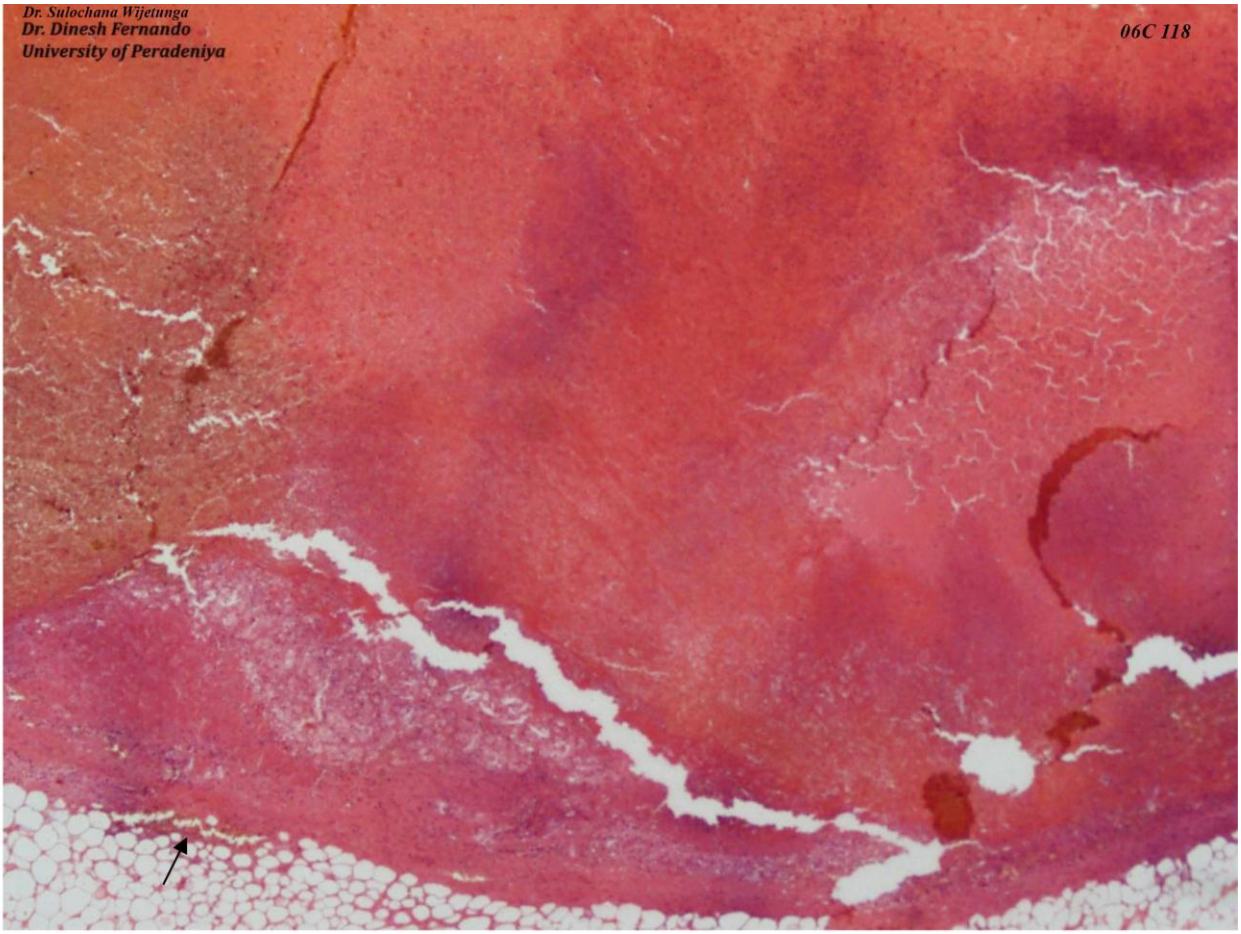

Figure 7: No viable tissue is seen and the entire region is necrotic occupied by a haematoma. There is bleeding extending to peri renal tissue (arrow)

There is extensive necrosis of adrenal tissue with haemorrhage.

Pathogenesis of adrenal haemorrhage in non-traumatic situations is not clear. It has been attributed to the limited venous drainage of the gland, i.e., although adrenal gland has a good arterial blood supply, its venous drainage is by a one single vein making it vulnerable to congestive necrosis and secondary haemorrhage. Adrenal vein spasms in extreme stressful situations, adrenal vein thrombosis in sepsis and other hypercoagulable situations such as heparin induced thrombocytopenia, anti-phospholipid syndrome and DIC have been implicated.

Extensive bilateral adrenal haemorrhage lead to acute adrenal failure, adrenal crisis, shock and death.

Cause of death

Adrenal Haemorrhage

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ACUTE PANCREATITIS

Acute Pancreatitis is characterized by reversible pancreatic injury due to an acute inflammatory reaction. It occurs due to an inappropriate release and activation of pancreatic enzymes which destroys the pancreatic parenchyma. The injury varies in severity, ranging from focal oedema and fat necrosis to widespread parenchymal necrosis. Also, the destruction of blood vessels leads to interstitial haemorrhage.
The released pancreatic enzymes are responsible for the secondary systemic response. Lipolytic enzymes self-digest pancreatic tissue and peripancreatic fat. The lysis of those fat cells releases fatty acids which precipitates with serum calcium in the form of soap. This is the phenomenon known as fatty saponification. It is thought to be responsible for the hypocalcemia which is seen in severe acute pancreatitis. There are two types of acute pancreatitis according to the presence or absence of necrosis; the mild (interstitial-oedematous) and severe (necrotizing pancreatitis). The necrosis may be seen in, either, the pancreatic gland, peripancreatic fat or mostly seen in both.

Gallstones and alcohol are the most common causes, while infections, drugs and metabolic disorders that injure the acinar cells or damage the duct epithelium, can also trigger acute pancreatitis. It presents with a sudden onset of epigastric pain which radiates to the back and is relieved on bending forward. There is associated nausea and vomiting. Cullen's sign (periumbilical bruising), Grey Turner sign (flank bruising) and Fox's sign (bruising over the inguinal ligament) are specific clinical features that may be seen rarely. Three-fold or greater rise in serum amylase or lipase supports the diagnosis.

History

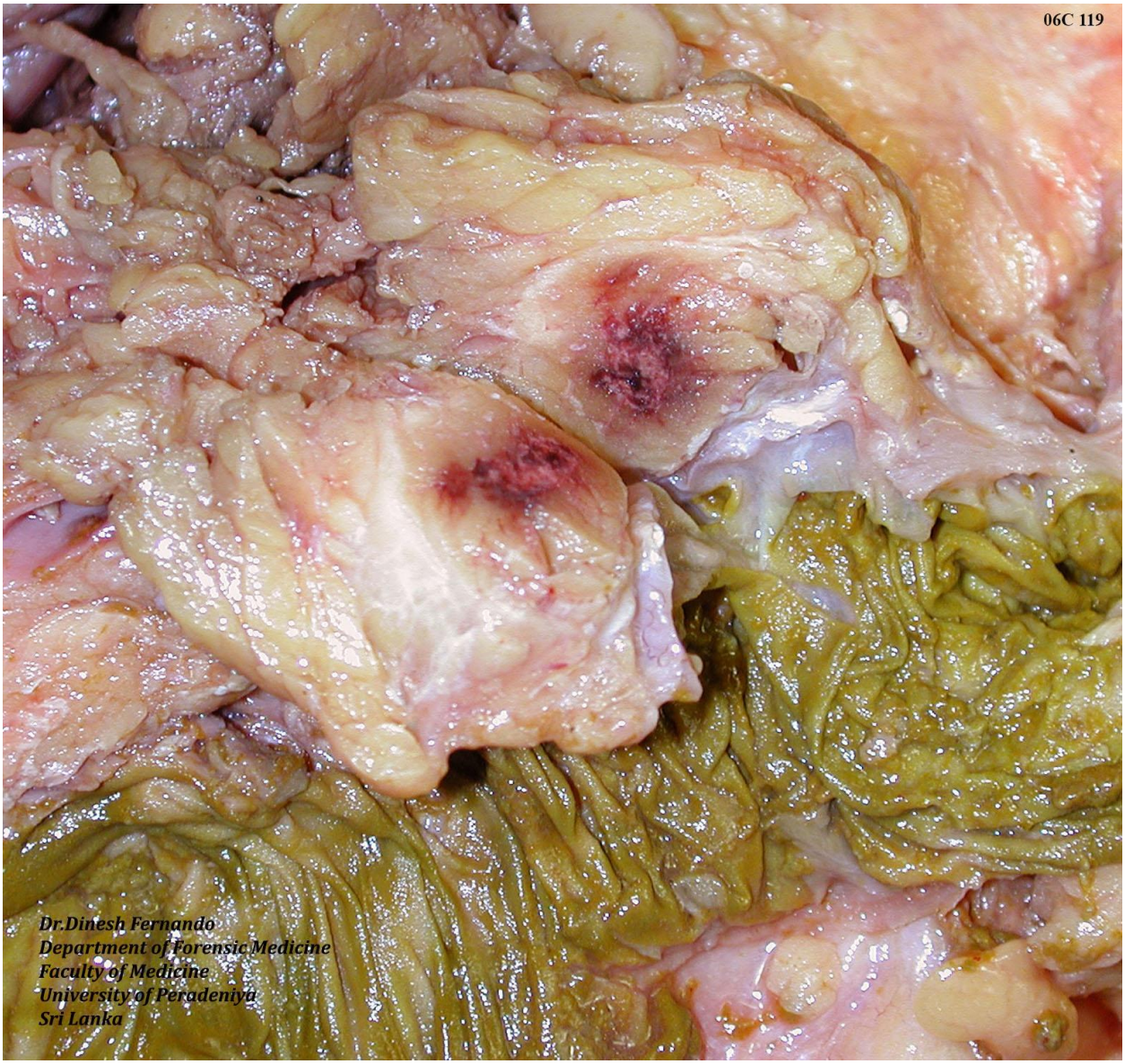
A 77-year-old male who had several strokes over the past six years and had pancreatitis for the past four years presented to the local hospital with a productive cough of a few days and severe abdominal pain, especially in the right upper quadrant. He had vomited bile. Investigations showed raised amylase. He was tachypnoeic with a respiratory rate of 36. The blood pressure had been 120/60 & GCS was 13/15. His condition had deteriorated and he had transferred to a tertiary care hospital ICU on the evening prior to his death. During transfer in the ambulance he had increasing respiratory distress and became more tachypnoeic with saturation being in the low to mid 90s. He had been ventilated in the ICU for 19 hours. Gradually, his condition worsened and treatment was subsequently withdrawn.

Internal Examination

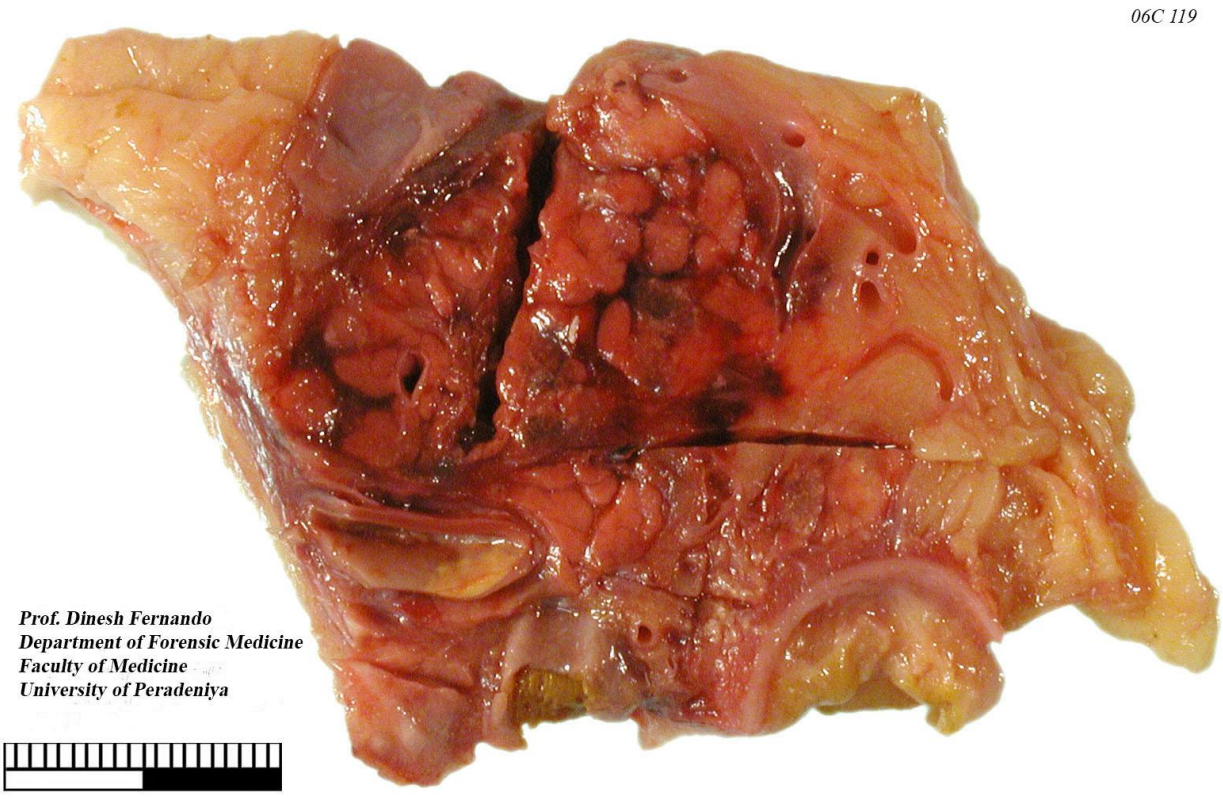
Endocrine system: The thyroid was unremarkable. The pancreas appeared haemorrhagic but macroscopic fibrosis or saponification was not seen.

In order to demonstrate saponification, images from a different case are shown below from Fig 4 to Fig 8. In addition, liver necrosis is shown from Fig 9 to Fig 12.

Central Nervous System: A gelatinous area was present on the inferior aspect of the left occipital lobe close to the midline. Multiple atheromatous plaques were present in the Circle of Willis which had dilated vessels. Multiple sections of the cerebral hemispheres revealed an area of old necrosis in the inferior left occipital lobe. For images see 'Cerebral Infarction' in Brain and Spinal cord.


06C 119
Dr.Dinesh Fernando
Department of Forensic Medicine
Faculty of Medicine
University of Peradeniya
Sri Lanka

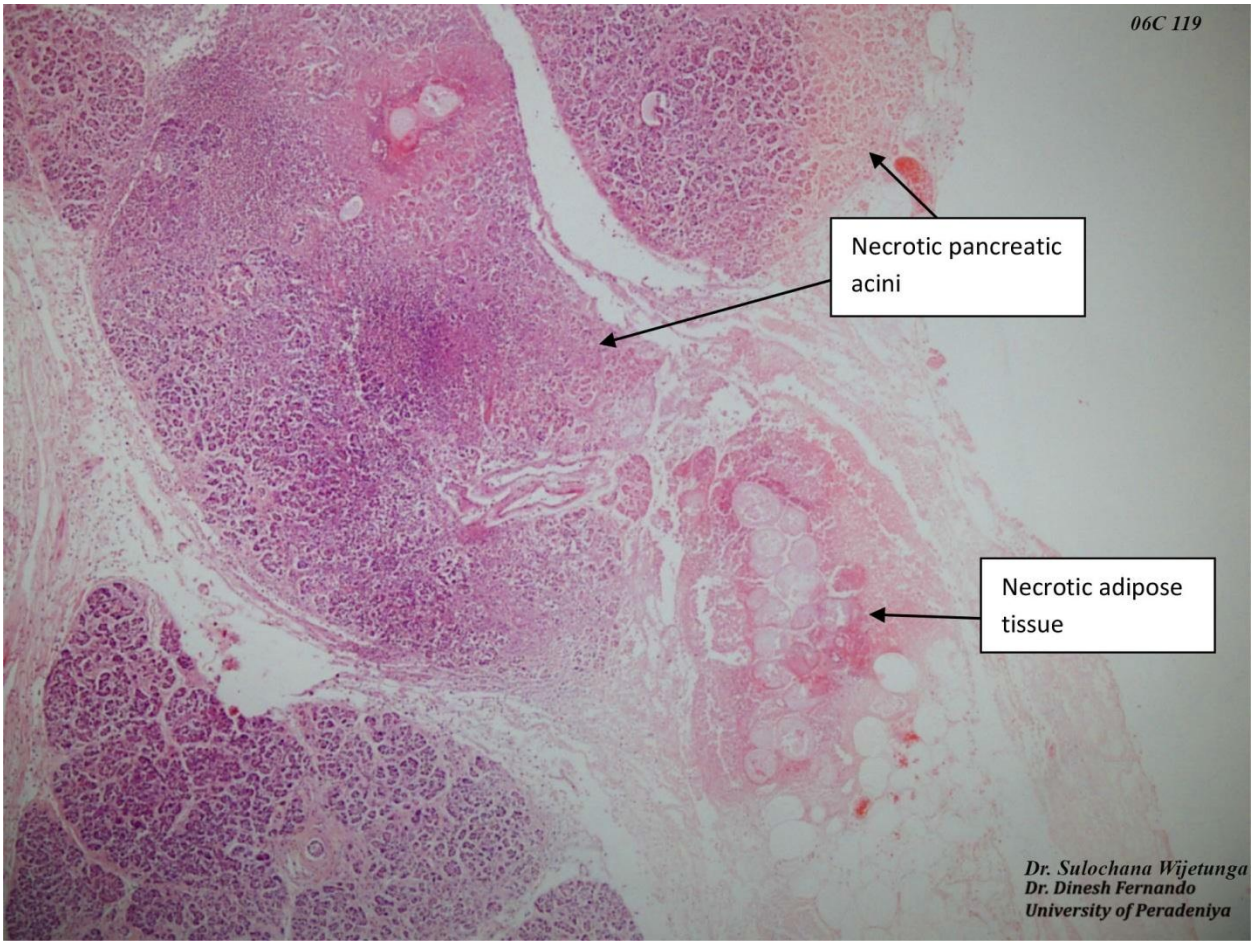
(a)



(b)

Figure 1(a & b): Cut section of pancreas showing haemorrhage

Microscopic Examination



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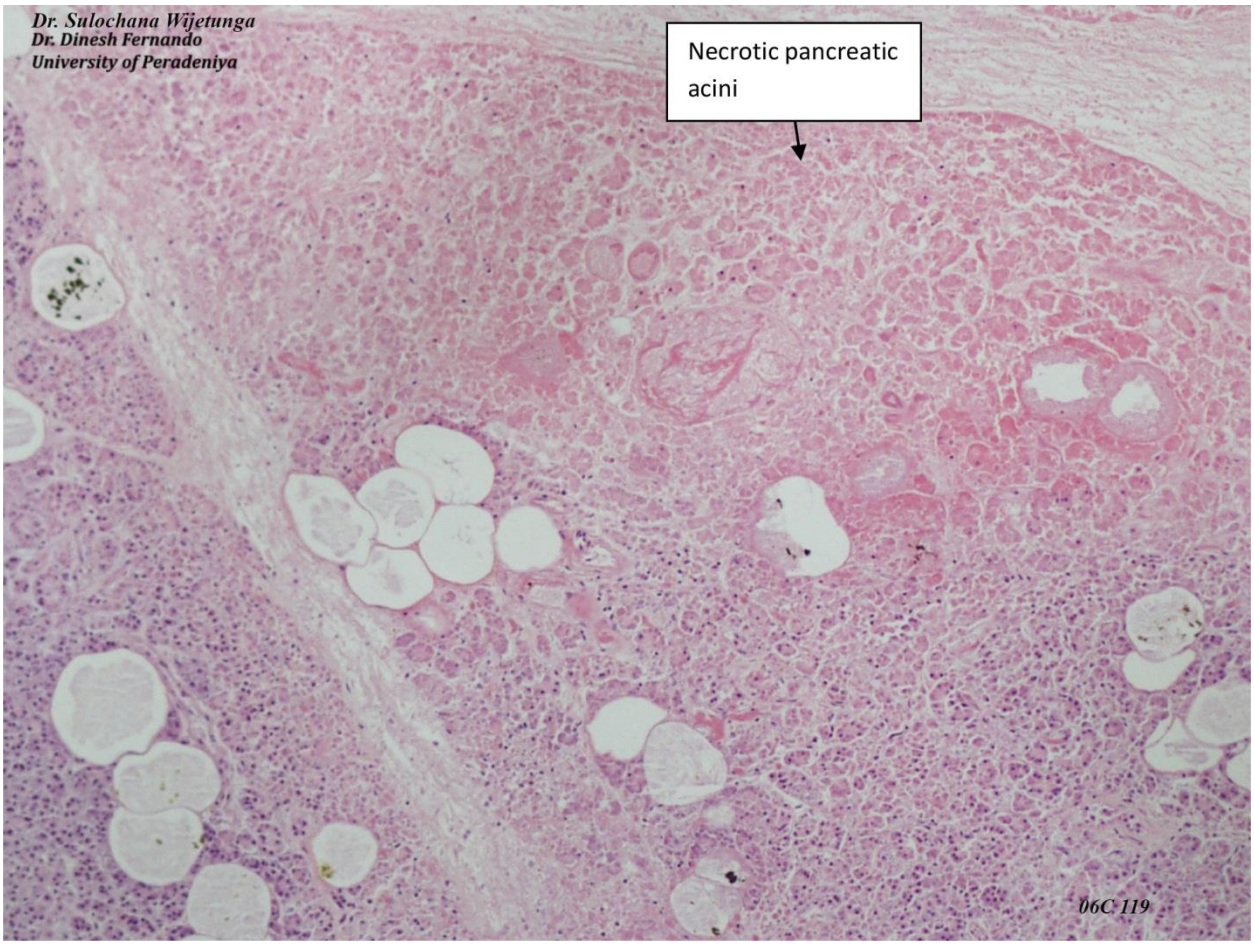
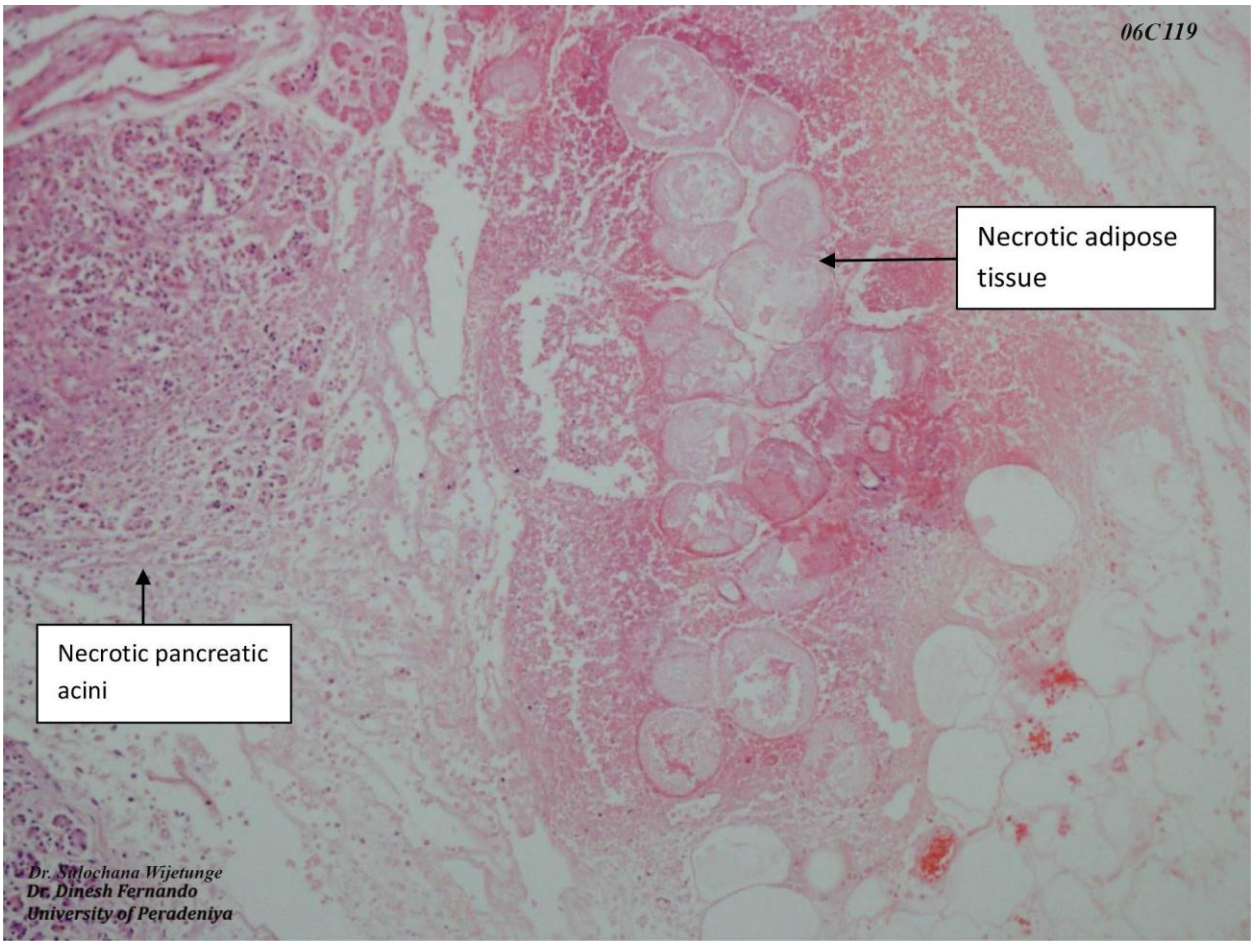

Dr. Sulochana Wijetunga
Dr. Dinesh Fernando
University of Peradeniya
Necrotic pancreatic acini
06C 119

(b)



(c)

Figure 2 (a, b, & c): Pancreatic acini are necrotic and the surrounding adipose tissue undergo enzymatic fat necrosis. Fatty acids released from necrotic adipose tissue form calcium salts and produce chalky white deposits within the peritoneal cavity. (Calcium deposits are seen as deeply basophilic material with haematoxylin and eosin stained histology sections)

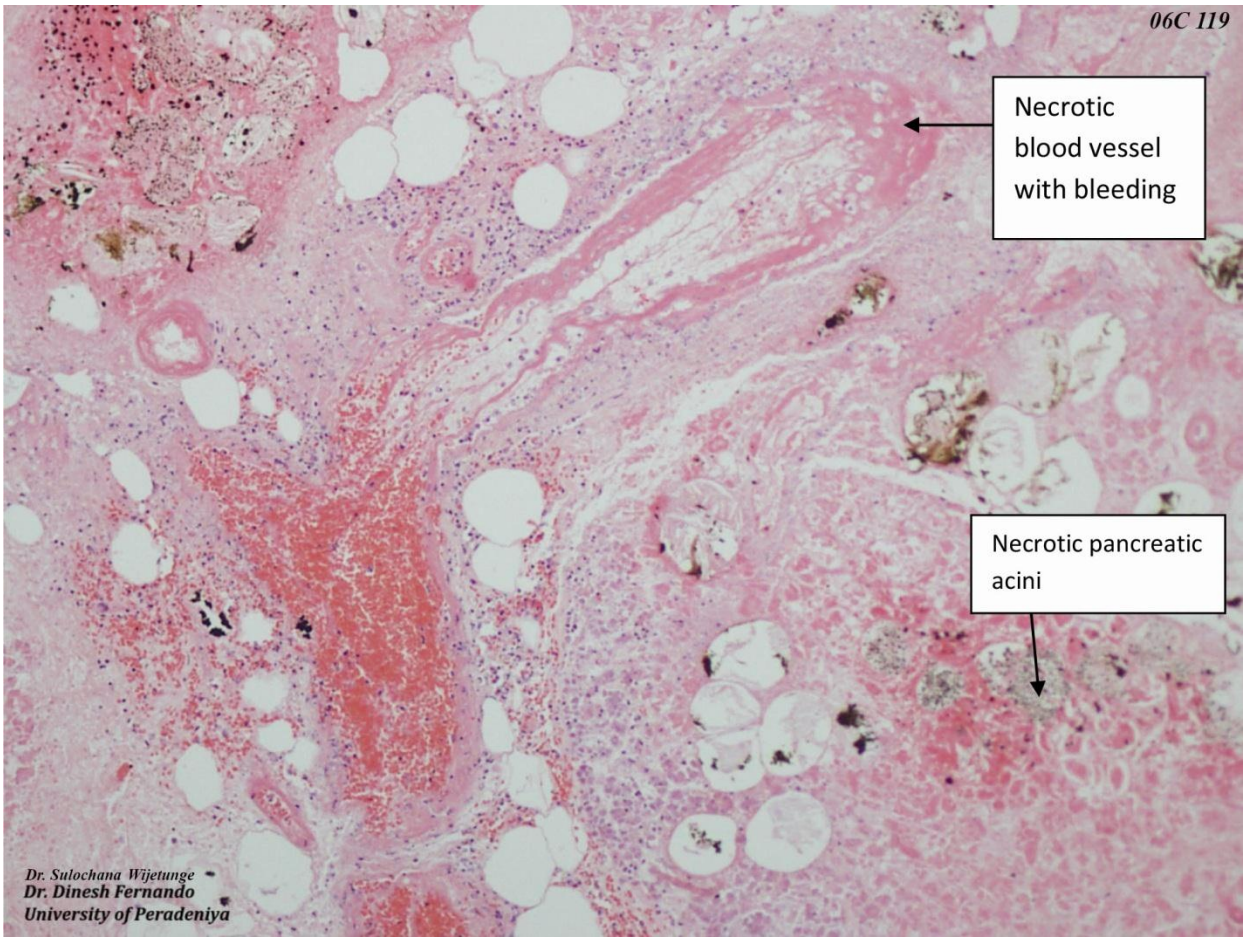


Figure 3: Necrotic blood vessel in the pancreas with bleeding

Blood vessels get digested due to released elastase from necrotic acini. In severe forms of acute pancreatitis with a lot of tissue necrosis extensive blood vessel injury could produce acute haemorrhagic pancreatitis.

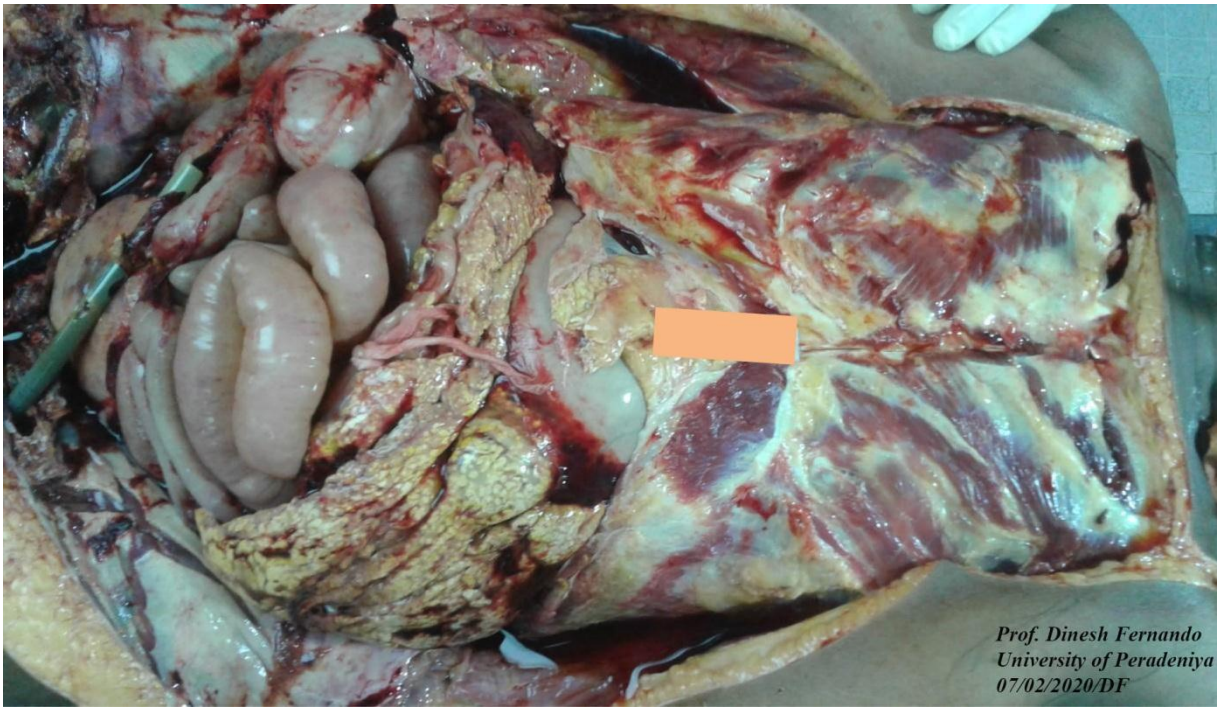
Cause of death

Acute pancreatitis in a person with previous cerebral infarction

Illustration of Saponification and Liver cell necrosis from a different case is given below.

Saponification

Macroscopy



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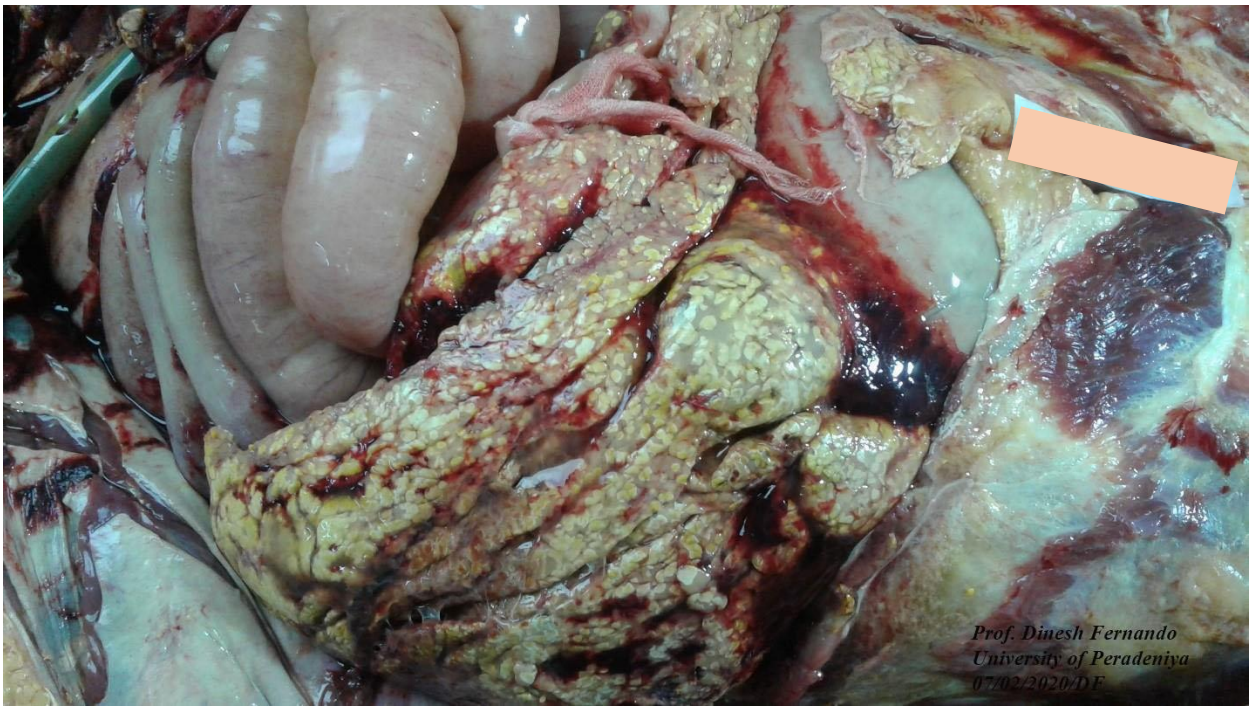

(b)

Figure 4(a & b): Opened abdominal cavity showing saponification of omentum

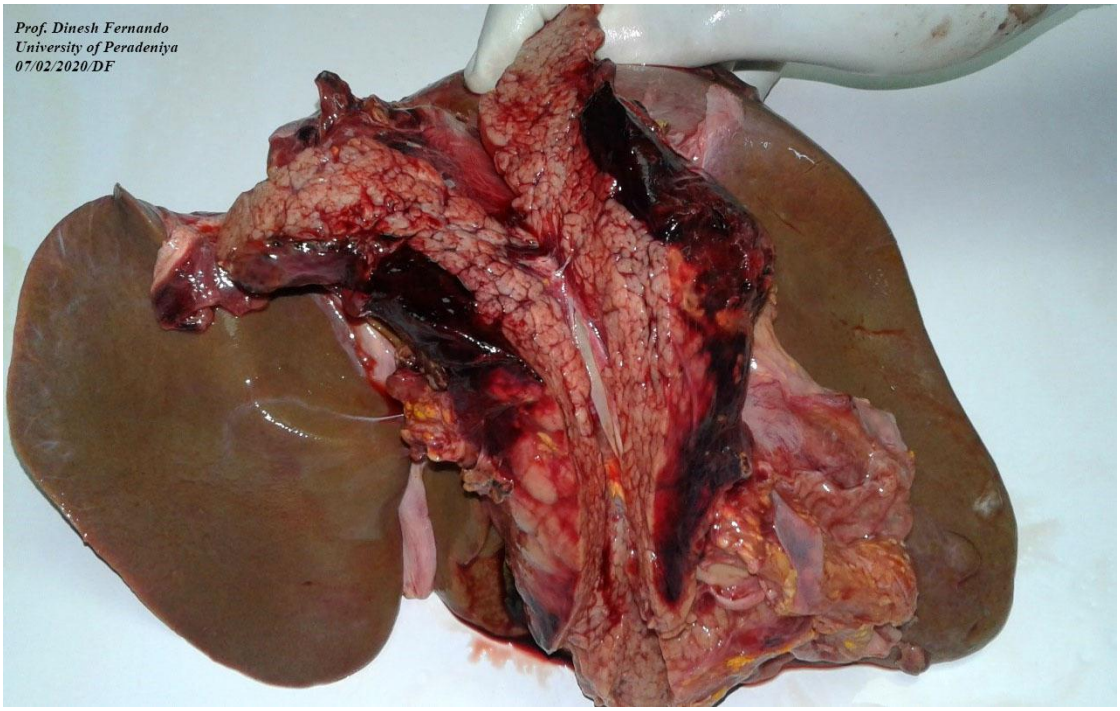

Figure 5: Haemorrhage around the pancreas

Microscopy

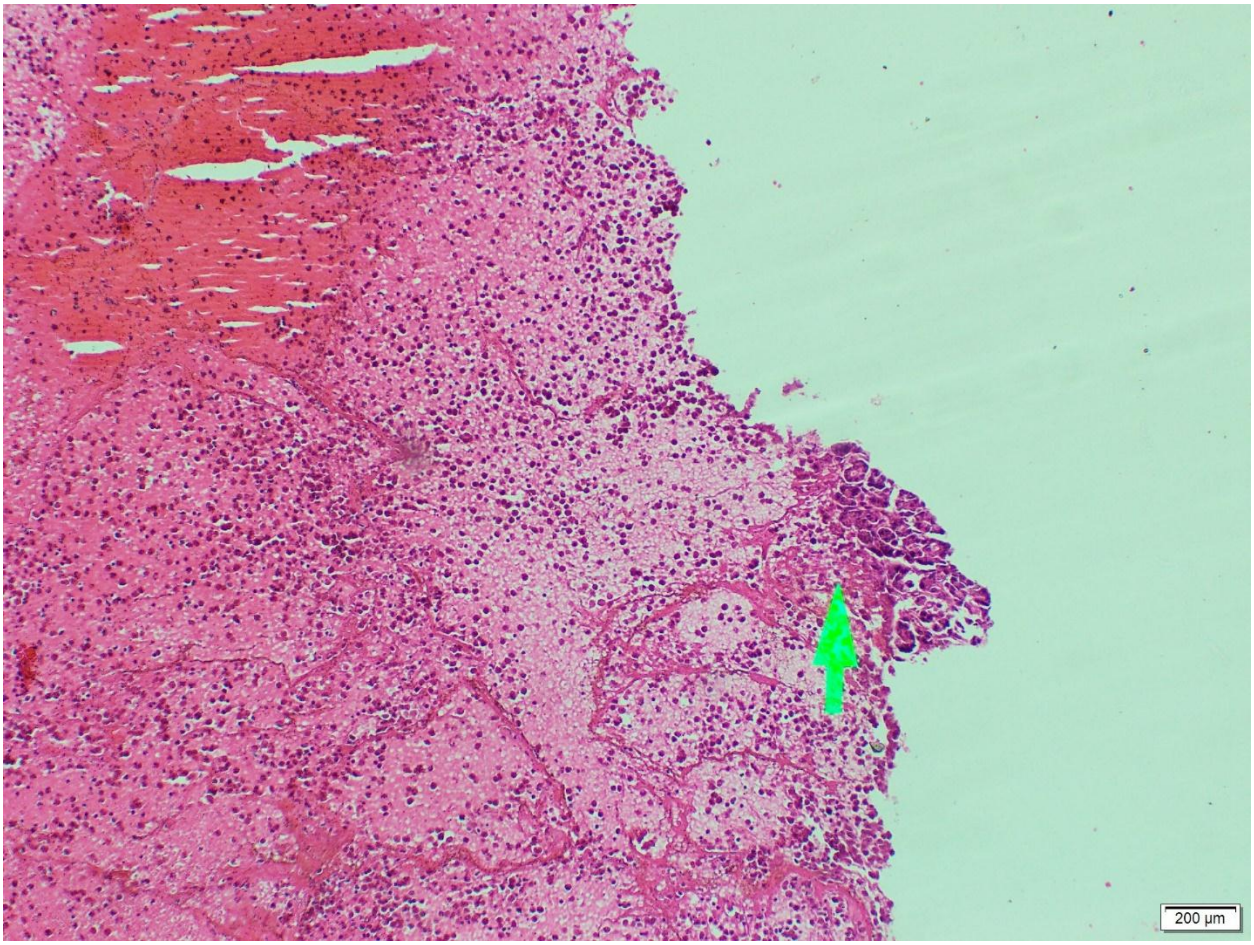

Figure 2: Pancreatic necrosis and haemorrhage with remaining pancreatic acini indicated by the arrow

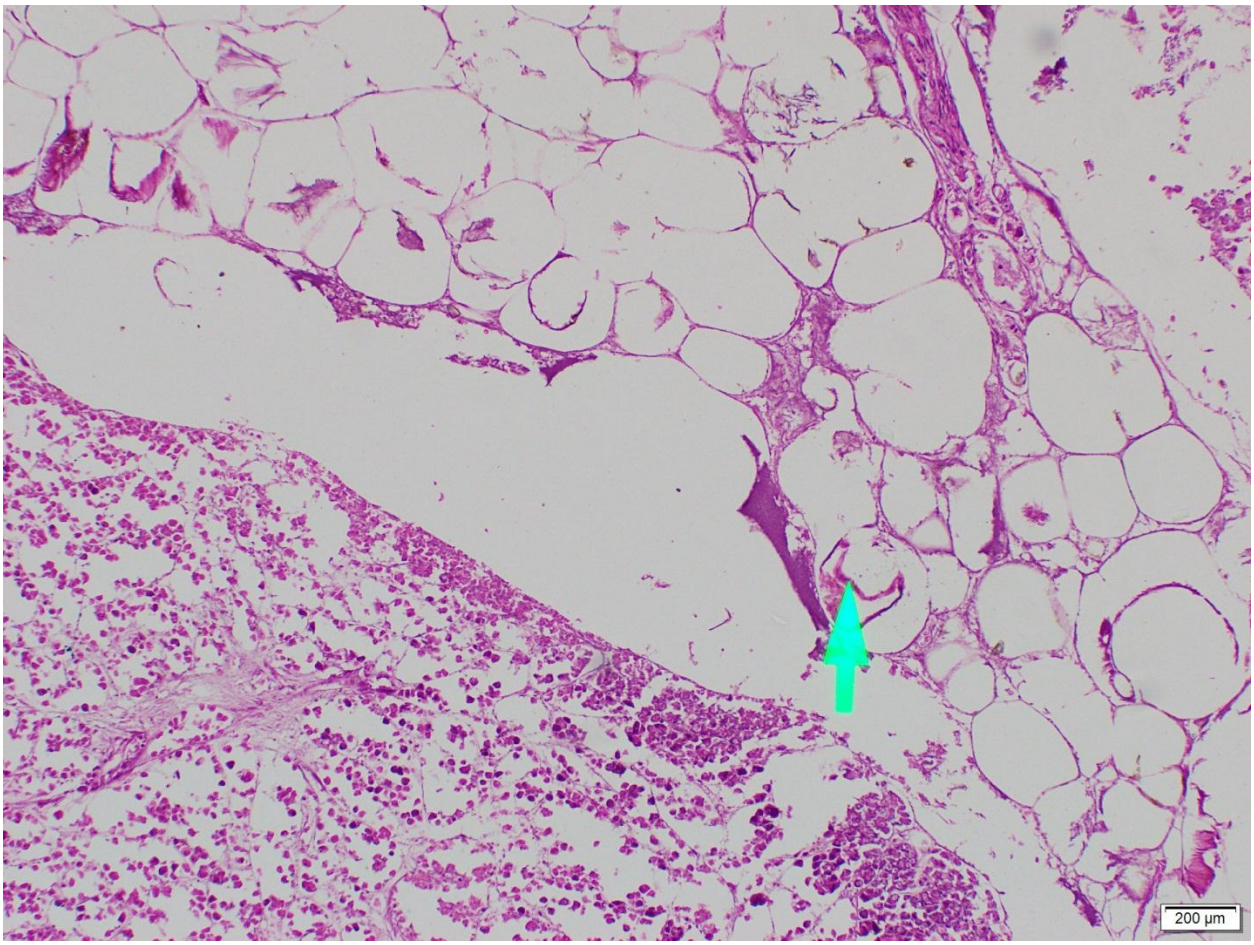

Figure 3: Saponification of the peripancreatic adipose tissue

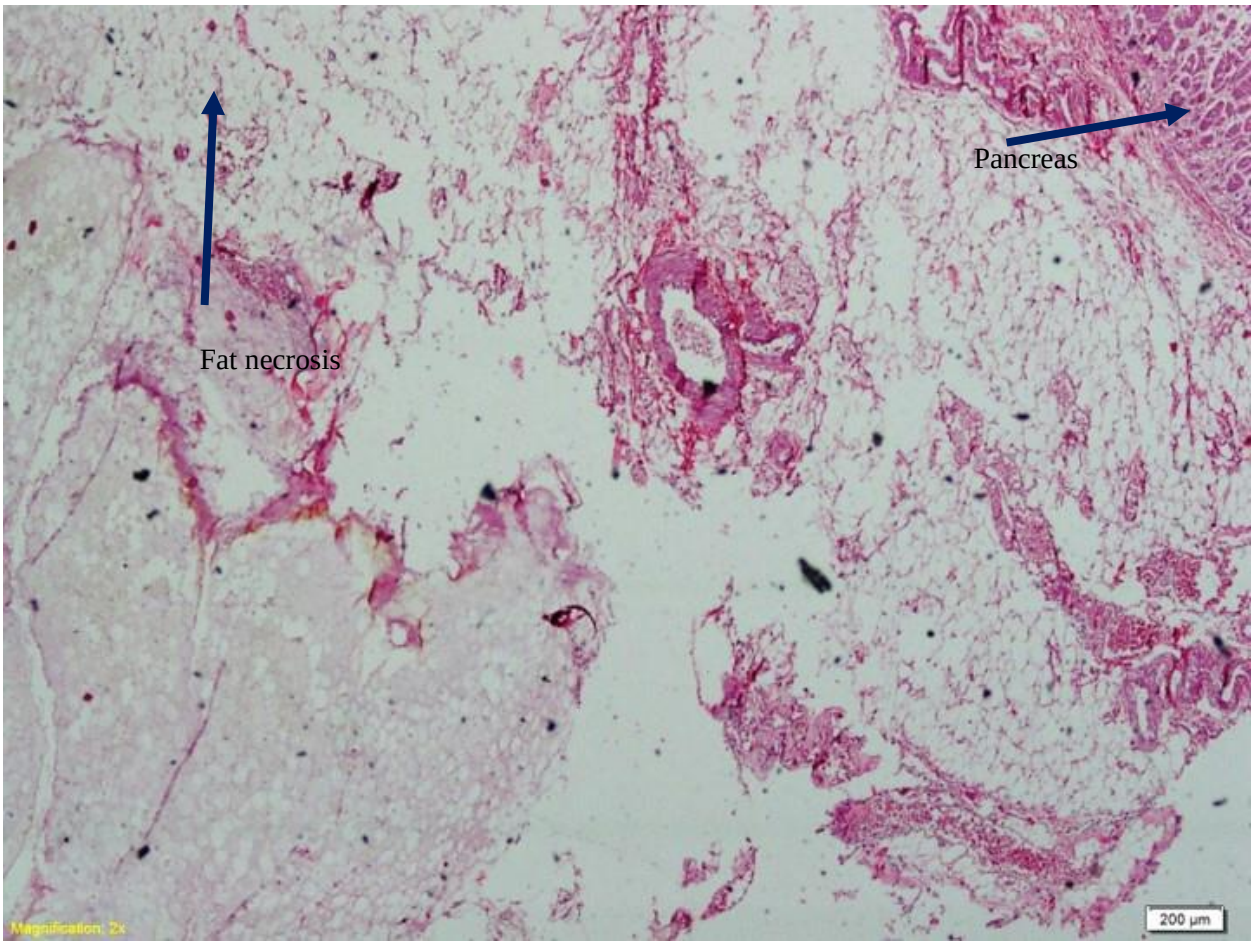

Figure 4:Areas of fat necrosis in the peripancreatic adipose tissue

Liver Cell Necrosis

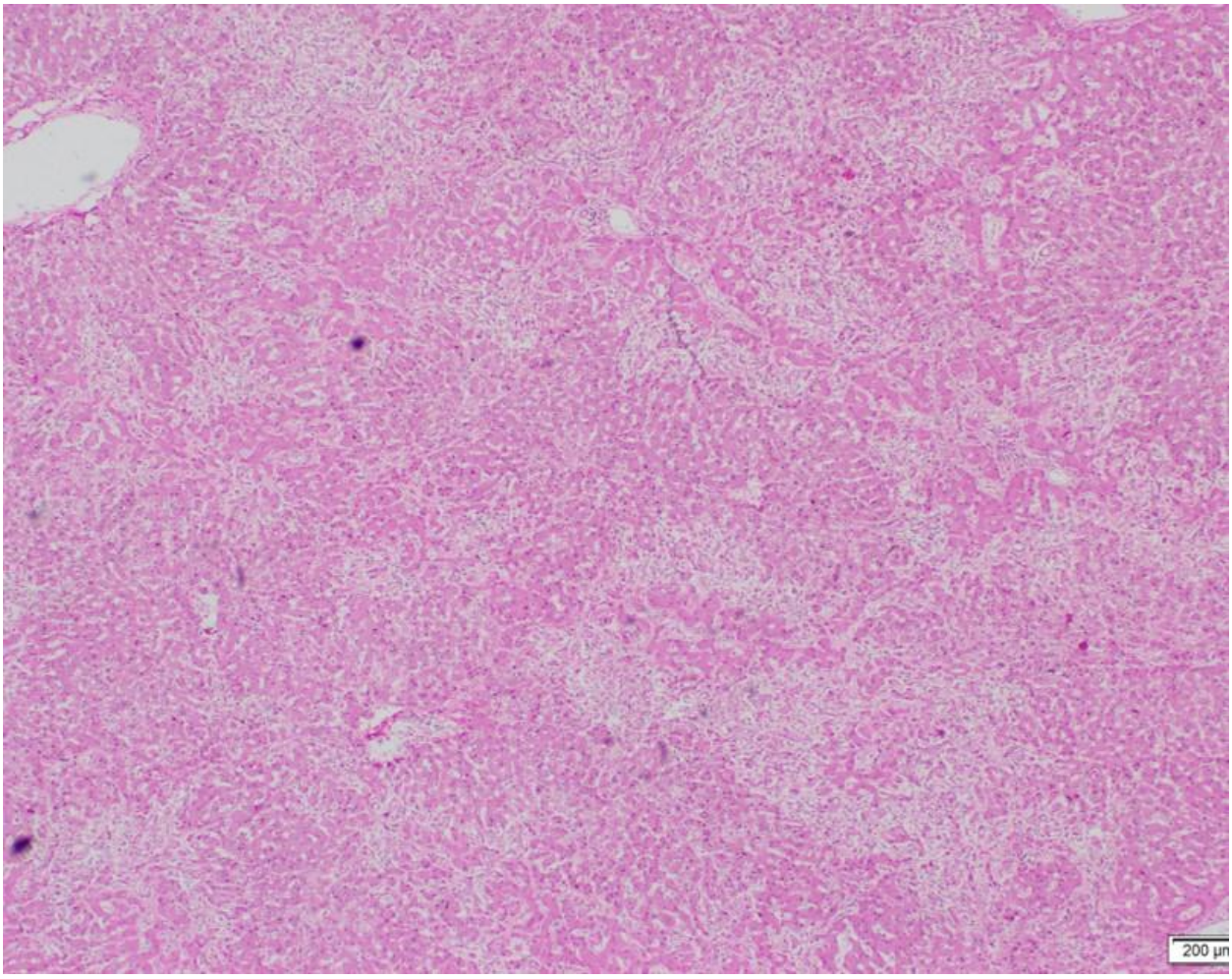

Figure 9: Diffuse bridging necrosis in the liver.

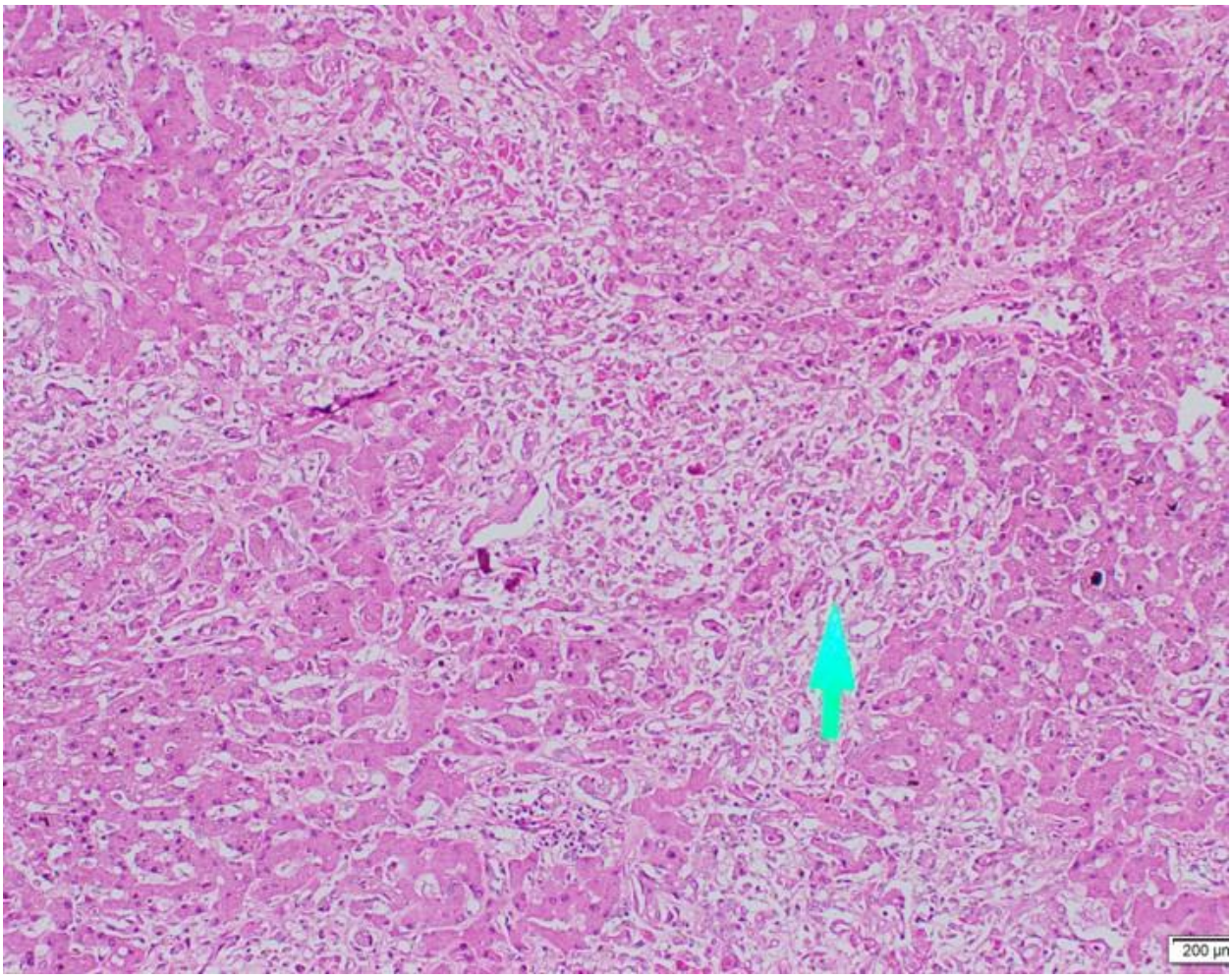

Figure 10: Liver cell necrosis

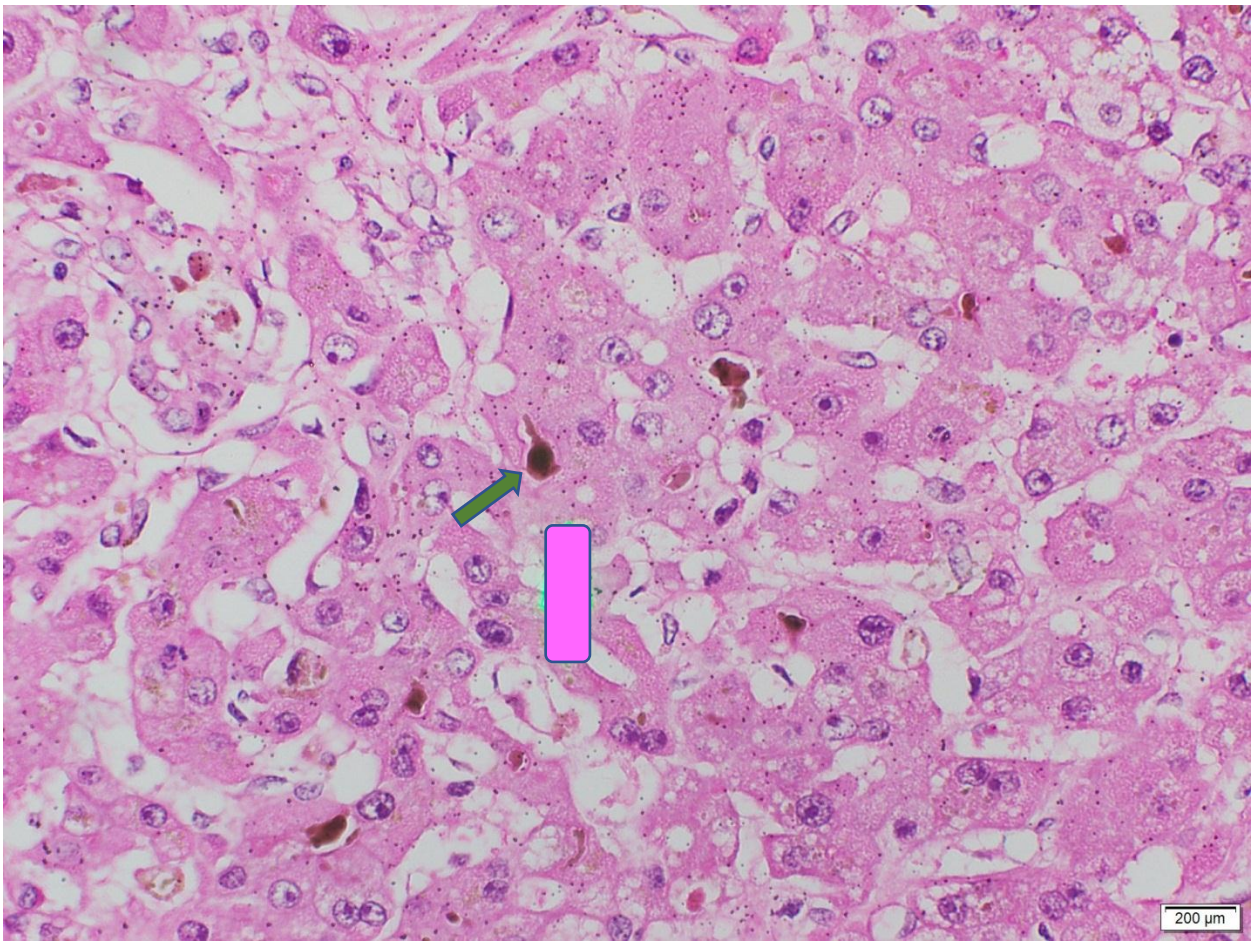

Figure 11: Liver with intra canalicular bile stasis (arrow)

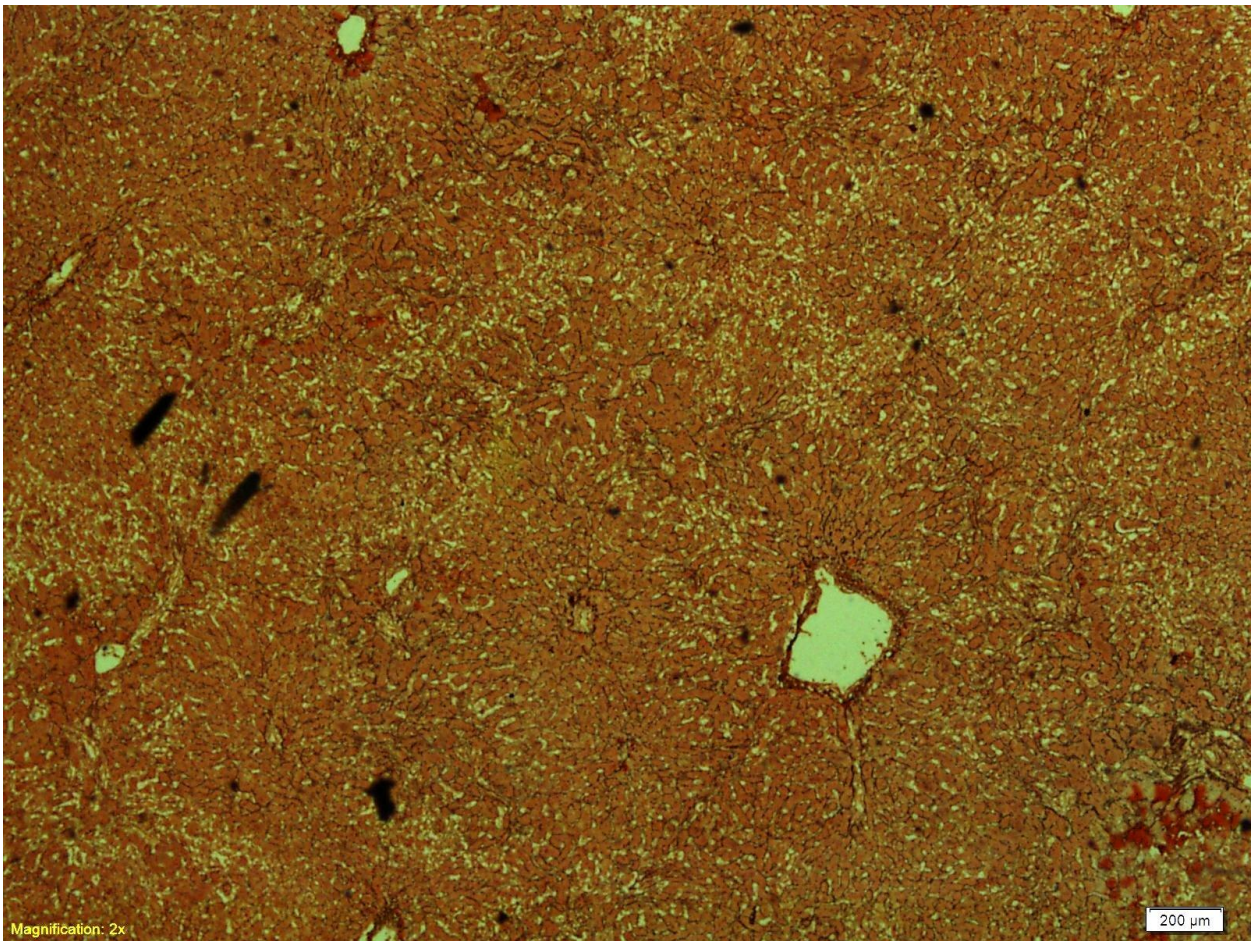

Figure 12: Liver necrosis (Reticulin stain)

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COLOR ATLAS OF

FORENSIC PATHOLOGY

GASTROINTESTINAL SYSTEM

CIRRHOSIS

Cirrhosis is defined as a diffuse process characterized by fibrosis and conversion of normal liver architecture into structurally abnormal nodules. As a result of necrosis of liver cells, which is followed by fibrosis, the liver architecture gets diffusely distorted. Clinical symptoms start with interference of the liver blood flow and impairment of the liver cell function. Most of the patients are asymptomatic, while others have non-specific symptoms like fatigability, anorexia and weight loss. Specific symptoms such as right hypochondrial pain, abdominal distension, ankle swelling, pruritus arises in later stages. Haematemesis and melaena are severe symptoms which occur due to gastrointestinal bleeding as a result of oesophageal varices and clotting derangements. Porto-systemic encephalopathy leads to confusion and drowsiness.

Cirrhosis is usually an end stage disease that has multiple causes; even though regression with remodelling and even restoration of liver function is possible, if the underlying cause is reversed. Alcohol, chronic hepatitis B and C and non-alcoholic fatty liver disease (NAFLD) are the most common causes. Less frequent causes are autoimmune biliary diseases and metabolic conditions like haemachromatosis and Wilson's disease.

Parenchymal nodules and bridging fibrous septa are the two main macroscopic features of cirrhosis that involves most of the liver. Fibrous septa are bands or scars which are formed around adjacent lobules of the liver as a result of long standing fibrosis. Connective tissues and extracellular matrix are distorted in all forms of cirrhosis, due to excessive production of collagen. Perisinusoidal stellate cells in the space of Disse starts getting activated and transformed into myofibroblasts with the onset of the disease. Reactive oxygen species, growth factors, and cytokines are produced by the damaged hepatocytes or by stimulated Kupffer cells and sinusoidal endothelial cells and further proliferation and collagen synthesis progresses. In some forms of cirrhosis, portal fibroblasts are also responsible. Fibrosis is a dynamic process that involves the synthesis, deposition, and resorption of extracellular matrix.

The fibrous bands encircle the parenchyma, so that parenchymal nodules are created. Parenchymal nodules consists of hepatocytes, which are derived from either pre-existent hepatocytes, with replicative senescence, or newly formed hepatocytes, which are derived from stem cells, having the replication capacity, or both. Nodules may range from very small nodules to large nodules. Micro nodules are less than 3 mm in diameter and are seen uniformly over the liver. This type is usually seen in on-going alcoholic damage or biliary tract disease. Macro nodules are over 1 cm in diameter, and are variable in size with normal acini seen within them. These are most often seen in chronic viral hepatitis. Sometimes a mixed picture with small and large nodules is seen. Finally the liver becomes contracted and smaller appearing as a greyish yellow hard mass which weighs about 800–1200 g.

Inflammation and thrombosis of portal veins, hepatic arteries, and/or central veins, cause parenchymal hypo perfusion, leading to parenchymal atrophy. Over compensation of regeneration may sometimes occur with hyper perfusion. This causes loss of sinusoidal endothelial cell fenestrations and the development of portal vein–hepatic vein and hepatic artery–portal vein vascular shunts is seen. Finally, the thin-walled sinusoids are converted into higher pressure, fast-flowing vascular channels, leading to portal hypertension.

Hepatic failure is usually precipitated by imposition of a metabolic load on the liver, such as systemic infection or a gastrointestinal haemorrhage. Cirrhosis may lead to progressive liver failure, complications related to portal hypertension or development of hepatocellular carcinoma, which eventually leads to death.

Macroscopic Examination

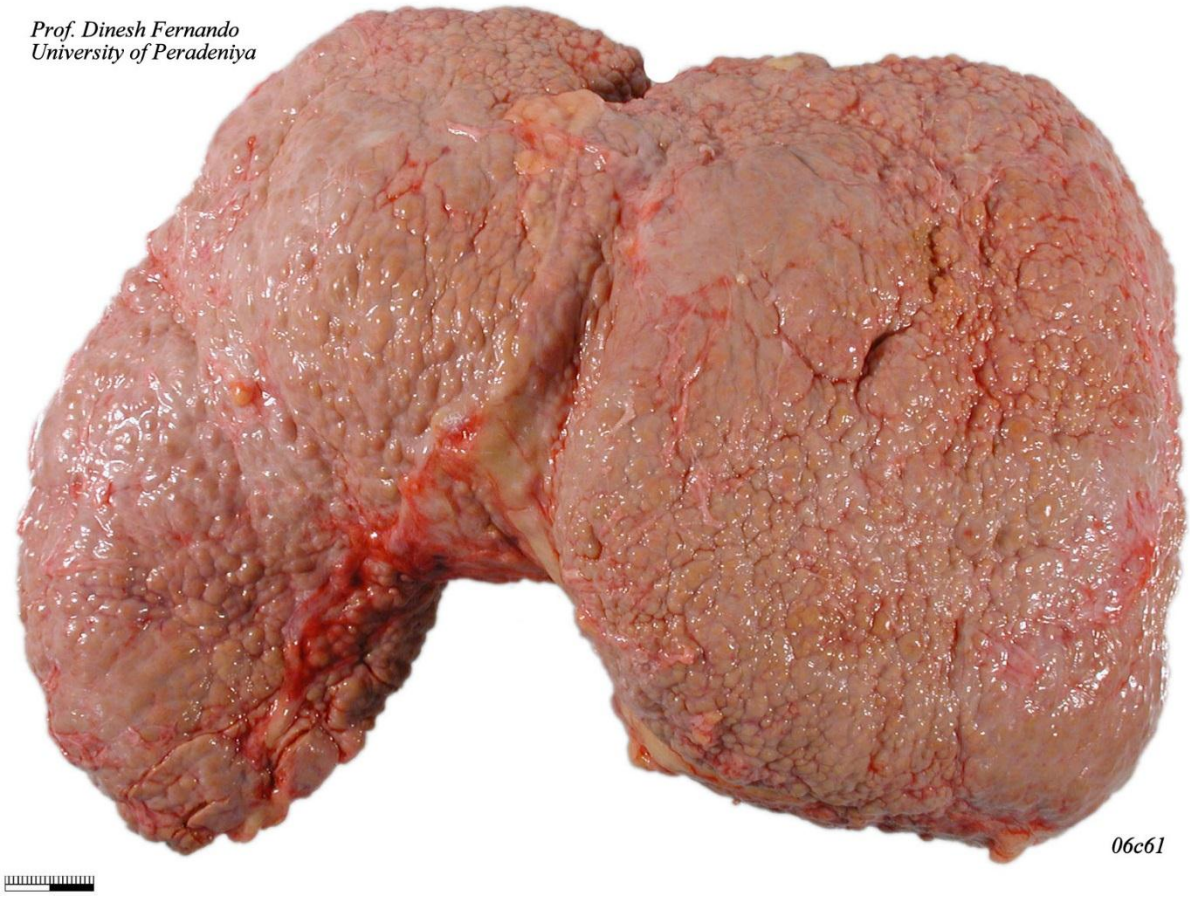
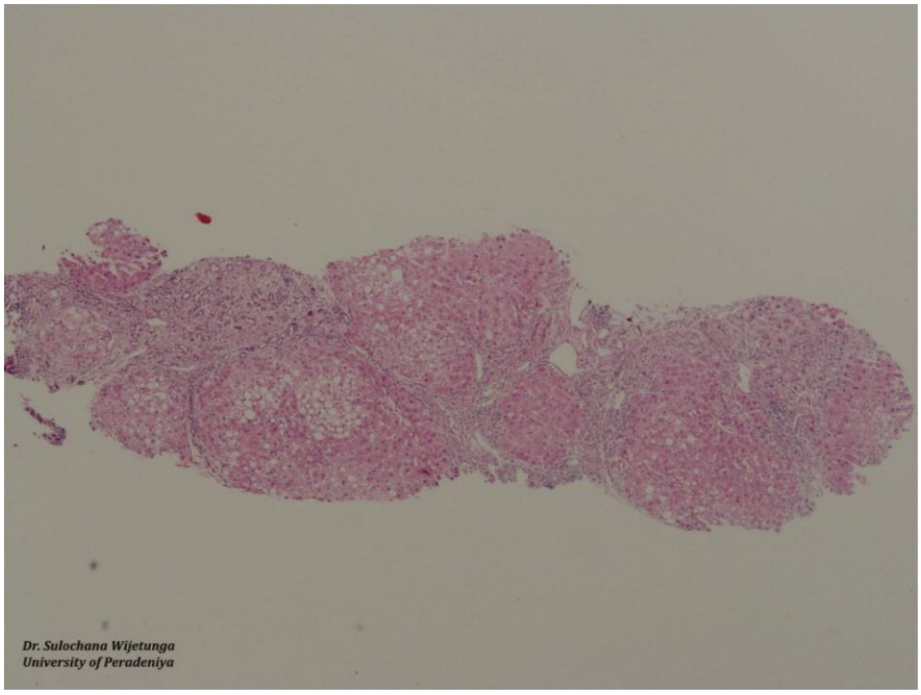
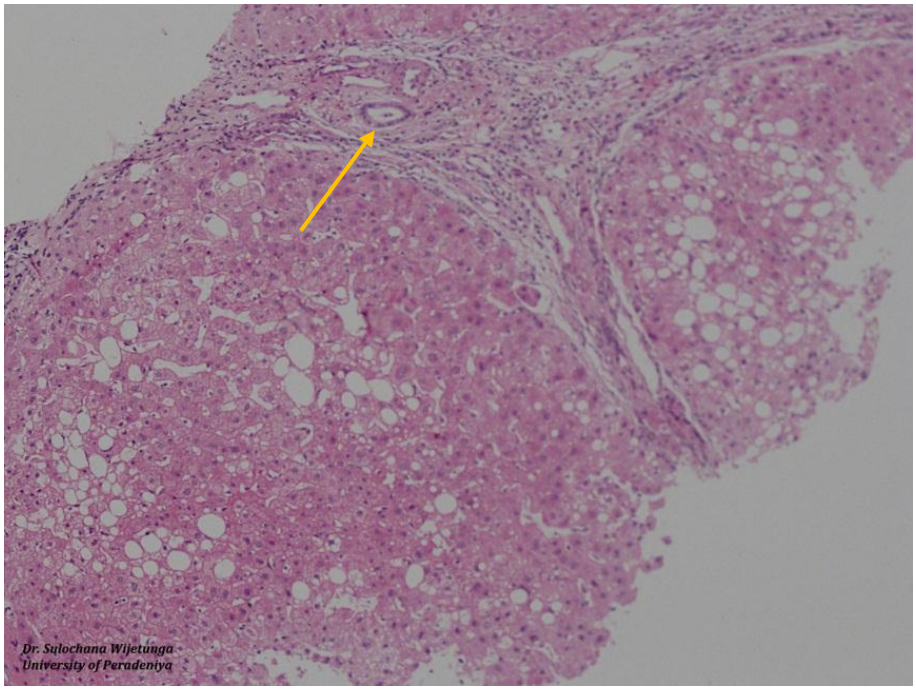


Figure 1: Enlarged 1,800 gram liver with a nodular surface and fibrotic parenchyma (cirrhosis)

Microscopic Examination



(a)



(b)

Figure 2(a,b): Microscopic views of cirrhosis. (a) Scanning power view shows that the normal hepatic architecture is replaced by nodules of hepatocytes. (b) The high power view shows that the nodules are separated by fibrous septae. Note the bile ductular profilerations in the fibrous septae.

Fatty liver disease

Fatty liver disease occurs due to an abnormal accumulation of triglycerides within parenchymal cells which is known as steatosis or fatty change. There are mainly two types of fatty liver disease namely, alcoholic steatohepatitis and non-alcoholic fatty liver disease (NAFLD). Alcoholic fatty liver disease is the commonest worldwide, while NAFLD is commonly associated with metabolic syndrome. Other causes are toxins, protein malnutrition, diabetes mellitus, obesity and anoxia.

Fatty liver disease is graded into four grades depending on the percentage of fat within the hepatocytes. Grade 0 (healthy, <5%), grade 1 (mild, 5%-33%), grade 2 (moderate, 34%-66%), and grade 3 (severe, >66%). Simple hepatic steatosis is a reversible condition that can be corrected by lifestyle modifications such as physical activity and dietary interventions.

There are three categories of liver alterations in fatty liver disease namely, steatosis (fatty change), hepatitis (alcoholic or steatohepatitis) and fibrosis (cirrhosis). Small (microvesicular) to large (macrovesicular) lipid droplets are seen in steatosis. The macrovesicular lipid droplets fill, and expand the cell, displacing the nucleus. As steatosis progresses, the lipid accumulation spreads outward from the central vein to the periportal region. Hepatocyte ballooning, Mallory-Denk bodies and neutrophilic infiltration are the features of hepatitis. These features are mostly associated with alcohol use. Steatohepatitis with fibrosis is the last stage. Similar to the other changes, fibrosis first appears in the centrilobular region, with central vein sclerosis. Next, a perisinusoidal scar appears in the space of Disse of the centrilobular region and then spreads outward, encircling individual or small clusters of hepatocytes.

Early in the course, the liver looks soft, yellow-tan, greasy and enlarged, weighing 4 to 6 kg or more. Persistent damage leads to a contracted fibrotic liver with a nodular appearance and distorted margins.

Macroscopic Examination

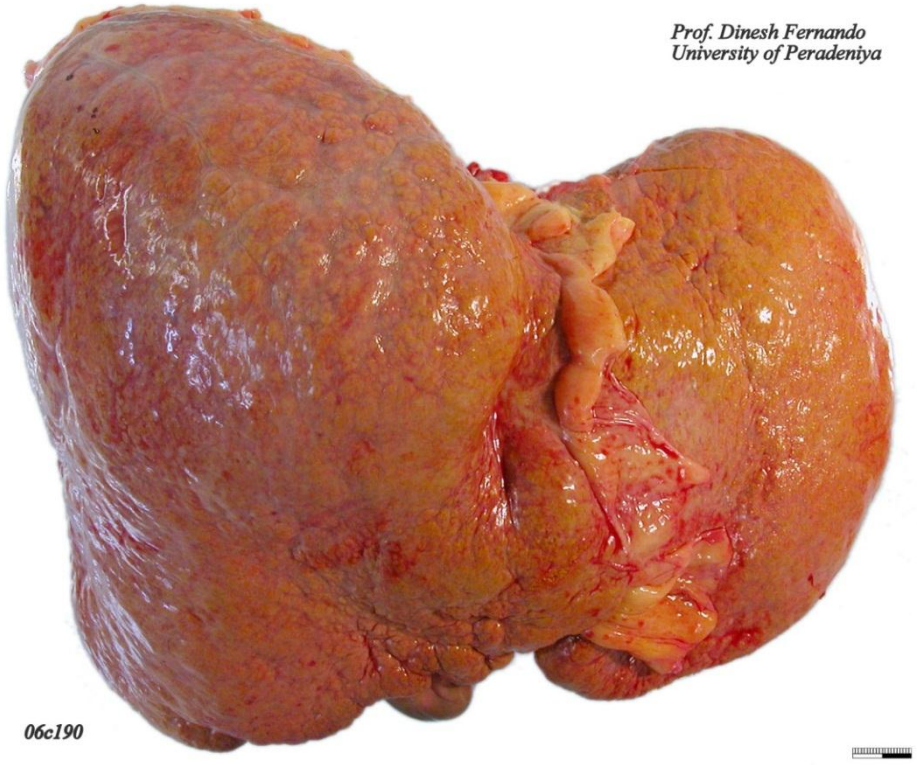
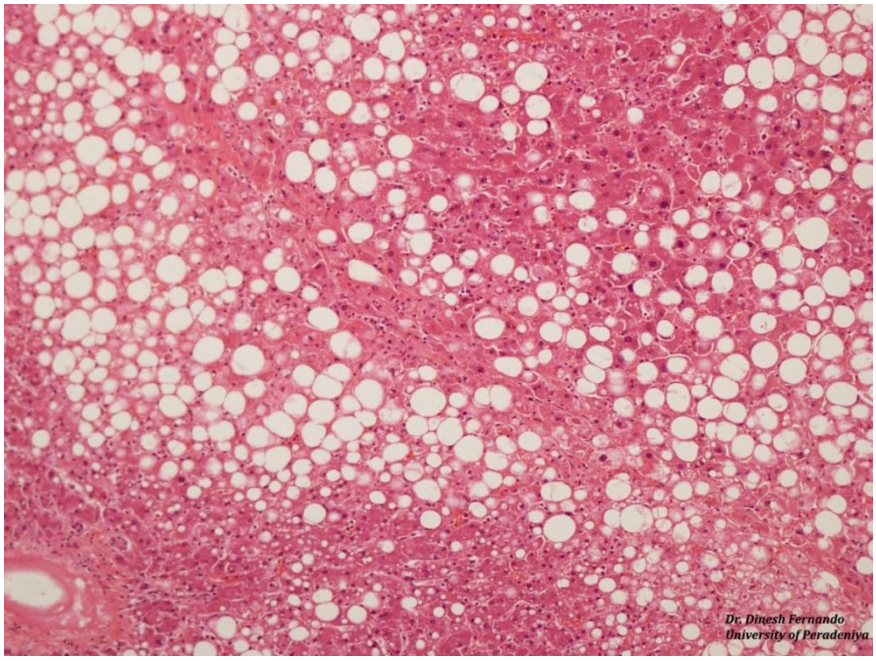


Figure 3: Grossly enlarged liver (3,162 gram) with a nodular surface and distorted margins

Figure 4: Note yellow-tan parenchyma

Microscopic Examination



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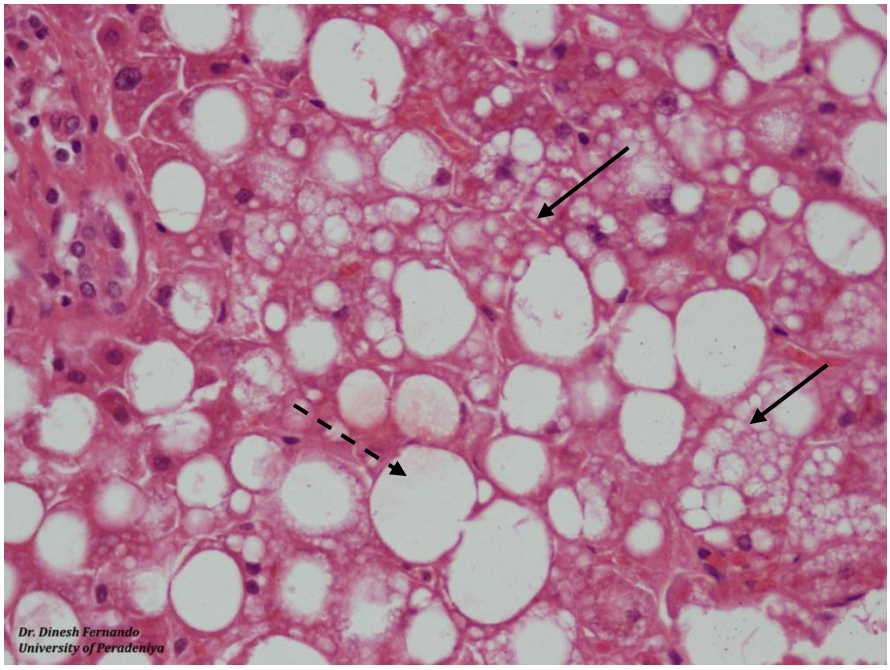


(b)

Figure 5(a,b): Microscopic views of hepatic steatosis. (a) The scanning power view shows many hepatocytes with fatty change seen as empty vacuoles. (b) The high power view shows triglyceride vacuoles occupying the cytoplasm. Some cells show one large vacuole (dotted black arrow) – macrovesicular steatosis. Some cells show many small vacuoles (solid arrows) – microvesicular steatosis.

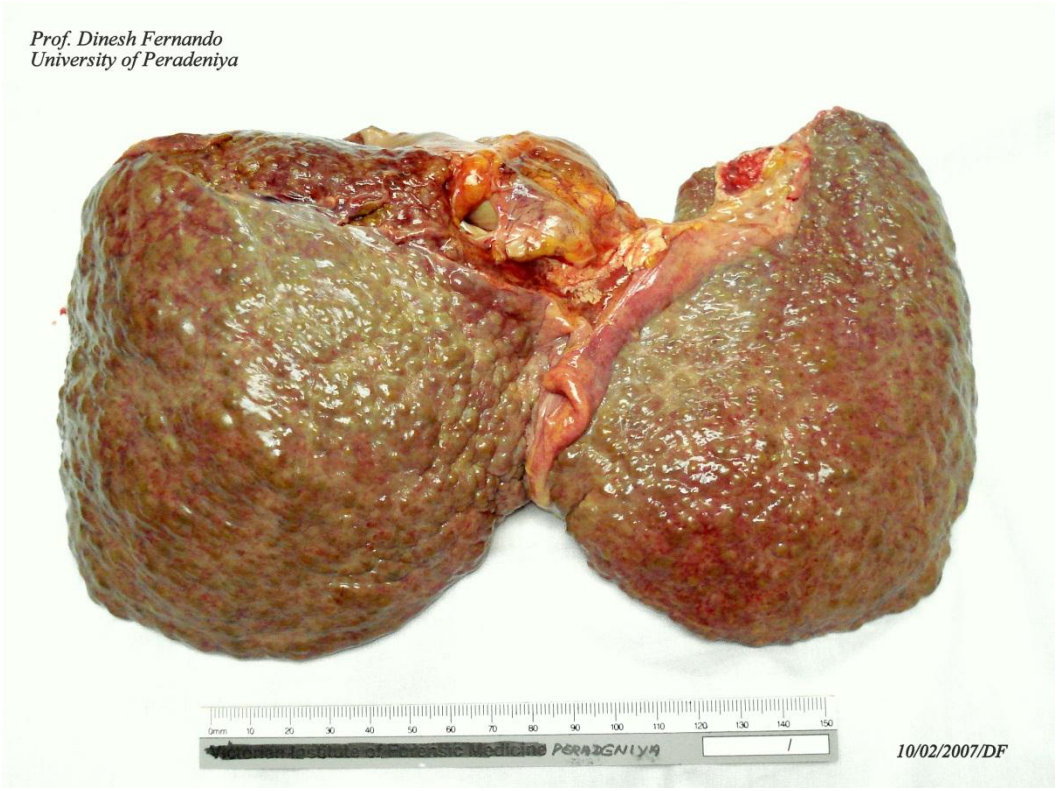
Haemachromatosis

Hereditary haemochromatosis (HH) is a genetic disease, characterized by excessive accumulation and deposition of iron in various organs, eventually leading to fibrosis and functional organ failure. Liver, pancreas and heart are the major organs involved. This disease is mostly seen among Caucasians with a prevalence of 1 in 400. Affected women have a later presentation than men, due to the physiological blood loss. There are many factors which affects the course of the disease, including gender, dietary iron intake, genotype and exposure to hepatotoxins like alcohol. Iron acts as a direct hepatotoxin. There are four genetic variants of HH; the most common form is autosomal recessive, which occurs due to a mutation in the HFE gene. Acquired iron overload results from many causes and is called secondary iron overload.

A normal person has 3-4 g of total body iron, while these patients tend to accumulate iron over the life time, and when it exceeds 20 – 40 g, they become symptomatic. The classic triad of haemochromatosis presents in gross iron overload, with bronze skin pigmentation due to melanin deposition, hepatomegaly and diabetes mellitus. Total iron accumulation may exceed 50 gm and over one third of it is found in the liver. Therefore, cirrhosis is a major manifestation of these patients.

Deposition of hemosiderin is responsible for the morphological changes of the organs. Hemosiderin granules first get deposited in the cytoplasm of the peri-portal hepatocytes of the liver. It appears golden yellow with Prussian blue stain. Further deposition of iron happens in the rest of the lobules, along with bile duct epithelium and Kupffer cells. In the early stages, the liver is slightly larger than normal, dense and chocolate brown in colour. Later fibrous septa are formed leading to cirrhosis. In this stage it is intensely pigmented giving a dark brown to black colour.

Macroscopic Examination



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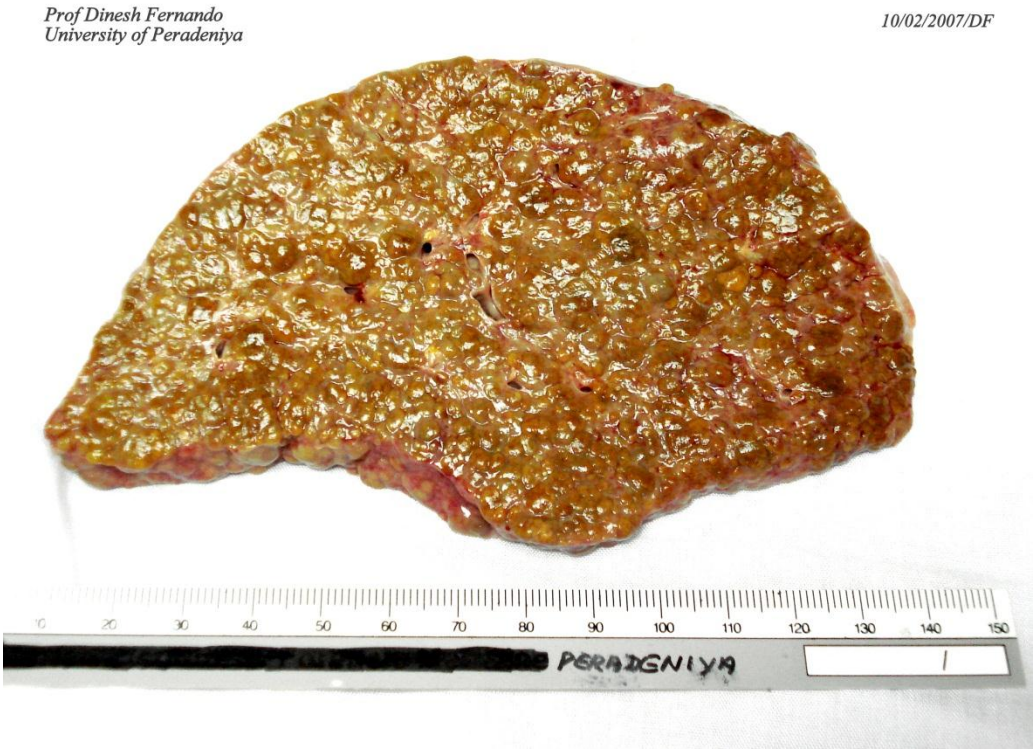

(b)

Figure 6(a,b): Intensely pigmented liver with nodular appearance

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COLOR ATLAS OF

FORENSIC PATHOLOGY

MUSCULOSKELETAL SYSTEM

ACUTE BACTERIAL MYOSITIS

History

A 65-year-old male was admitted to hospital with a history of a fall 10 days previously. After four days he had developed pain in his left shoulder which subsequently spread to his right shoulder and other large joints. He was treated by his GP with Paracetamol and Prednisone. However, the pain had increased and spread throughout his body and involved large muscles which were acutely tender, especially the quadriceps and the muscles of the forearm. Reduction of power and sensation of both the upper and lower limbs with absent reflexes had been observed**.** He had also developed a rash over his chest. When he was admitted to the hospital, he was in a semiconscious state and gradually his condition had deteriorated. In the ICU, he was unresponsive to treatment and gradually lost both sensation and movement in his limbs and started to develop rigidity prior to his death. His blood cultures had shown a growth of *Staphylococcus aureus*.

External examination

The upper part of the chest had an erythematous rash overlying the sternum. The skin of the penis and scrotum was very erythematous. Erythematous rashes were present over both forearms, thighs and calves, which were also swollen. Blisters containing reddish fluid was present on the medial aspect of the right thigh measuring 8cm in diameter. Extensive erythema and dark red mottling were present overlying both hips.

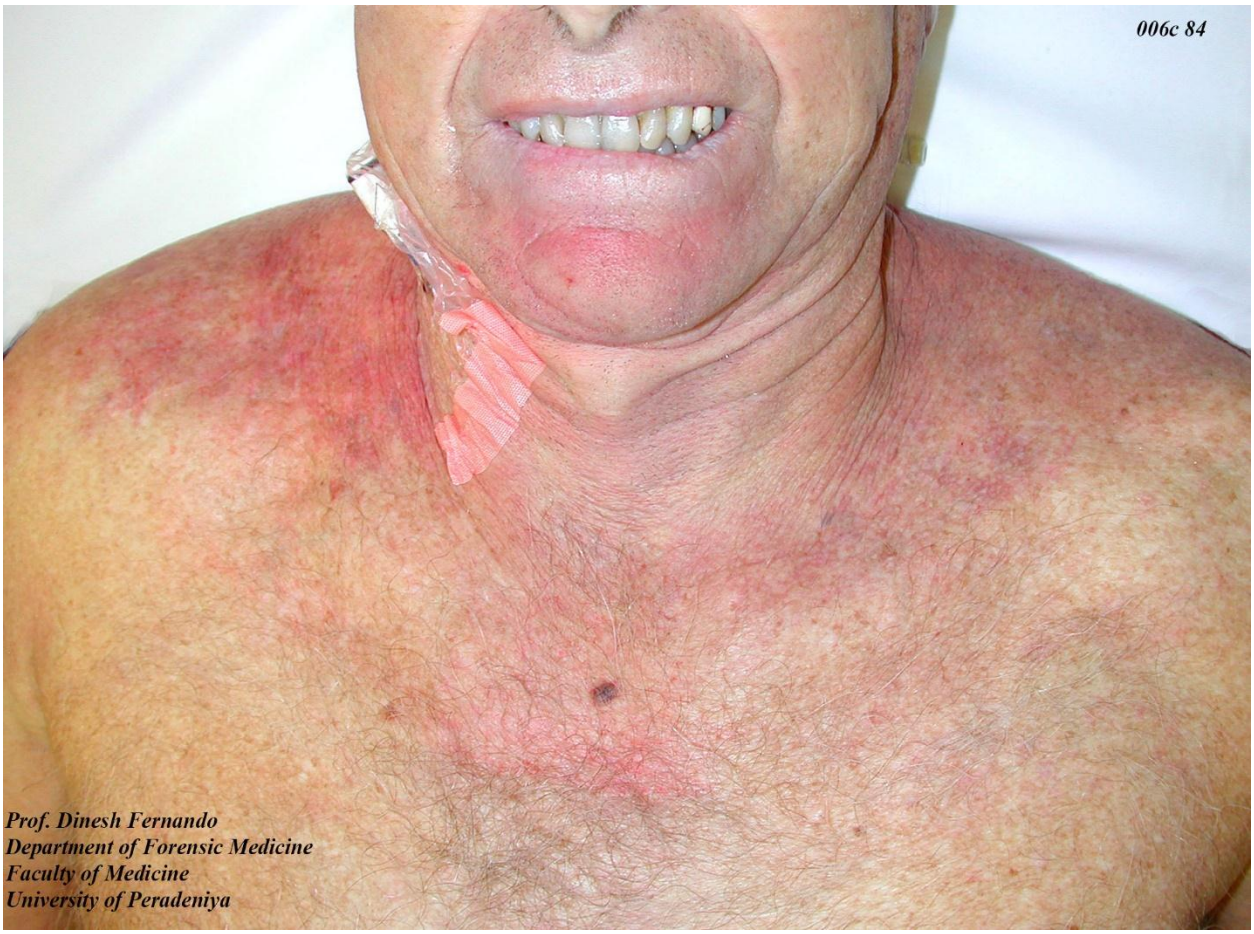

Figure 1: Erythematous rash on the upper part of the chest overlying the sternum

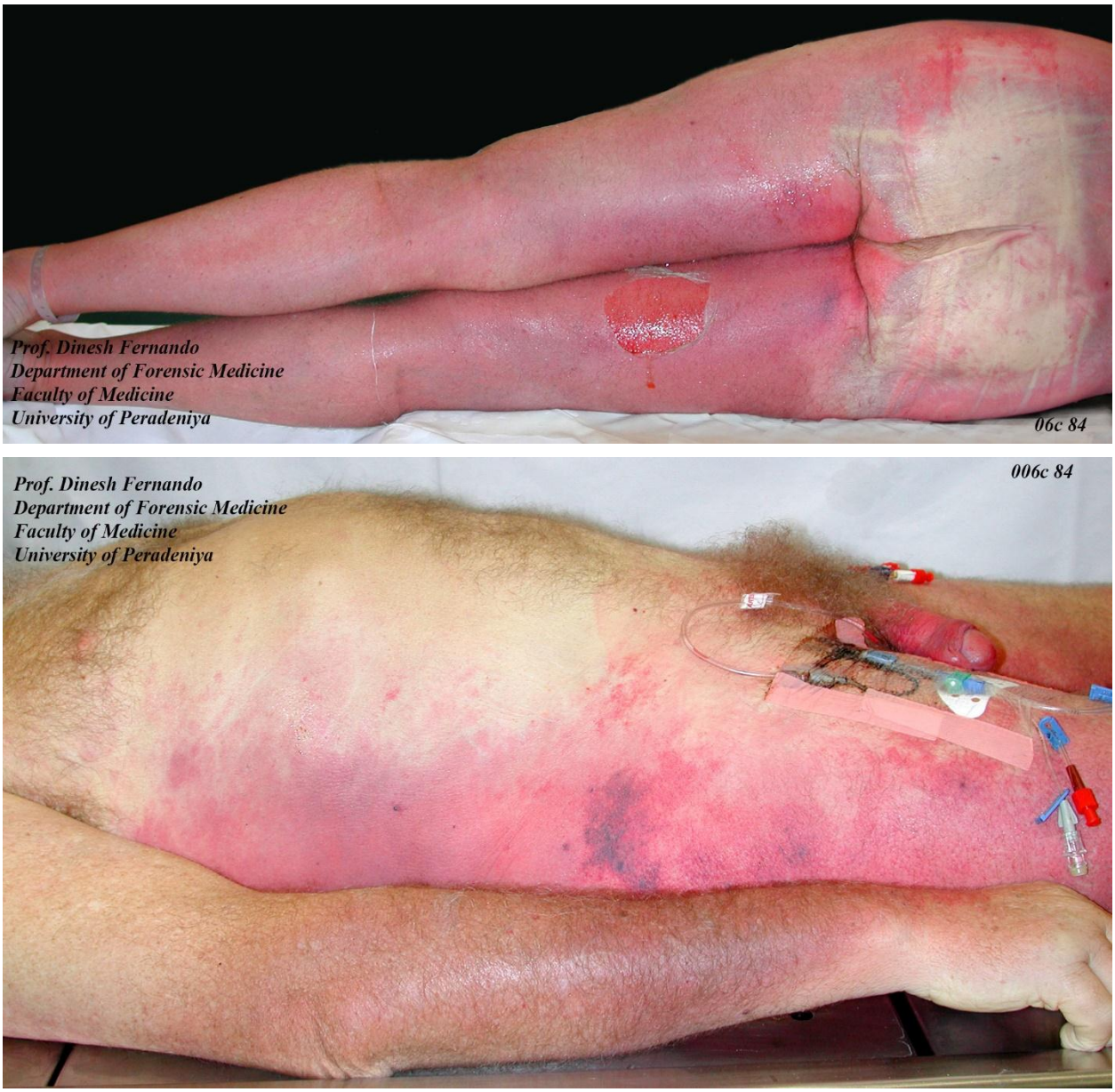

Figure 2: Erythematous rashes over thighs and calves

Internal examination

Respiratory Tract: Each pleural cavity contained approximately 250 ml of serosanguineous fluid. The larynx, trachea and mainstem bronchi had congested mucosal surfaces. The right and left lungs weighed 934 grams and 872 grams respectively. Multiple petechial haemorrhages (Tardieu's spots) were present in the posterior aspect and interlobar fissures.

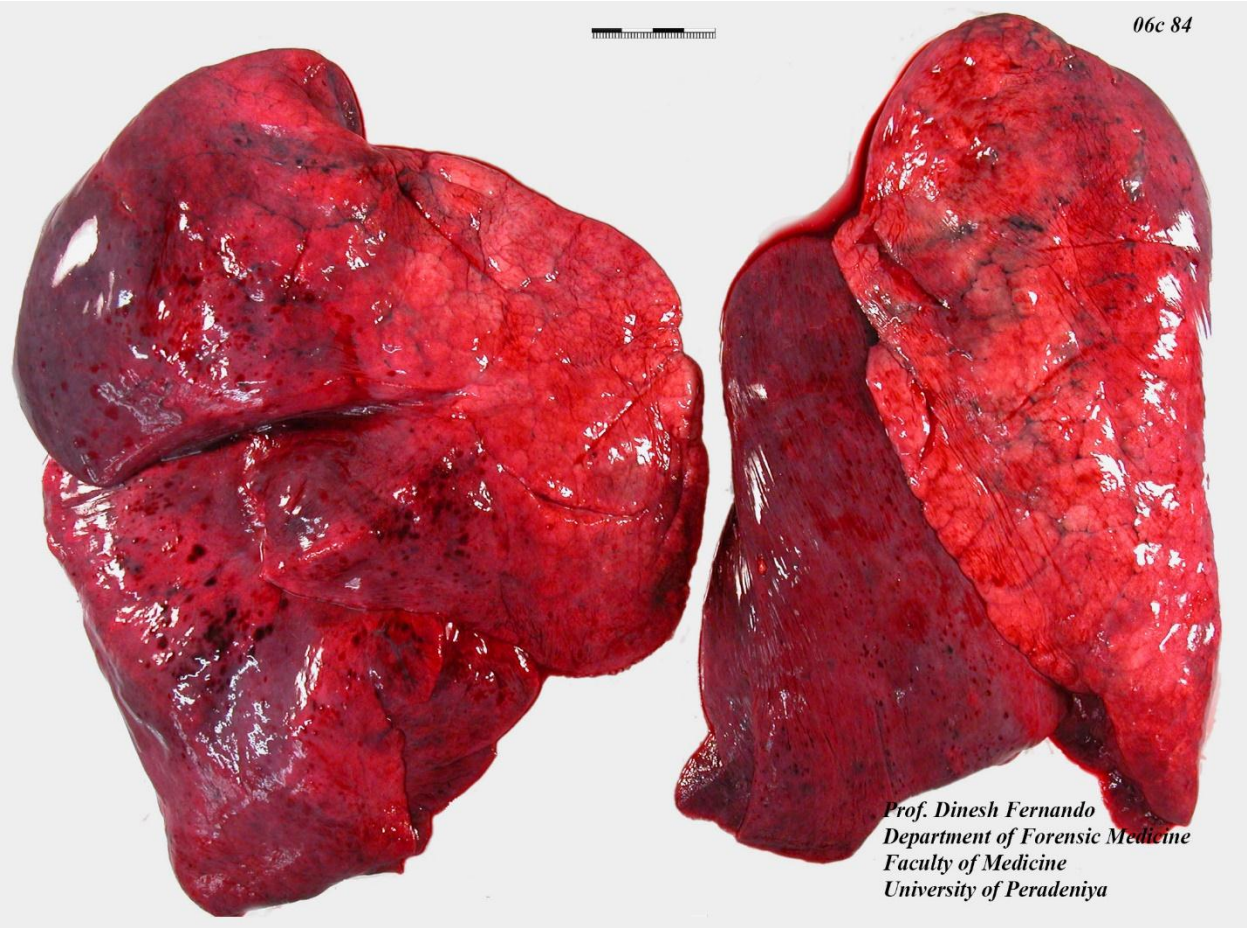

Figure 3: Multiple sub pleural petechial haemorrhages (Tardieu's spots)

Musculoskeletal System: The muscles were soft, red and friable. No localized areas of abscesses were seen.

Microscopic examination – Sections of muscles taken from both thighs, left psoas and left gluteus showed focal acute suppurative myositis

Microbiology – aspirate from both shoulder joints grew *Staphylococcus aureus*

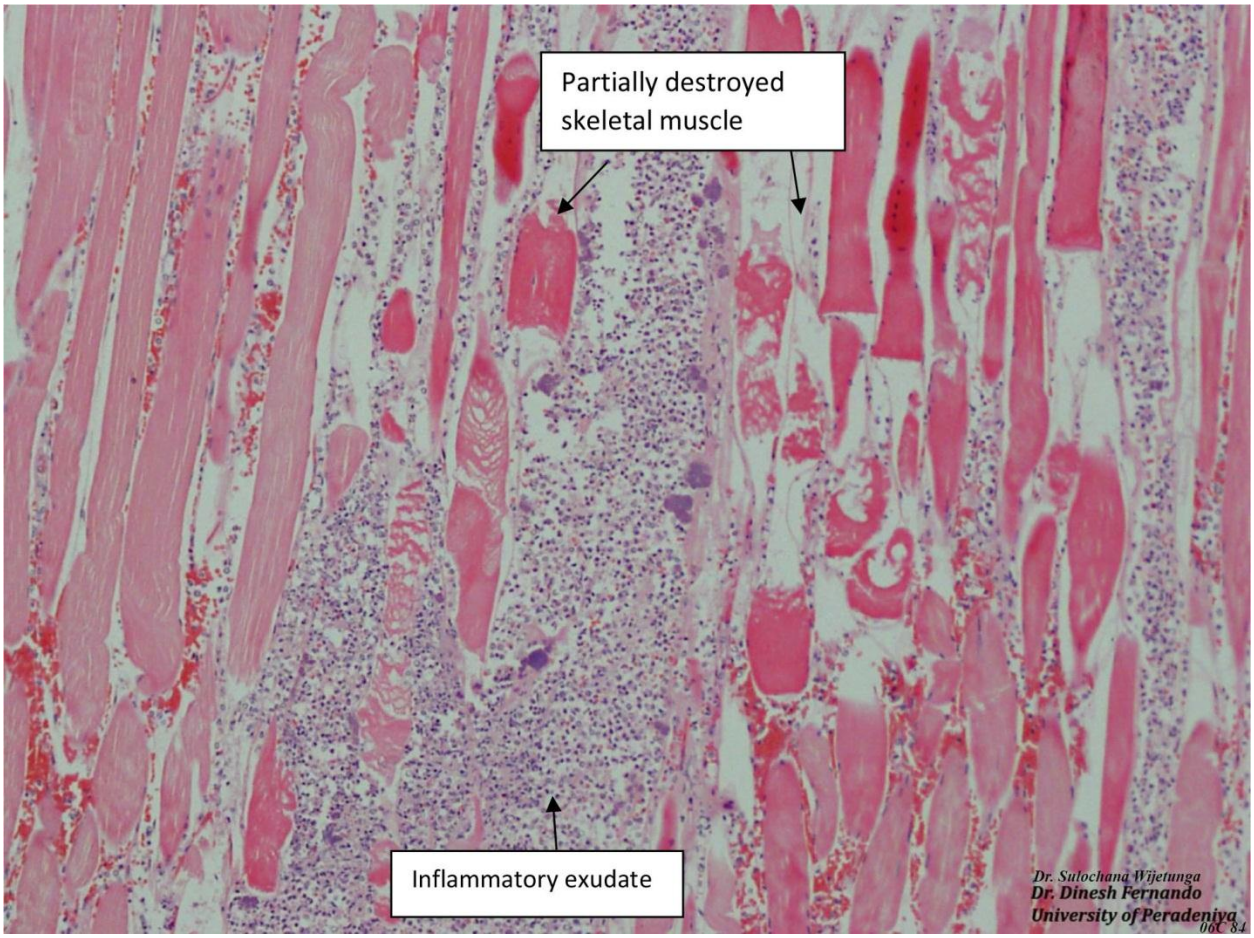

Figure 4: Skeletal muscle fibres infiltrated with an acute inflammatory exudate. The skeletal muscle fibres get destroyed by the inflammatory exudate.

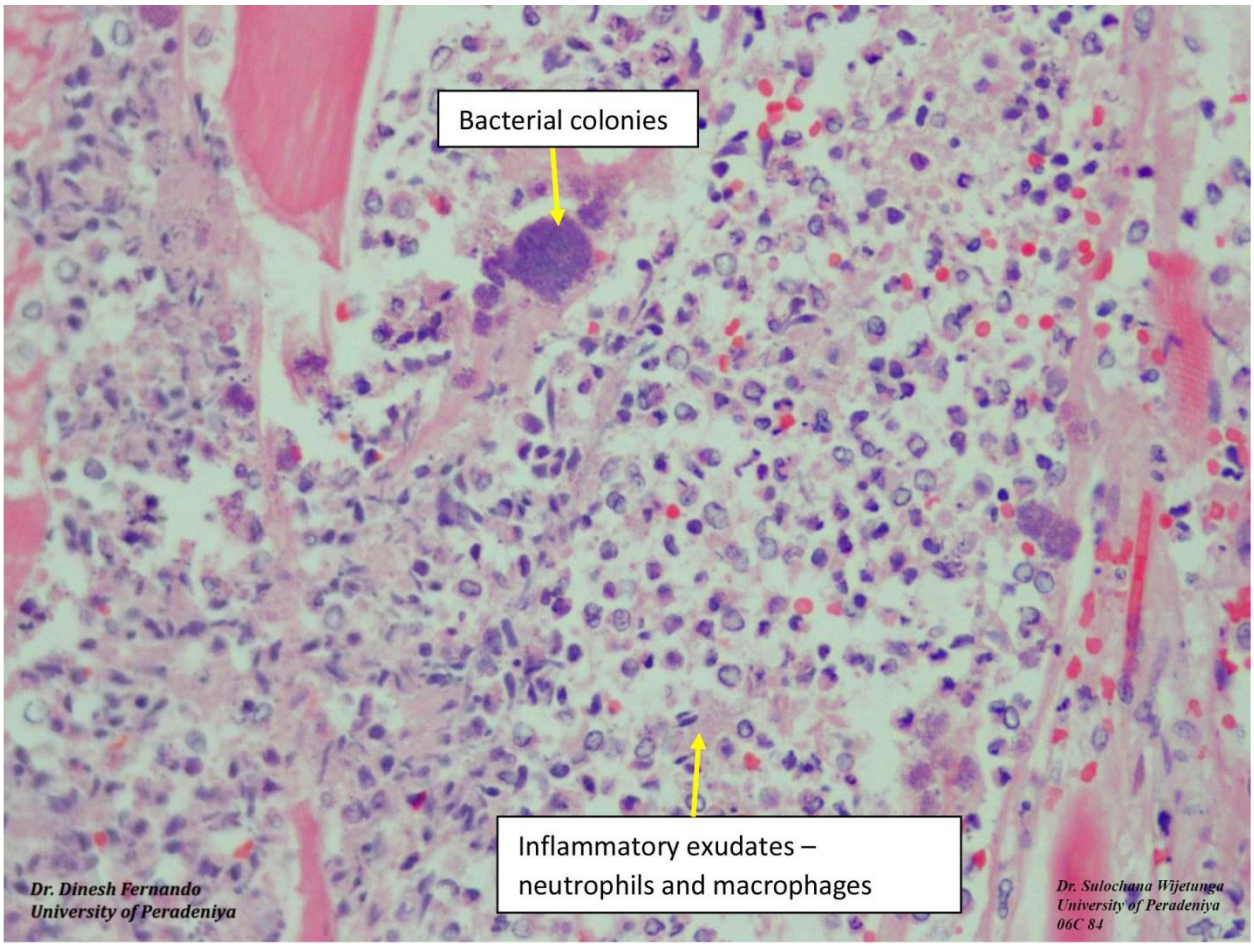

Figure 5: The inflammatory exudate composed of neutrophils and macrophages admixed with bacterial colonies observed under high power

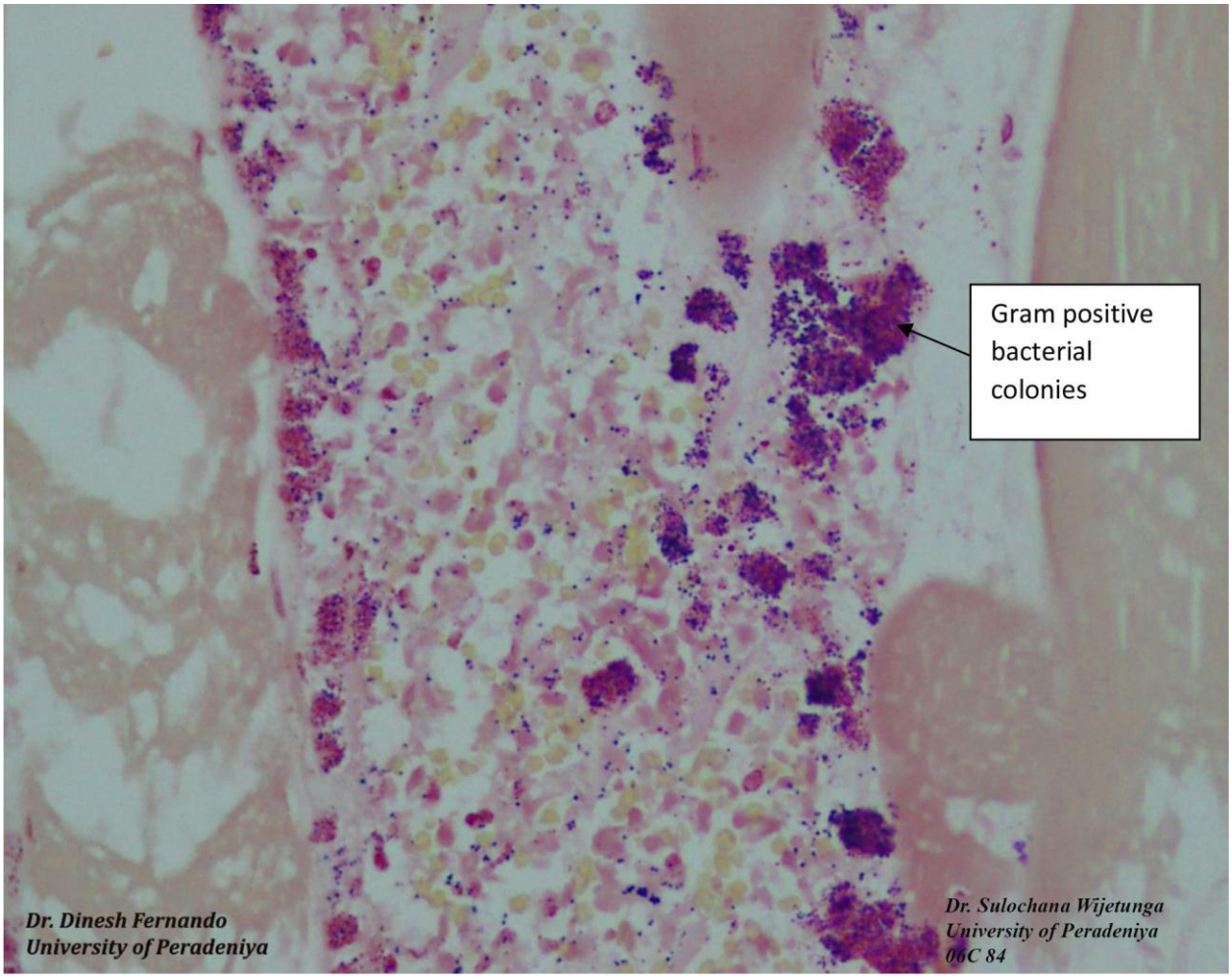

Figure 6: Gram positive cocci bacteria on gram stained sections

Cause of death

Multi organ failure due to staphylococcal septicaemia due to acute bacterial myositis

Bibliography

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Dr. Dinesh Fernando is a merit Professor in Forensic Medicine at the Faculty of Medicine, University of Peradeniya and honorary Judicial Medical Officer, Teaching Hospital Peradeniya. He obtained his MBBS in 1994 with Second class honours from the North Colombo Medical College, Sri Lanka, and was board certified as a specialist in Forensic Medicine in 2004. He obtained the postgraduate Diploma in Medical Jurisprudence in Pathology from London in 2005, and possesses a certificate of eligibility for specialist registration by the General Medical Council, UK. He underwent post-doctoral training at the Victorian Institute of Forensic Medicine, Melbourne, Australia. He has also worked at the Wellington hospital, New Zealand, as a locum Forensic Pathologist and as an Honorary Clinical Senior Lecturer at the Wellington School of Medicine and Health Sciences, University of Otago, New Zealand. He was invited to visit and share experiences by the Netherlands Forensic Institute in 2019

Dr. Sulochana Wijetunge is a Senior Lecturer serving at the Department of Pathology, Faculty of Medicine, University of Peradeniya and Teaching Hospital, Peradeniya. She obtained her undergraduate education at the Faculty of Medicine, University of Colombo, and her postgraduate training from Postgraduate Institute of Medicine, University of Colombo, Sri Lanka. International exposure includes training at the University of Southern California, USA and Royal Marsden NHS Foundation Trust, UK. She has 17 years of experience in undergraduate teaching and 12 years of experience as a board certified histopathologist and a post graduate trainer. She has an interest in forensic histopathology and trains the forensic medicine postgraduate students in Pathology.

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