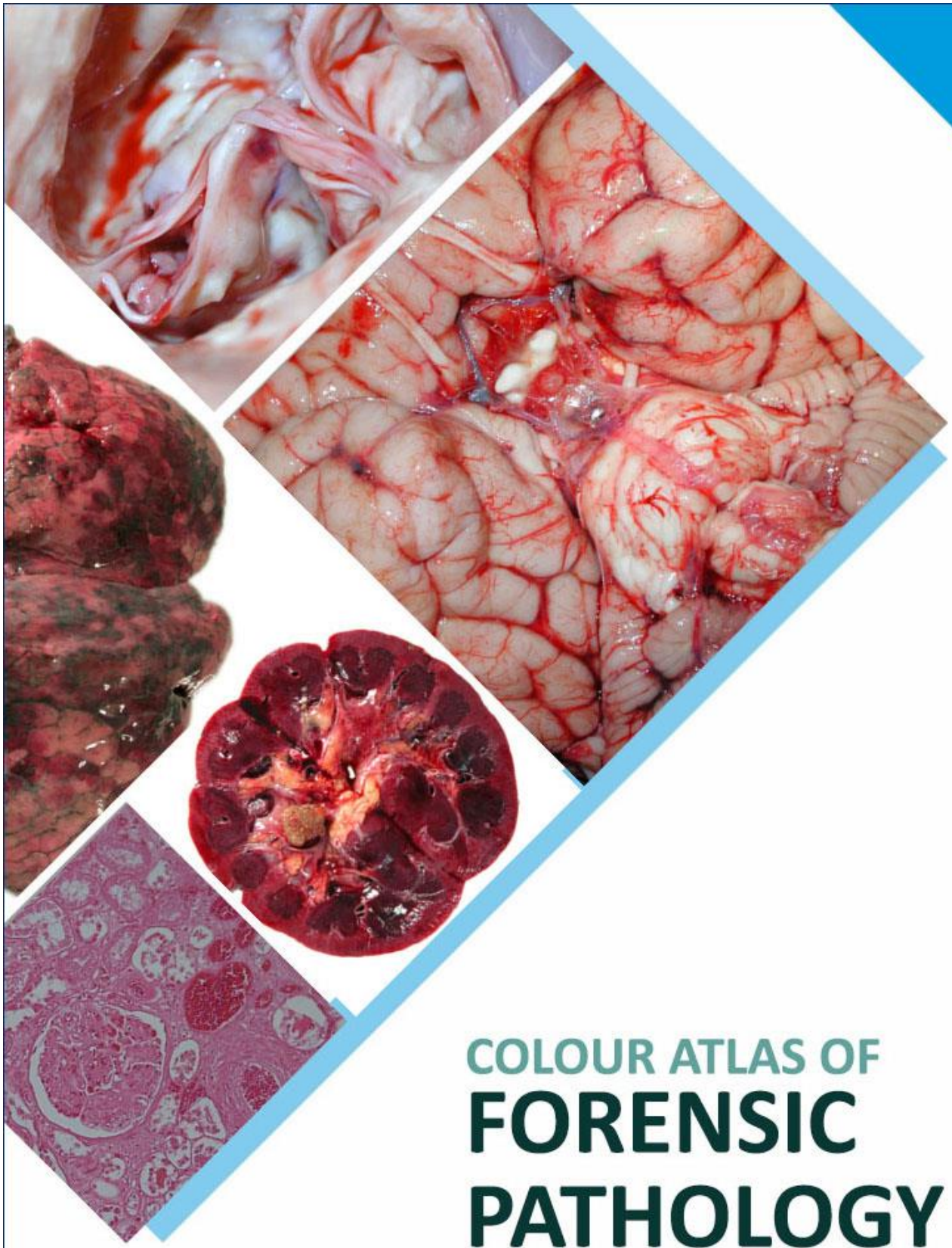

COLOUR ATLAS OF FORENSIC PATHOLOGY

CARDIOVASCULAR DISEASES

COLOR ATLAS OF FORENSIC PATHOLOGY

Version 1

CARDIOVASCULAR DISEASE

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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)
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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. Deshanee Herath is a Temporary Lecturer at the Department of Forensic Medicine, Faculty of Medicine, University of Peradeniya. She obtained her MBBS in 2021 with Second class honours from the Faculty of Medicine, University of Peradeniya.

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, macro and micro images are from cases conducted by Prof. Dinesh Fernando. The compilation of the case and photographs for publication was done by Dr. Deshanee Herath. Ms. Chaya Wickramarathne did a yeomen service in design, lay out and formatting the booklet.

The content herein may be used for academic purposes with due credit given. Any clarifications, suggestions, comments or corrections are welcome.

COLOUR ATLAS OF

FORENSIC PATHOLOGY

ILLUSTRATIVE CASES

Aortic Diseases

AORTIC DISEASES

Aortic diseases

Aorta, the largest artery of the body, begins at the aortic valve and carries oxygenated blood to the systemic circulation. It is divided into 4 parts, namely, ascending aorta, aortic arch, thoracic descending aorta and abdominal aorta. It gives off branches from each part and bifurcates into common iliac arteries.
The aortic wall has three distinct tissue layers: intima, media and adventitia.
Aortic diseases include atherosclerosis, aneurysms, dissection, coarctation and aortitis.

Atherosclerosis

Atherosclerosis is a chronic inflammatory disease of the arteries that causes significant morbidity and mortality since it underlies the pathogenesis of coronary, cerebral and peripheral vascular disease. A combination of inherited and acquired risk factors predisposes to the development of atherosclerosis.
The non-modifiable risk factors for atherosclerosis include age, gender and family history of atherosclerosis related conditions. The modifiable risk factors include hyperlipidaemia, hypertension, cigarette smoking and diabetes mellitus.
Atherosclerosis can cause cardiovascular events, even, in the absence of these overt risk factors. The additional risk factors that identified to be causing atherosclerosis are inflammation, hyperhomocysteinaemia, metabolic syndrome, factors affecting haemostasis and lipoprotein a.
In atherosclerosis, intimal lesions known as atheromata protrude into the vascular lumen obstructing the blood flow. These atheromatous plaques are dynamic lesions that consist of dysfunctional endothelial cells, proliferating smooth muscle cells, macrophages, foam cells and the lipids and necrotic debris released by the death of these cells.
There can be fatty streaks on the vessel wall, composed of lipid filled foamy macrophages, some of which can progress into advanced lesions. These lesions begin as multiple flat yellow spots, and later coalesce into elongated streaks. Intimal thickening and lipid accumulation are the two key processes in the development of atherosclerosis. The atheromatous plaques are white-yellow in colour and protrude into the vessel lumen. There can be superimposed thrombi over ulcerated plaques which are red brown in colour.
Atherosclerotic plaques can undergo several pathological changes including rupture, ulceration or erosion of the surface leading to thrombosis, haemorrhage into the plaque, atheroembolism and aneurysm formation.
Complications of atherosclerosis include coronary artery disease, cerebrovascular disease, transient ischaemic attacks, peripheral arterial disease, abdominal aneurysm formation and renal artery stenosis.
The most affected vessels are the lower abdominal aorta, coronary arteries, internal carotid arteries and vessels of the circle of Willis. The aorta is affected earliest followed by the other vessels.

History

A 79-year-old female, who underwent surgery of the spine following a fall from height, developed abdominal distention and vomiting associated with metabolic acidosis. A CT of the abdomen showed free air with thickened small bowel. A laparotomy showed an ischaemic colon in the inferior mesenteric artery territory and a perforated sigmoid colon. A subtotal colectomy and formation of transverse colostomy was done. Post surgically, she had deteriorating renal function with increasing acidosis. Following discussions with the family, treatment was withdrawn.

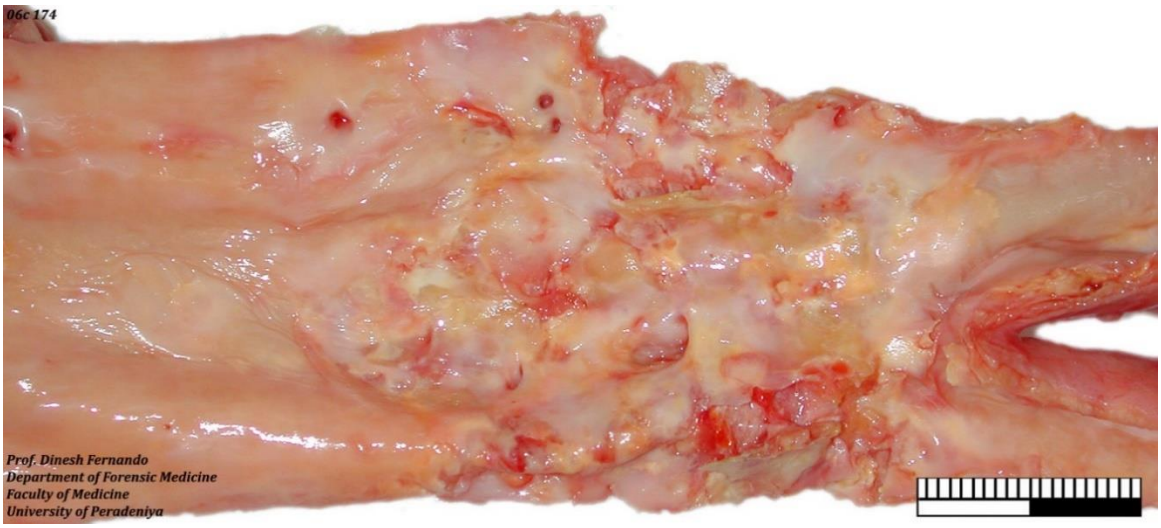

Figure 1: Lower end of abdominal aorta; Note the atheromatous plaques at the origin of the common iliac arteries

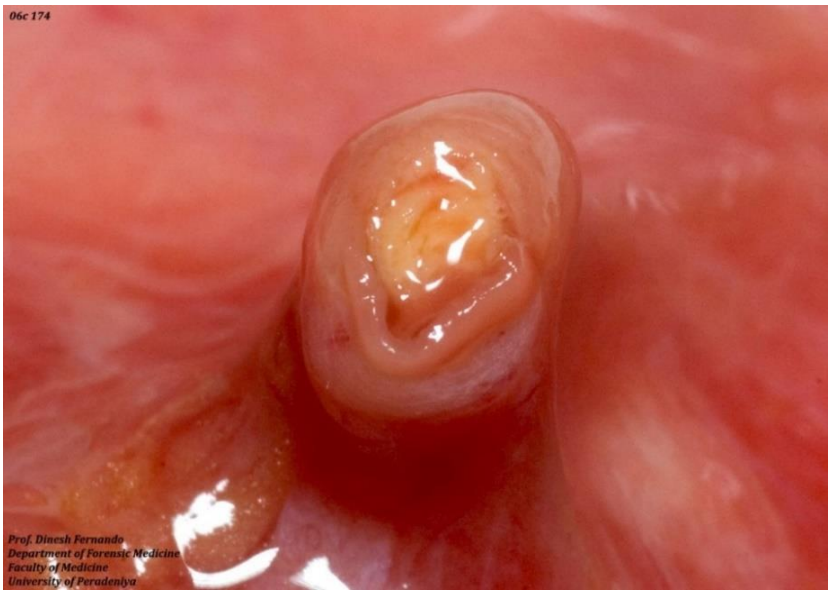

Figure 2: Atheroma obstructing the inferior mesenteric arterial lumen

History

A 42-year-old female, diagnosed with type 2 diabetes mellitus, hypertension, end stage renal failure, and hypercholesterolemia presented with hypotension and tachycardia. Subsequently, she suffered a cardiac arrest and passed away.

Internal examination

The aorta had significant complicated atheroma throughout its length. Bilateral granular contracted kidneys were present.

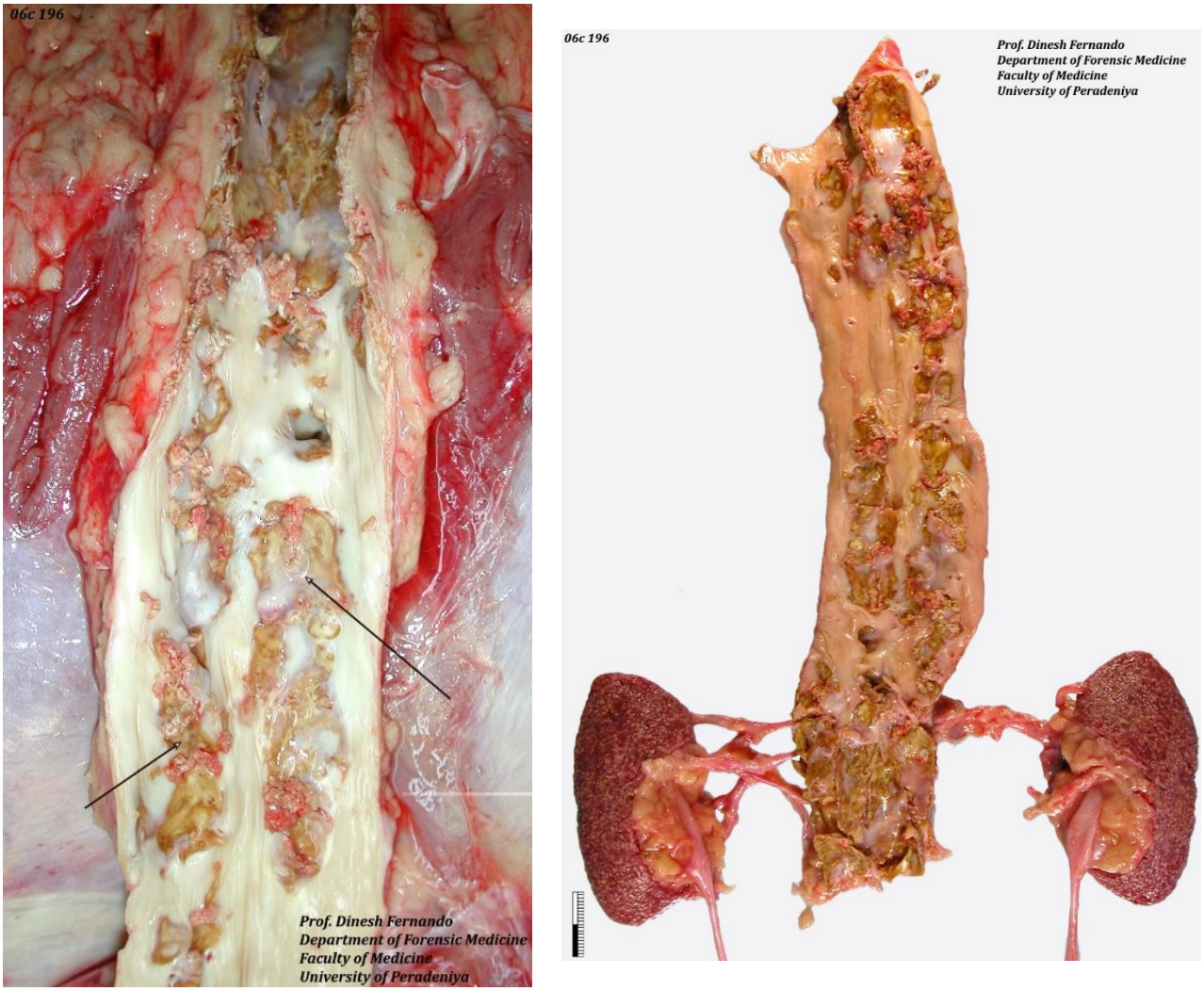

Figure 3: Significant atherosclerosis along the length of aorta
Note the granular surface of the contracted kidneys which have multiple renal arteries on the left side

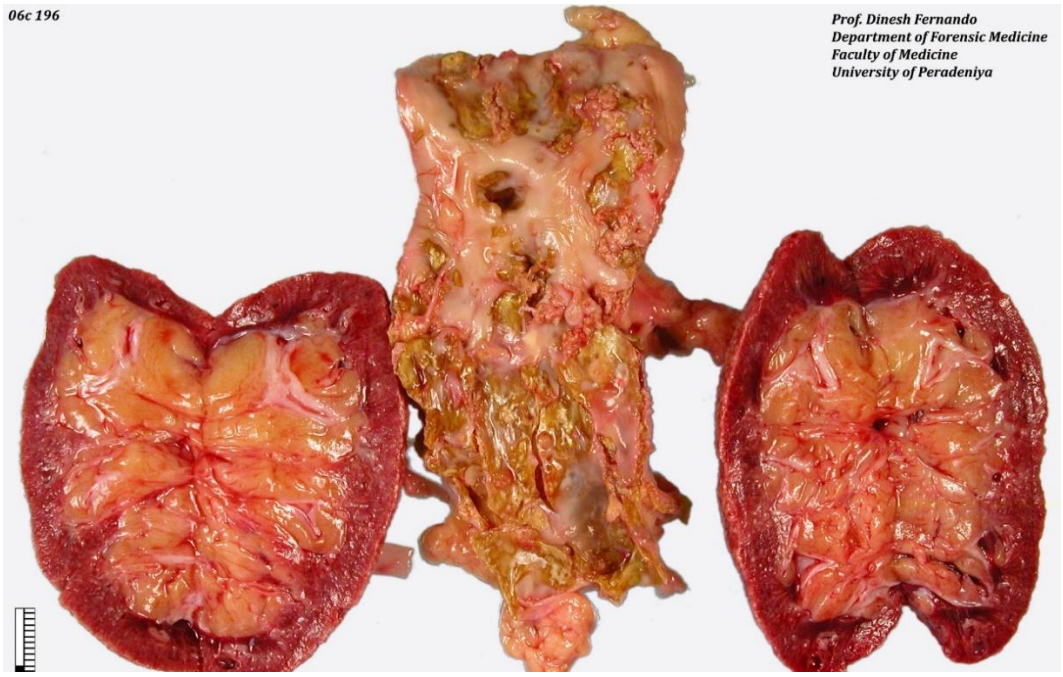

Figure 4: Origin of the renal arteries from the aorta; renal ostia cannot be identified due to the atheromatous plaques. Note the thin cortex and increased fat in the renal sinus.

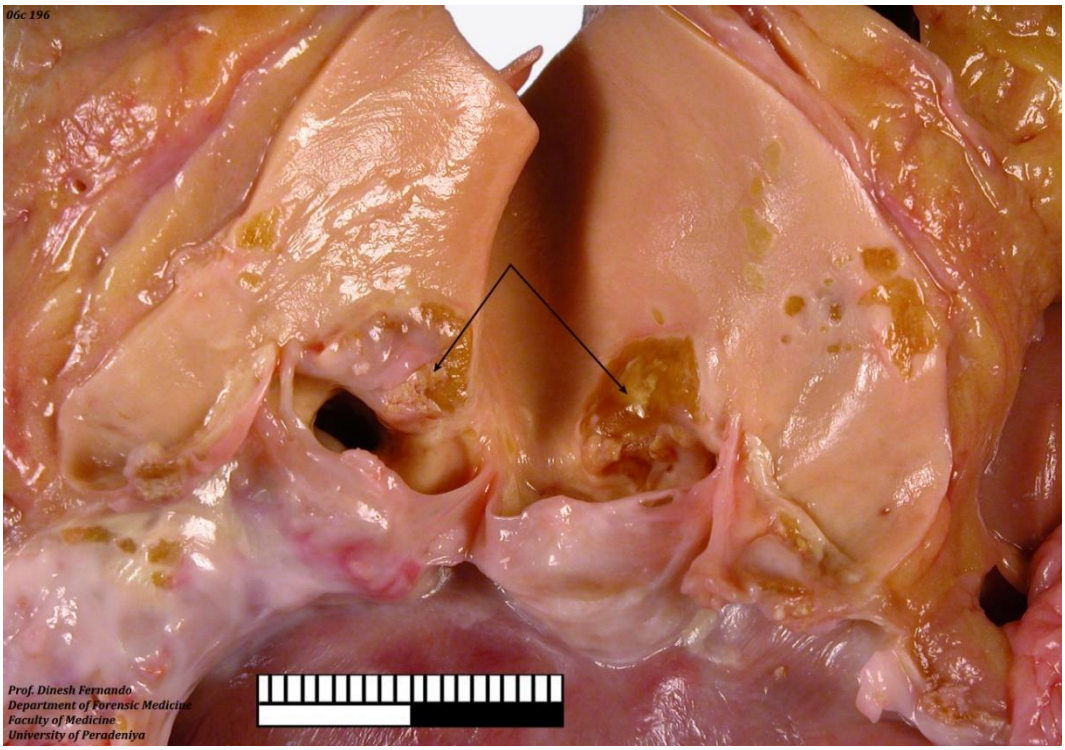

Figure 5: Aortic valve; note the atheromatous plaques above the coronary ostia

Aortic aneurysms

Aneurysms are defined as a focal dilatation in an artery, with at least a 50% increase over the vessel's normal diameter. They can be classified into true and false aneurysms. The wall of a true aneurysm is formed by all three layers of the vessel wall. In a false or pseudo aneurysm, only the outer layer of the vessel wall forms the wall.

The two major causes of aortic aneurysms are atherosclerosis and hypertension. Abdominal aortic aneurysms are mostly caused by atherosclerosis, while ascending aortic aneurysms are most commonly associated with hypertension. Trauma, vasculitis, congenital defects like fibromuscular dysplasia and infections are the other causes for aneurysms.

Abdominal aortic aneurysms

Abdominal aortic aneurysms are common and are potentially life threatening. They occur more frequently in men who are older than 65 years and in patients with peripheral vascular disease. An enlargement of the diameter of the abdominal aorta to 3 cm or more can be considered as an aneurysm.

These are usually (90%) located below the renal arteries (infrarenal) and can be saccular or fusiform in shape. Usually there is severe complicated atherosclerosis, associated with destruction and thinning of the aortic media. Proteolytic degradation of aortic wall connective tissue, inflammation and immune responses, biochemical wall stress and molecular genetics are identified as mechanisms contributing to the development of an abdominal aortic aneurysm. Inflammation, with infiltration by lymphocytes and macrophages, thinning of media and marked loss of elastin are some of the microscopic features seen.

Abdominal aortic aneurysms commonly contain laminated, poorly organized mural thrombi which can embolize and cause end organ damage.

Patients with abdominal aortic aneurysms are usually asymptomatic. They are sometimes discovered incidentally with a palpably pulsating abdominal mass. These can gradually expand and rupture causing a massive, potentially fatal haemorrhage into the peritoneal cavity or retroperitoneal tissues. If the aneurysm obstructs a branching vessel from the aorta, it can cause ischaemic injury to the supplied tissue. eg: iliac (leg), renal (kidney)

History

A 79-year-old male, diagnosed with emphysema, presented with severe shortness of breath and died soon after admission. At autopsy, a large abdominal aortic aneurysmal dilatation which measured 35 x 40 mm was observed as an incidental finding.

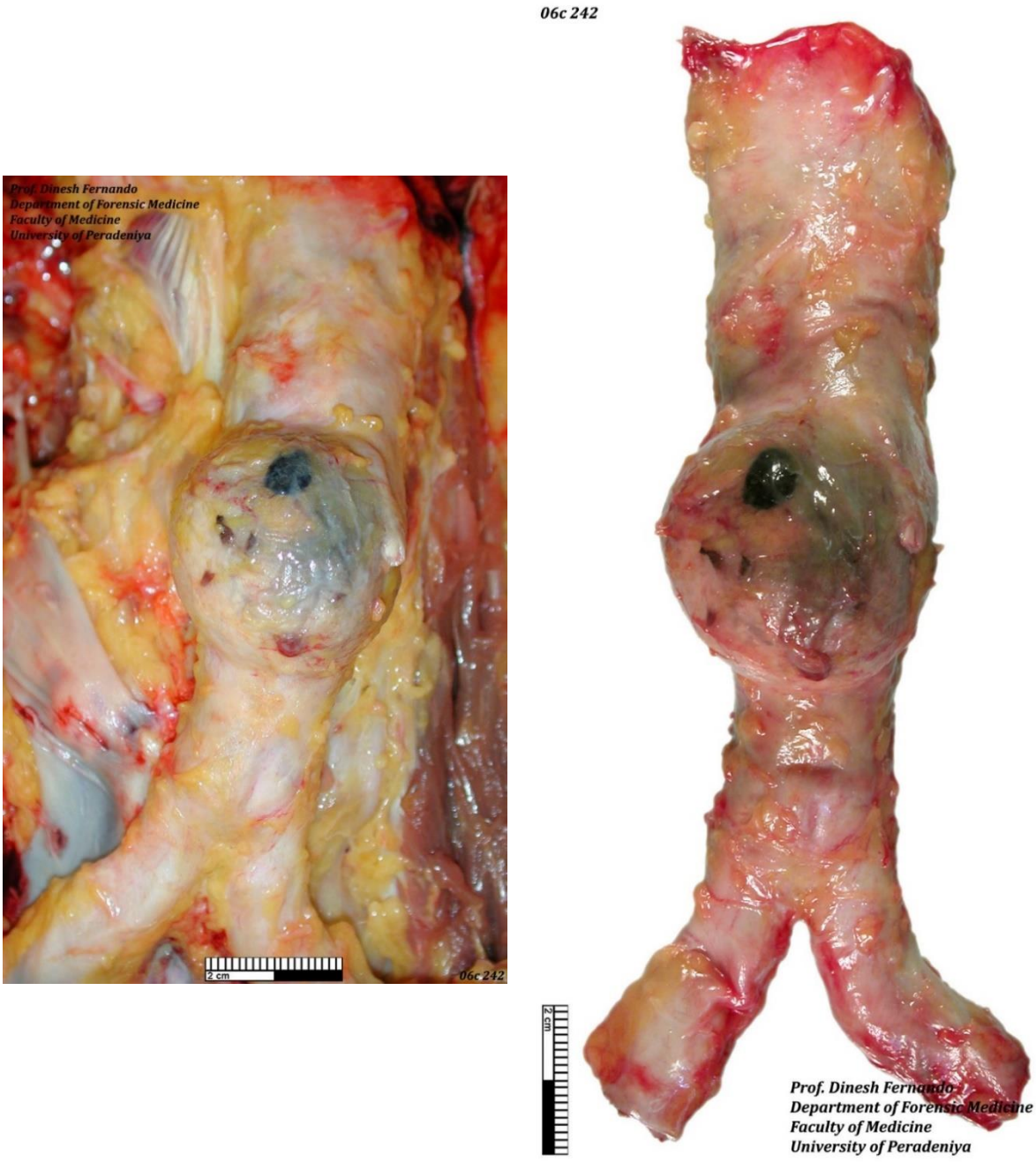

Figure 6: Abdominal aorta with a saccular aneurysm

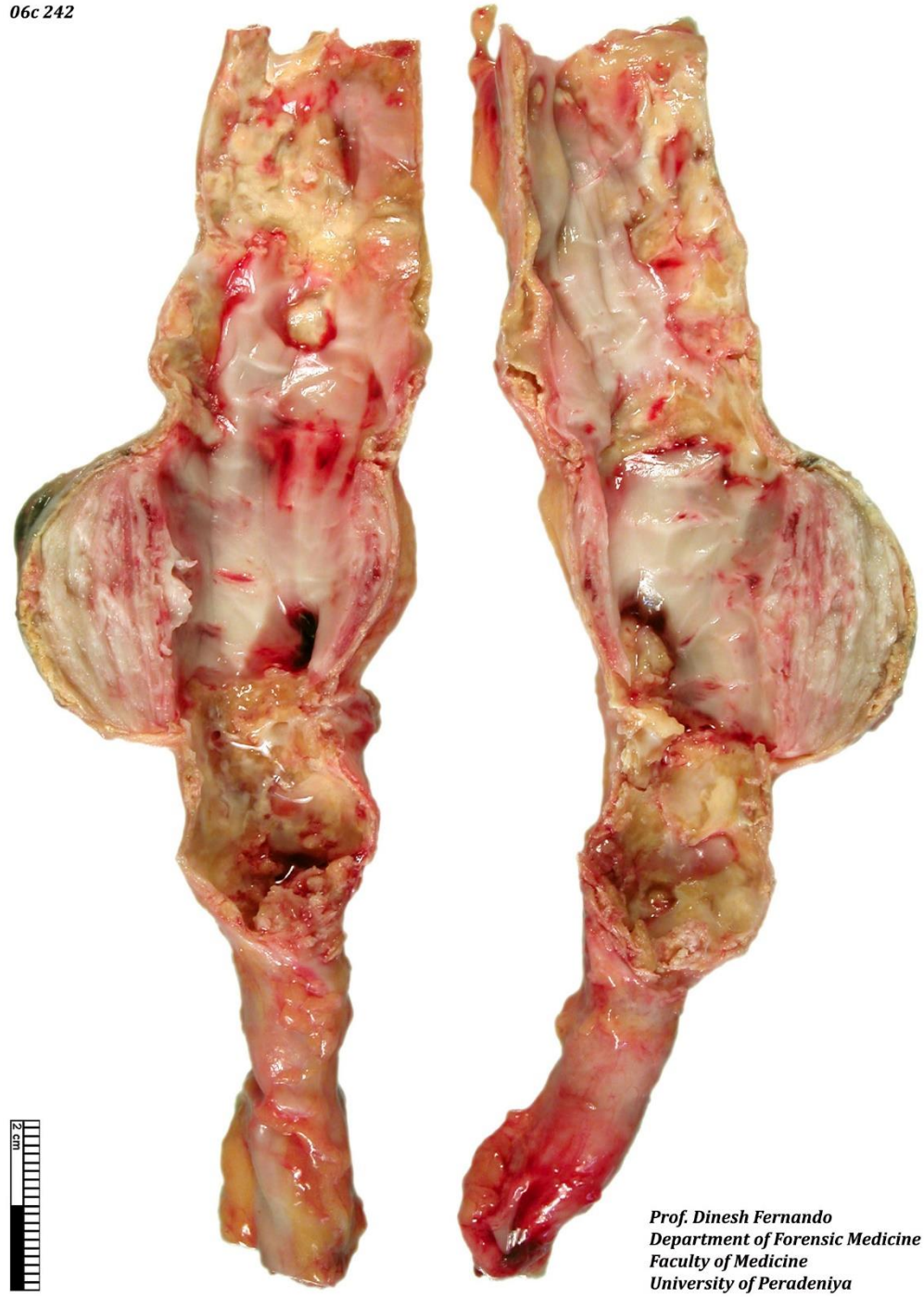

Figure 7: Longitudinal section of the saccular abdominal aortic aneurysm

History

An 86-year-old female, with a past history of coronary artery disease, who underwent an angiogram and stent insertions, was detected to have low blood pressure, 3-4 hours after the procedure. Blood was drained from around the heart and the CT scan demonstrated blood in the abdominal cavity.

Internal Examination

Cardiovascular System: The pericardial sac was intact. An organising blood clot of 75 ml was present in the pericardial cavity overlying the right side of the heart. The heart weighed 412 grams.

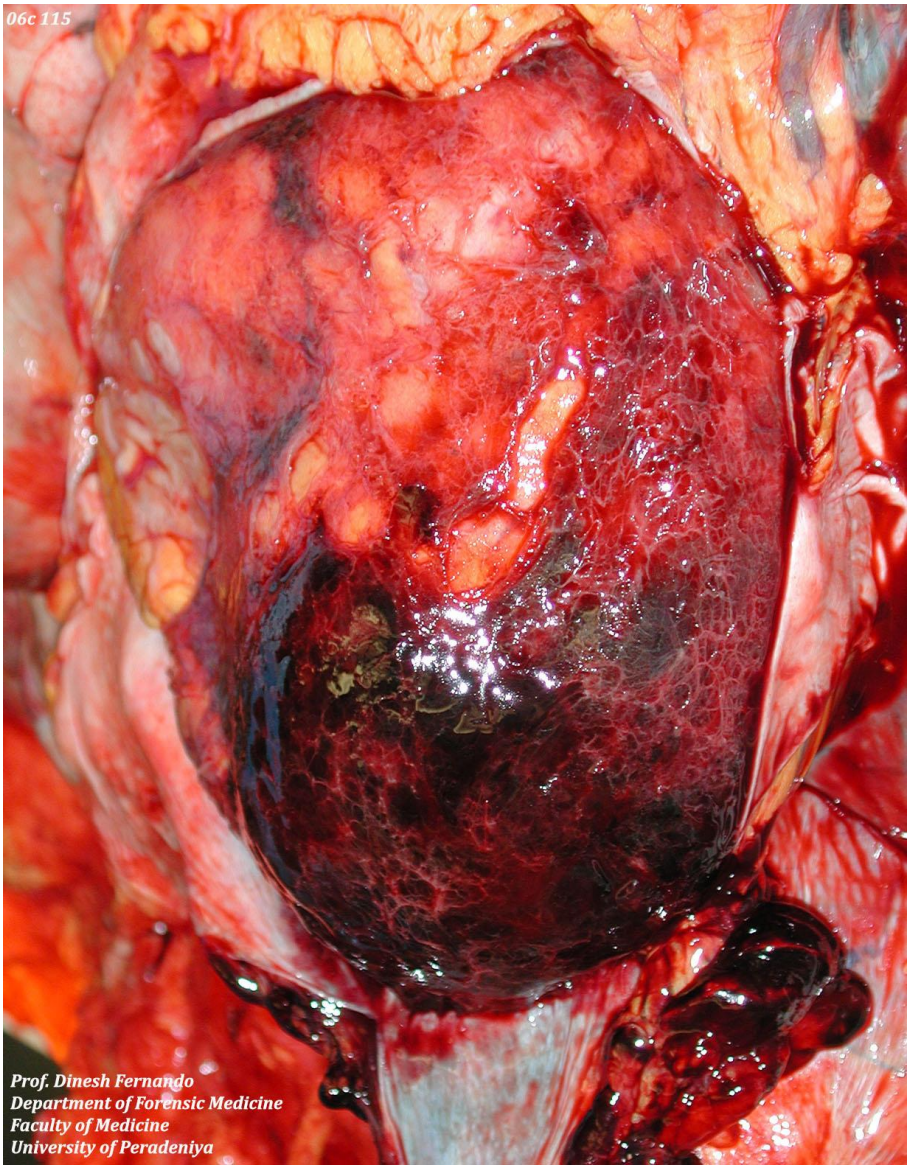

Figure 8: Pericardium is cut open; note the organising blood clot in the pericardial cavity

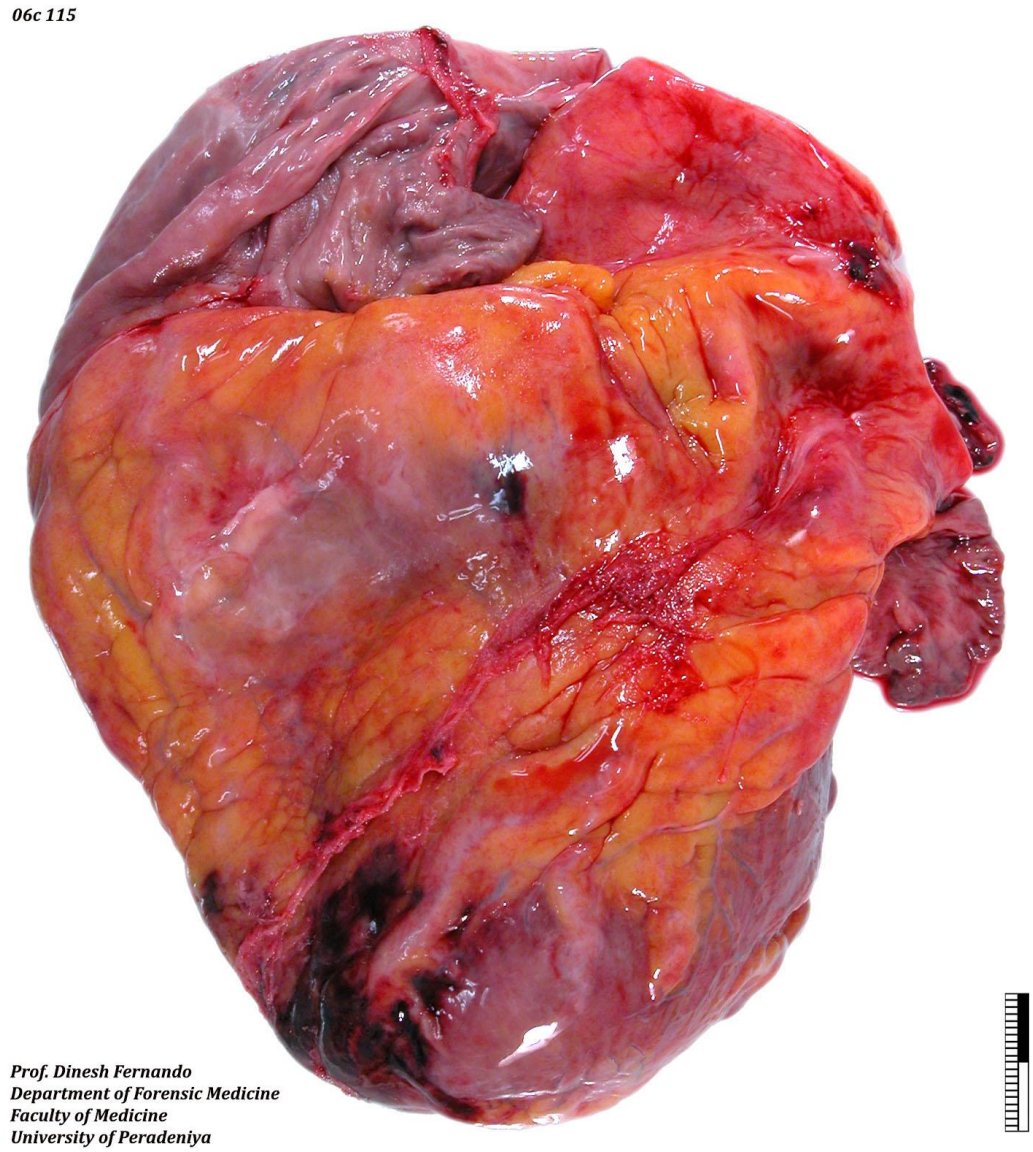

Figure 9: Posterior surface of the heart; note the erythematous haemorrhagic areas along the course of the tortuous posterior descending artery and apex of the right ventricle

The peritoneal cavity contained 1850 ml of fluid and clotted blood. The aorta had multiple ulcerated atheromatous plaques. An aneurysmal dilatation measuring approximately 8 cm in circumference was present in the lower abdominal aorta just above the common iliac branches and it contained an organised thrombus. At this site the wall of the aorta was thin and had an irregular small tear.

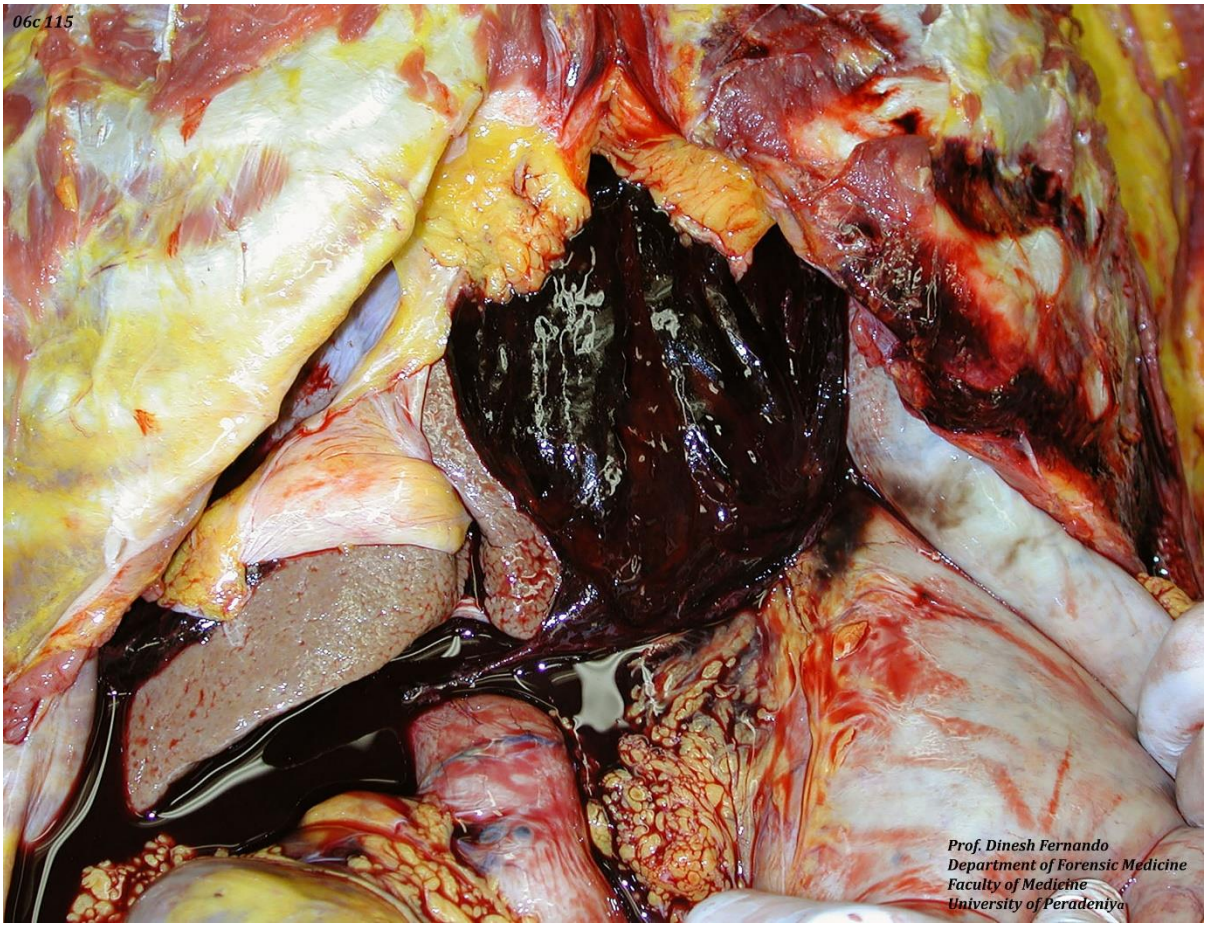

Figure 10: Haemoperitoneum

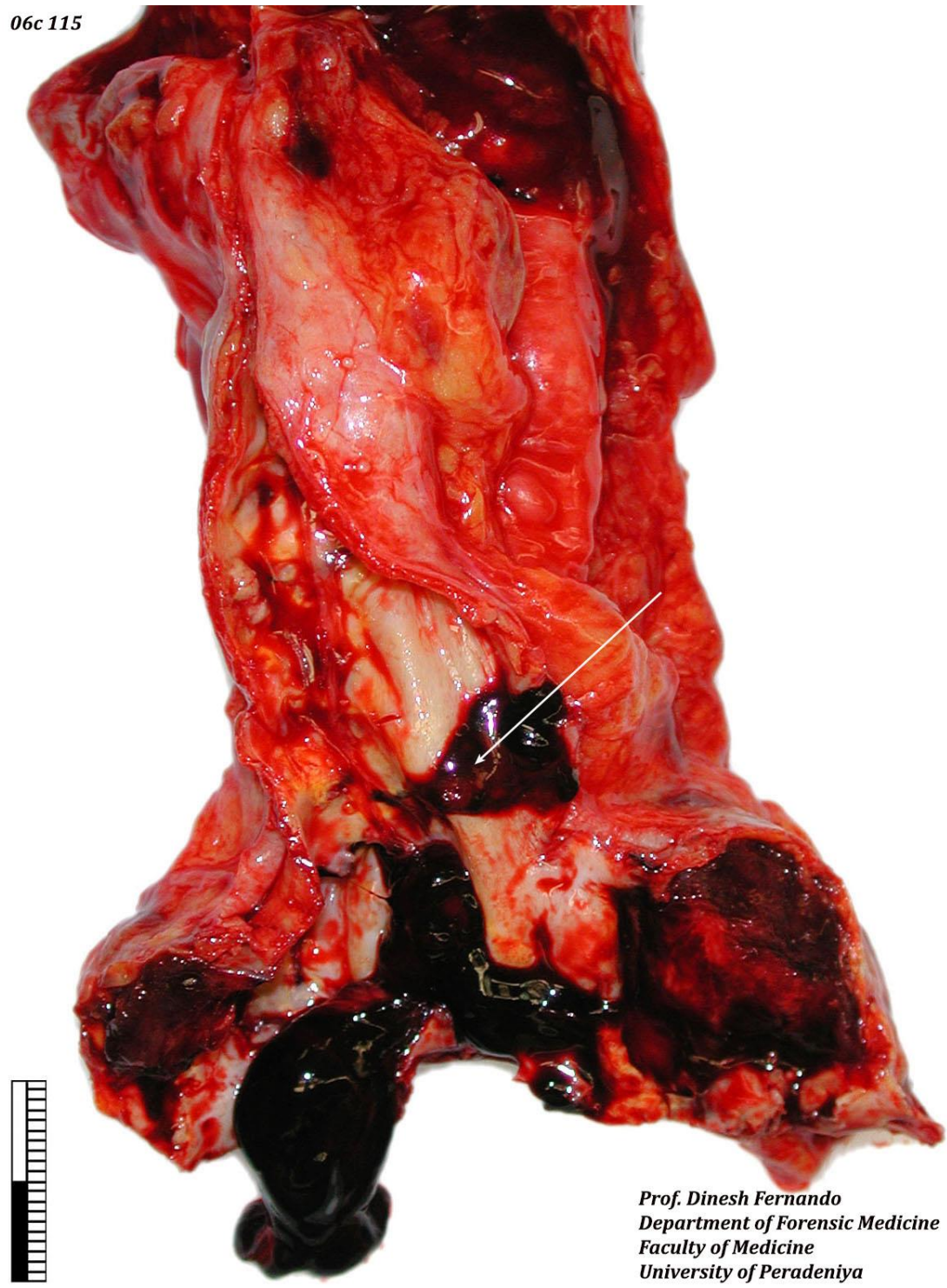

Figure 11: Posterior aspect of aorta showing the irregular tear

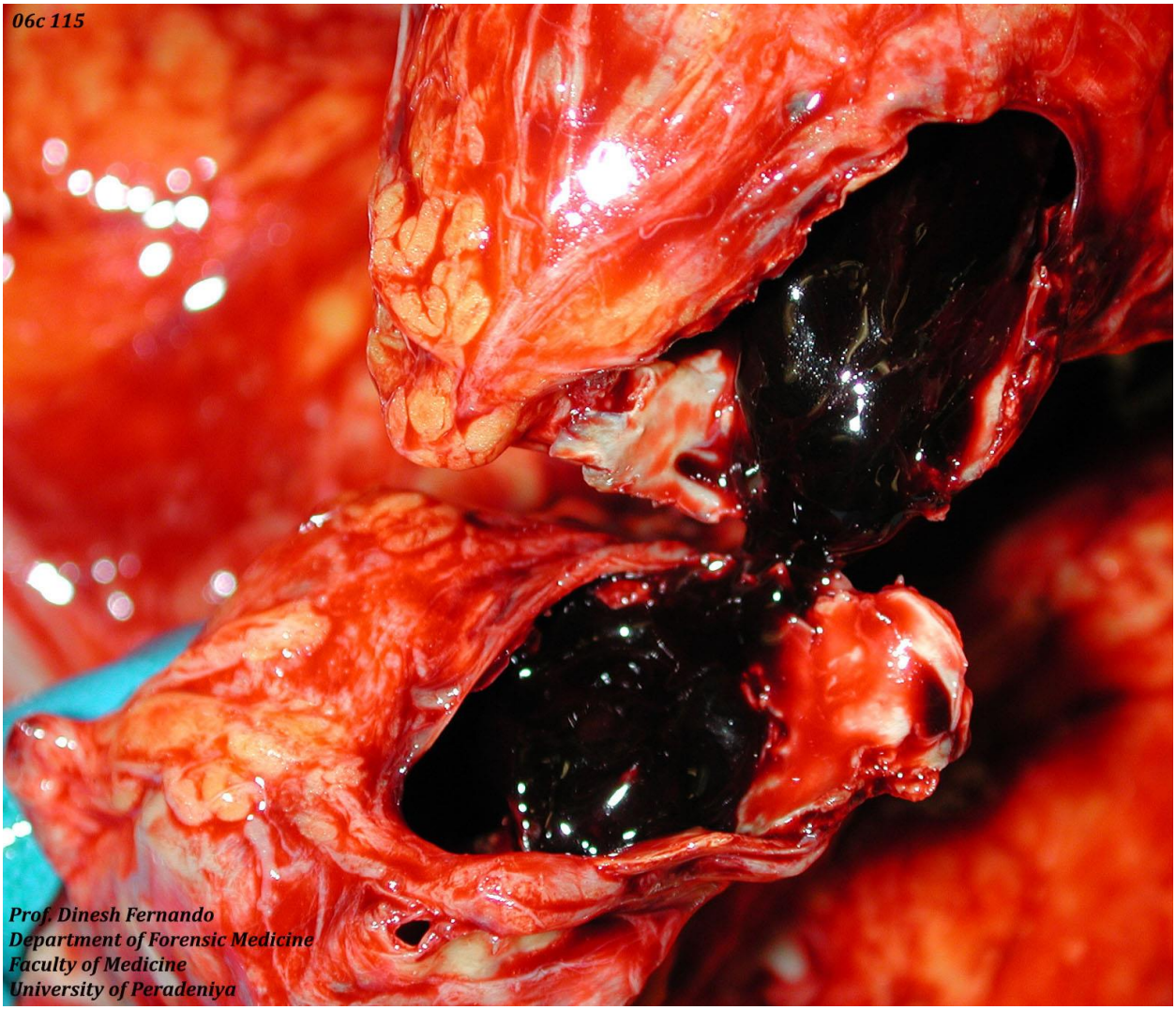

Figure 12: Closer view of the tear

Cause of death

Hypovolaemic shock caused by a ruptured abdominal aortic aneurysm.

Coronary artery atherosclerosis with recent stenting and perforation, leading to bilateral haemothorax and haemopericardium, were identified as other significant factors contributing to death.

Thoracic Aortic Aneurysm

The aneurysmal degeneration that occurs in the thoracic aorta is termed as thoracic aortic aneurysm. These can coexist with abdominal aortic aneurysms which are then named as thoracoabdominal aneurysms. Thoracic aortic aneurysms can be divided into three groups based on the location, namely, ascending aortic aneurysms, aortic arch aneurysms and descending thoracic aneurysms or thoracoabdominal aneurysms.

This aneurysmal dilatation occurs more commonly in the aging population. The collagen and elastin content in the aortic wall reduces with the advancing age, causing weakening of the wall. Elastic fibre fragmentation and cystic medial necrosis can be considered as pathological sequelae of aging.

The most common cause of thoracic aneurysms is degenerative arteriosclerotic disease. Other associated risk factors include family history of aortic aneurysms, Marfan syndrome, Ehlers-Danlos syndrome, infection, arteritis, trauma, and rarely, syphilis.

Deceleration injury due to high-speed motor vehicle crashes or falls from heights, can lead to traumatic dissection of the thoracic aorta. Usually this happens at a point of fixation, such as at the root of the aorta, aortic isthmus (site of attachment of the ligamentum arteriosum, which is just distal to the origin of the left subclavian artery) and diaphragmatic hiatus.

Most of the patients with thoracic aortic aneurysms are asymptomatic and are discovered incidentally on chest x ray and cardiological investigation. They may present with severe chest pain radiating to the upper back (due to rapid expansion), haemoptysis (due to aortobronchial fistula), or, rarely, with haematemesis due to aorto-oesophageal fistula. Ascending aortic aneurysms may erode into the sternum and ribs causing pain. Rupture of an aneurysm can cause hypotension, tachycardia and death. Aneurysms are treated medically by risk factor reduction and surgically by aortic replacement with a Dacron tube graft.

History

An 82-year-old female, with a history of a previously repaired abdominal aortic aneurysm, presented to the hospital unconscious. Subsequently, her blood pressure dropped and she went into cardiac arrest.

Internal Examination

The right pleural cavity contained 1,500 ml of fluid and clotted blood and the left pleural cavity contained 400 ml of fluid blood. The aorta had multiple ulcerated atheromatous plaques. An intimal tear measuring 7 cm in length was transversely placed in the descending thoracic aorta towards the right side. The circumference of the aorta at the site of rupture was 10 cm. The abdominal aorta had an intact repair measuring 7 cm by 9 cm at the site of an aneurysmal dilatation.

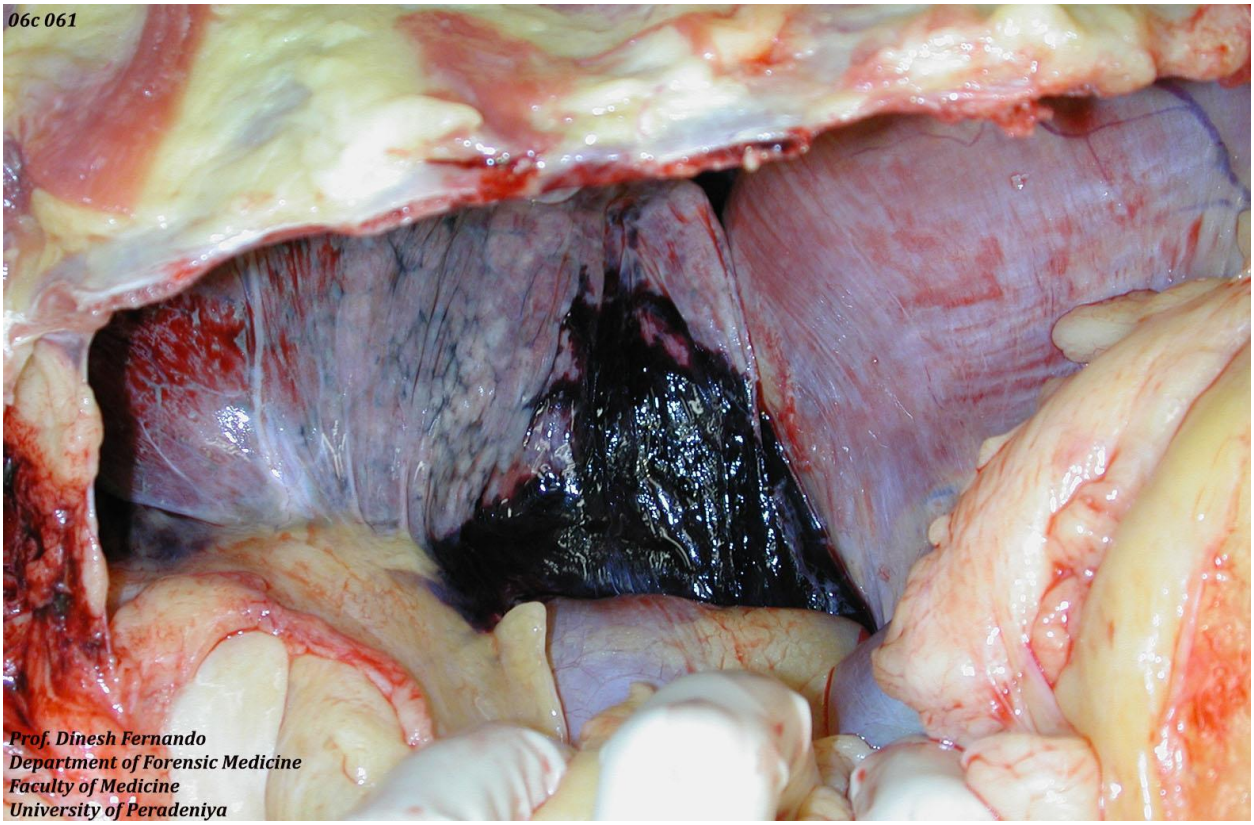

Figure 13: Haemothorax

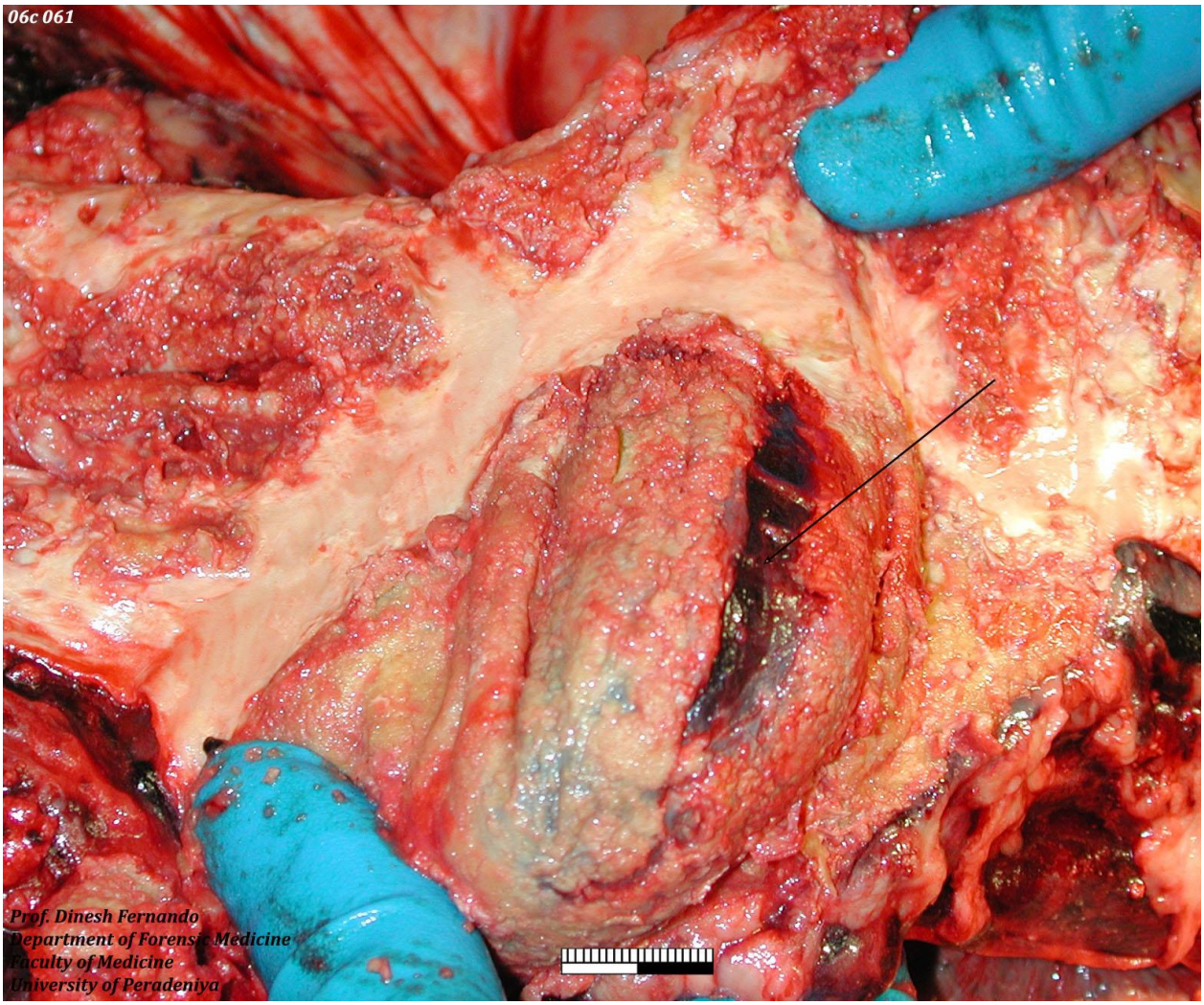

Figure 14: Aneurysm of the descending thoracic aorta: note the 7 cm long intimal tear

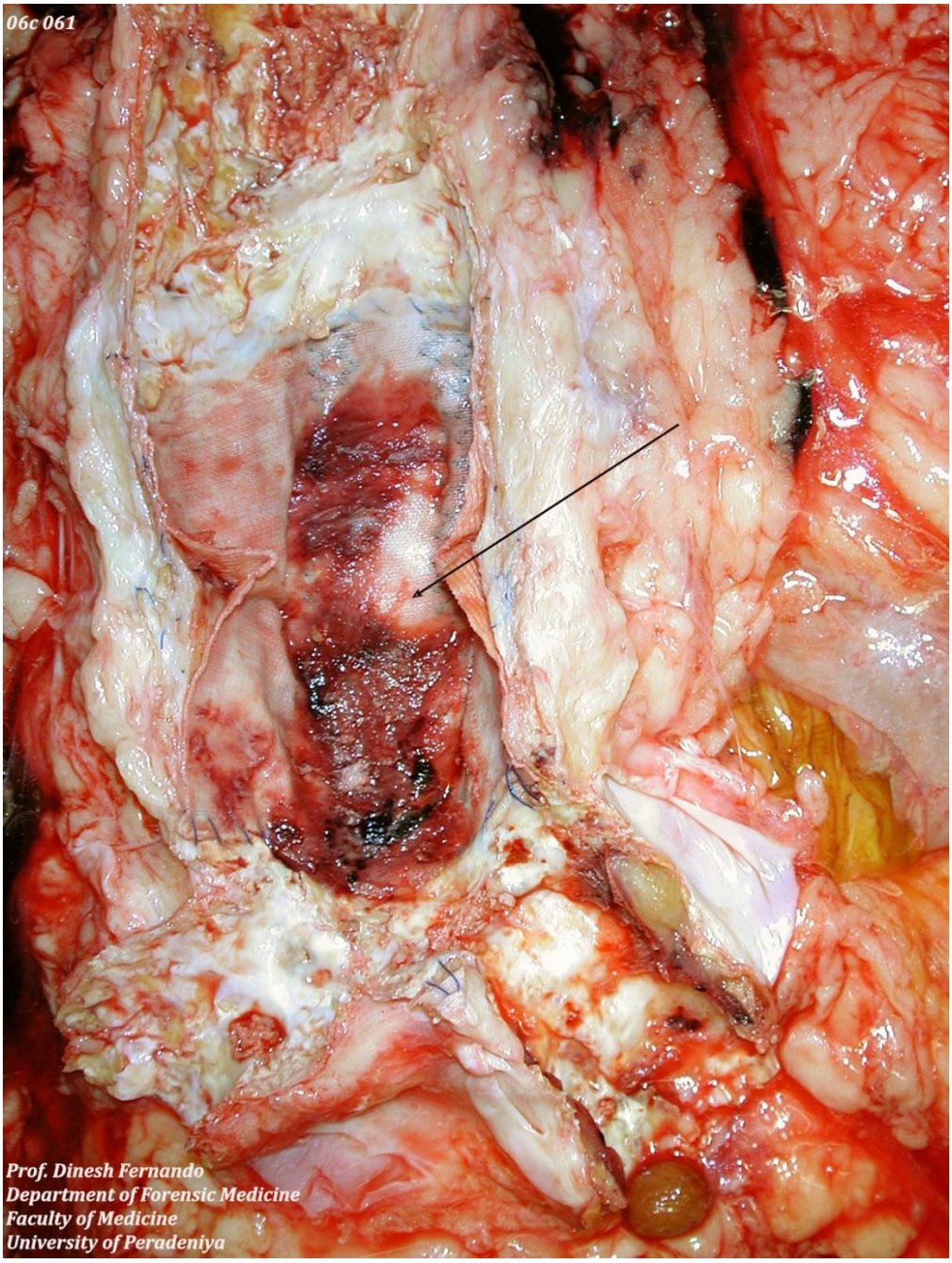

Figure 15: Repair of the abdominal aortic aneurysm which was intact

Cause of death

Haemothorax due to a ruptured descending thoracic aortic aneurysm.

The repair of the abdominal aortic aneurysm was intact. The rupture of the thoracic aorta has occurred at the site of an atherosclerotic aneurysm which has caused bilateral haemothorax and subsequent death.

History

A 70-year-old hypertensive male was found unconscious. Three days previously, he had complained of pain and difficulty in opening the mouth and burning type chest pain. He was declared dead, on admission to the hospital.

Internal Examination

The pericardial cavity contained 350 ml of clotted and fluid blood. An aneurysmal dilatation with a circumference of 10 cm at the site of ulcerated atheroma was present in the ascending aorta.

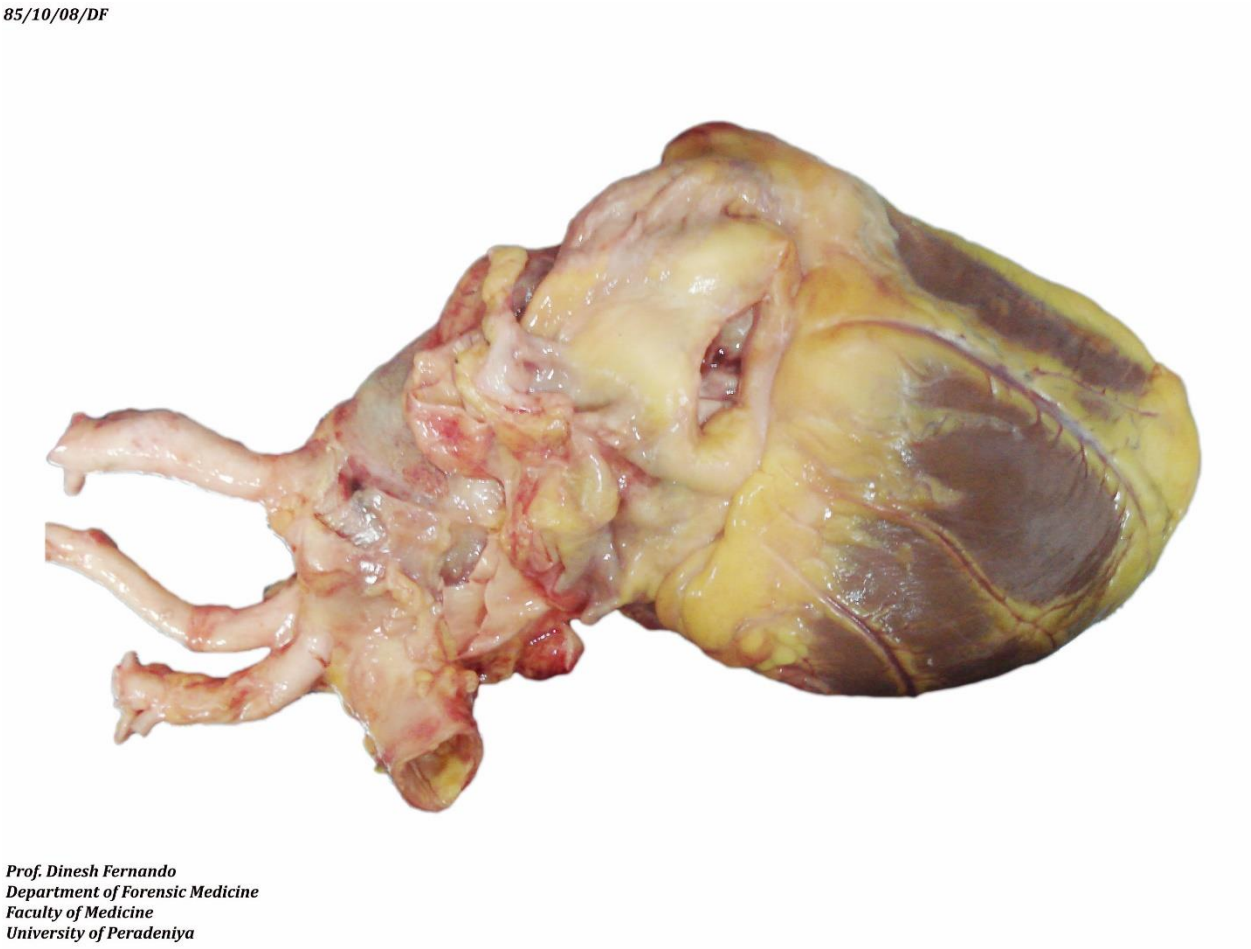

Figure 16 : A 4 cm long tear in the wall of the ascending aorta

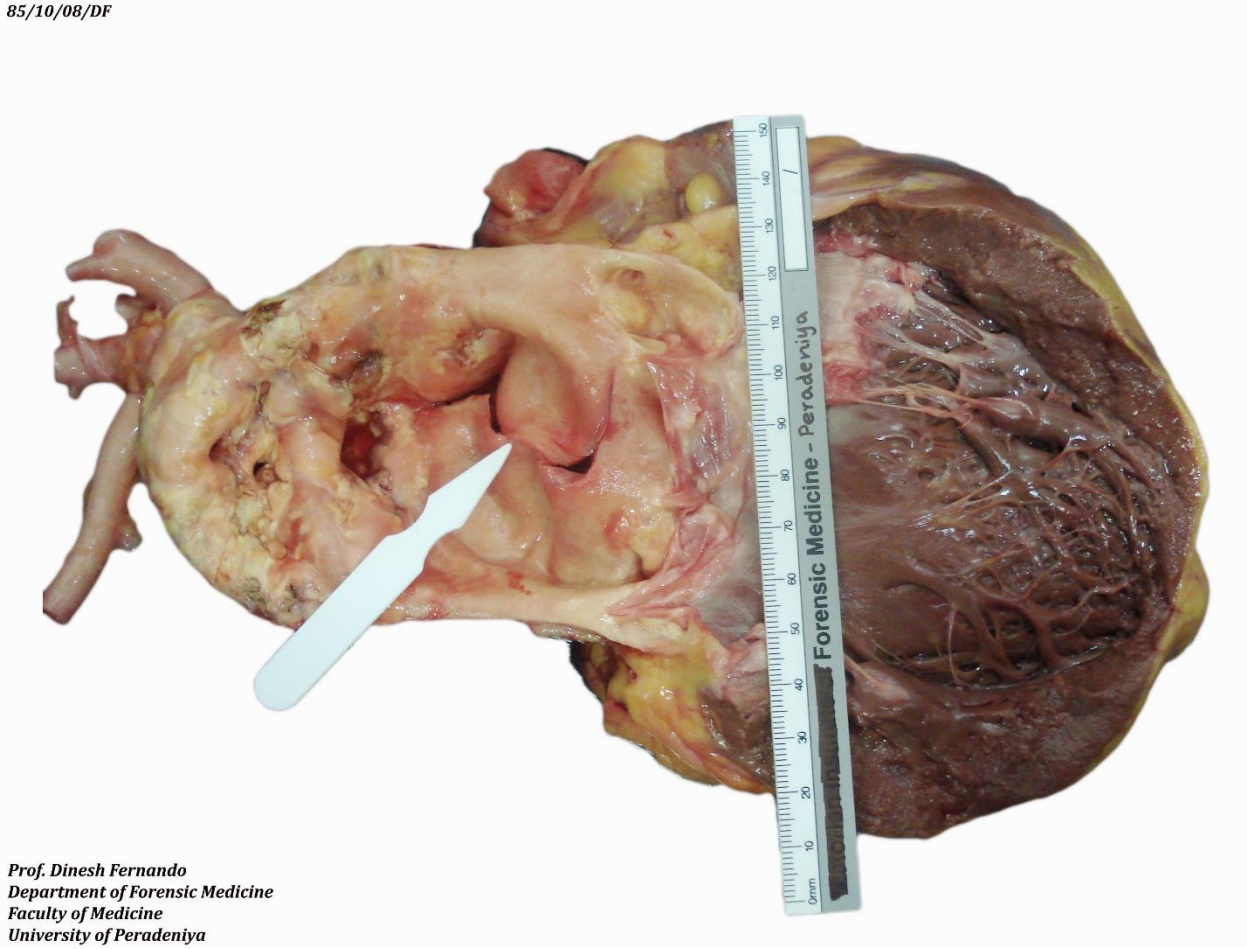

Figure 17: A 6x2 cm 'L' shaped tear of the intima at the site of the aneurysmal dilatation

Cause of death

Haemopericardium due to a ruptured ascending aortic aneurysm.

Aortic dissection

Aortic dissection usually begins with a tear in the intima. Blood penetrates, and then cleaves, the medial layer causing a dissection. Aortic dissection usually occurs due to a weakness in the media. A spontaneous rupture of vasa vasorum in the aortic media may cause an intramural haematoma (IMH), which propagates along the medial layer of the aorta, resulting in weakening and dissection. IMH can also be caused by microscopic intimal tears, a deep penetrating atherosclerotic ulcer and traumatic medial injury. Patients with autoimmune rheumatic disorders, Marfan and Ehlers- Danlos syndrome are more prone to dissection.

Aortic dissection may be classified anatomically according to the region of aorta affected (Stanford type A and B, De Bakey type I, II and III). The clinical features of aortic dissection include sudden onset of, severe, tearing type, central chest pain that radiates to the back.

History

A 60-year-old male, a diagnosed patient with hypertension, was found dead in his sleep.

Internal Examination

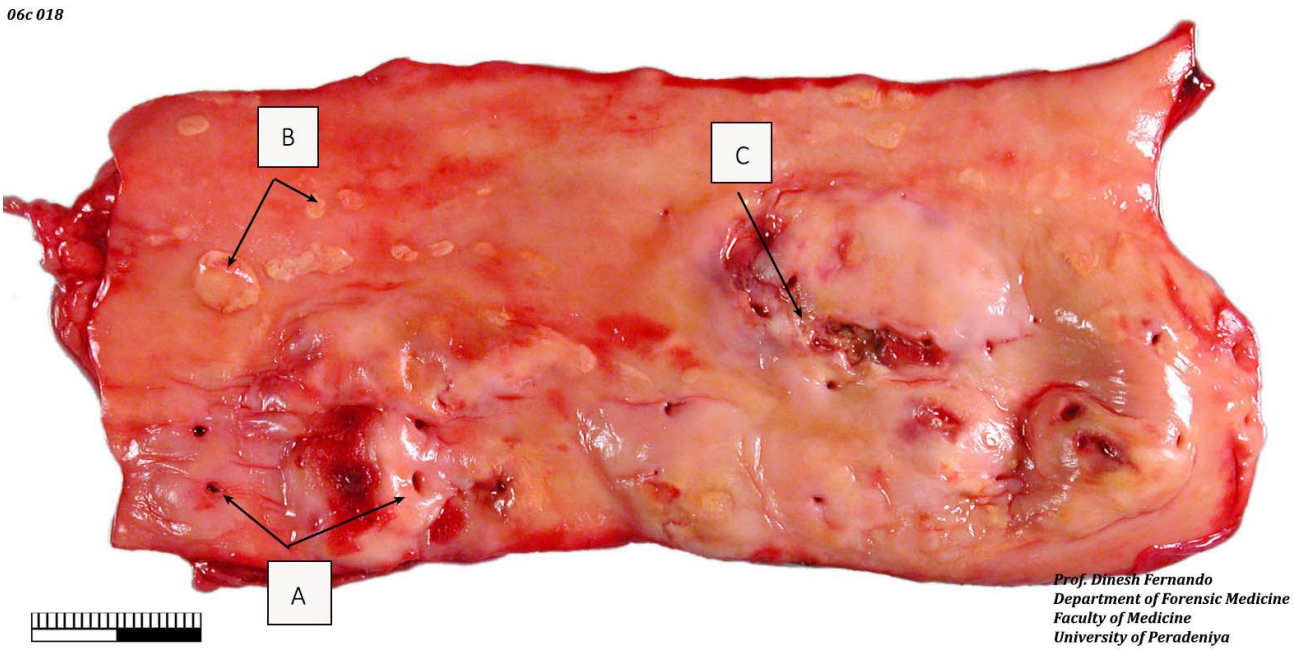

Figure 18: Descending thoracic aorta; (A) ostia of the intercostal vessels, (B) raised yellowish atheromatous plaques, (C) 1.5 cm long intimal tear associated with an atheromatous plaque

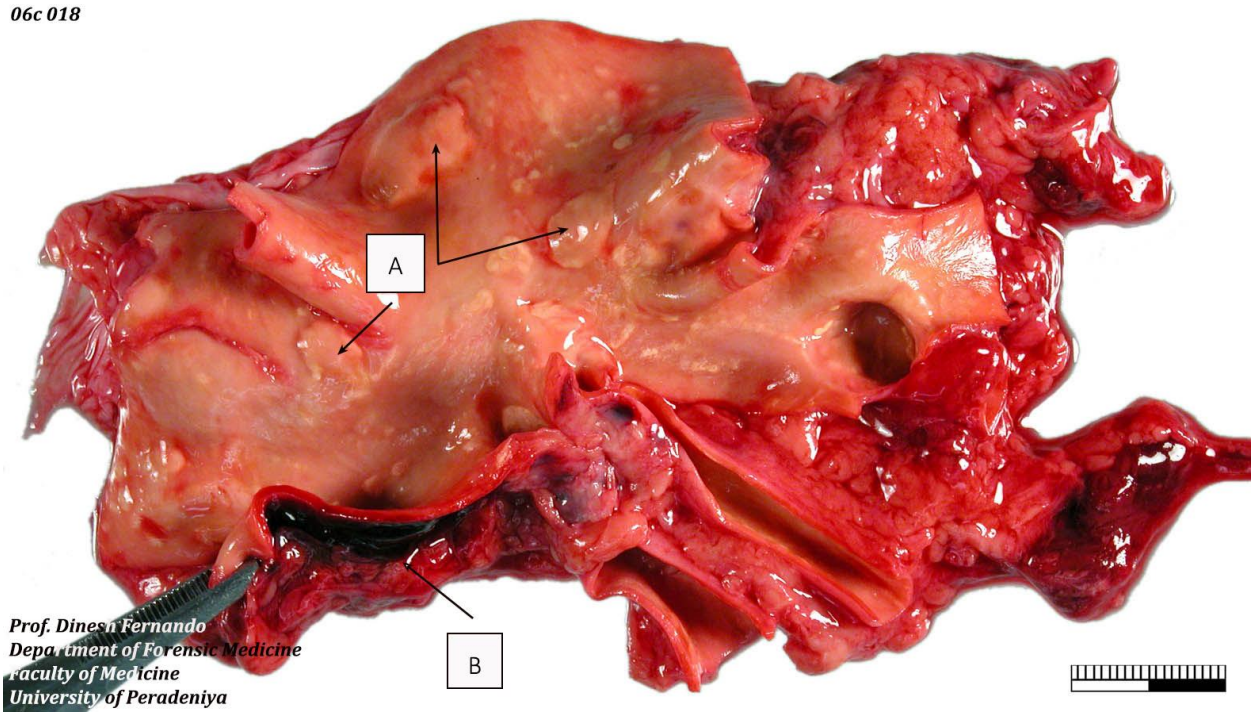

Figure 19: Arch of the aorta; (A) atheroma, (B) dissection which has extended in a retrograde manner up to the aortic arch (no re-entry into lumen)

Aortitis

Aortitis is defined as inflammation of the wall of the aorta, with or without, disruption of elastic fibres, aortic wall necrosis or fibrosis. The causes for aortitis include giant cell arteritis, syphilis and Takayasu arteritis.

Giant cell arteritis is a granulomatous inflammation affecting large to small arteries, mainly those supplying the head – commonly the temporal arteries – and is extremely rare under the age of 50 years of age. Patients present with severe headache, tenderness of the scalp or temple, claudication of the jaw when eating, and tenderness and swelling of, one or more, temporal or occipital arteries. The definitive diagnostic test is a temporal artery biopsy from the affected side. The histological features include cellular infiltrates of CD+ T lymphocytes, macrophages and giant cells in the vessel wall, granulomatous inflammation of the intima and media, breaking up of the internal elastic lamina and giant cells, lymphocytes and plasma cells in the internal elastic lamina. Giant cell arteritis may also affect the vertebral and ophthalmic arteries, as well as the aorta (giant cell aortitis).

Takayasu arteritis, known as pulseless disease, is a granulomatous inflammation of the aorta and its major branches. Transmural scarring and thickening of aorta occur. It is characterized by ocular disturbances and marked weakening of pulses in the upper extremities. The aortic lesions share many of the features of giant cell arteritis and the distinction between the two entities is made largely on the basis of the patient's age. Giant cell aortitis is considered as the diagnosis if the patient is older than 50 years of age and Takayasu aortitis is considered if the age is less than 50 years.

History

The deceased was a 78-year-old female who lived on her own. Since she was not seen for some time, the police were notified. When the police gained access, she was found face down near the front door. She had a past history of hypertension, chronic lower back pain, polymyalgia and temporal arteritis on the right side. A diagnosis of active giant cell arteritis had been made following right temporal artery biopsy. A duplex scan of the aorta and lower limbs had not shown any abnormality

External Examination

Hypostasis was apparent on the anterior aspect of the body and more prominent on the right side of the body. Marbling of skin was seen in the arm and forearms. Post mortem peeling of the skin of the limbs was present.

There were areas of pallor on the pressure points on the right side of the body, namely, the outer aspect of the right knee, anterolateral aspect of the right thigh, right side of the chest, outer aspect of the right arm, nose and on the bony prominences above and below the right eye.

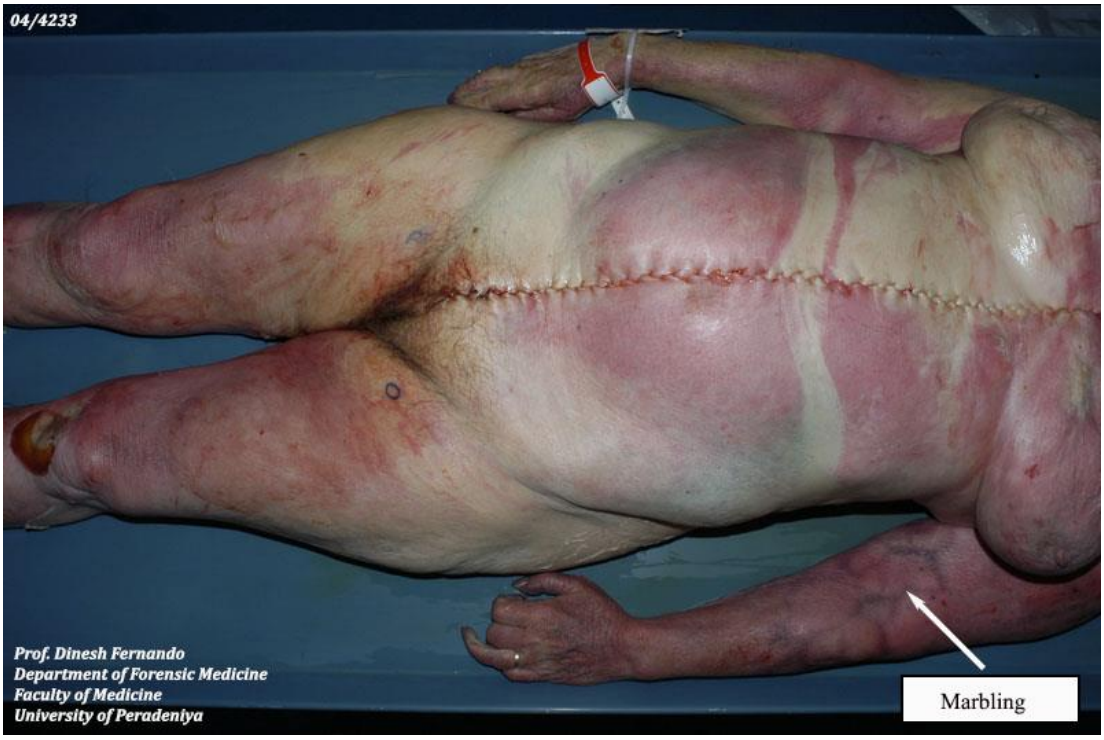

Figure 20: Distribution of hypostasis with areas of pallor and marbling of skin

Figure 21: Hypostasis, with pale areas due to pressure, on the face.

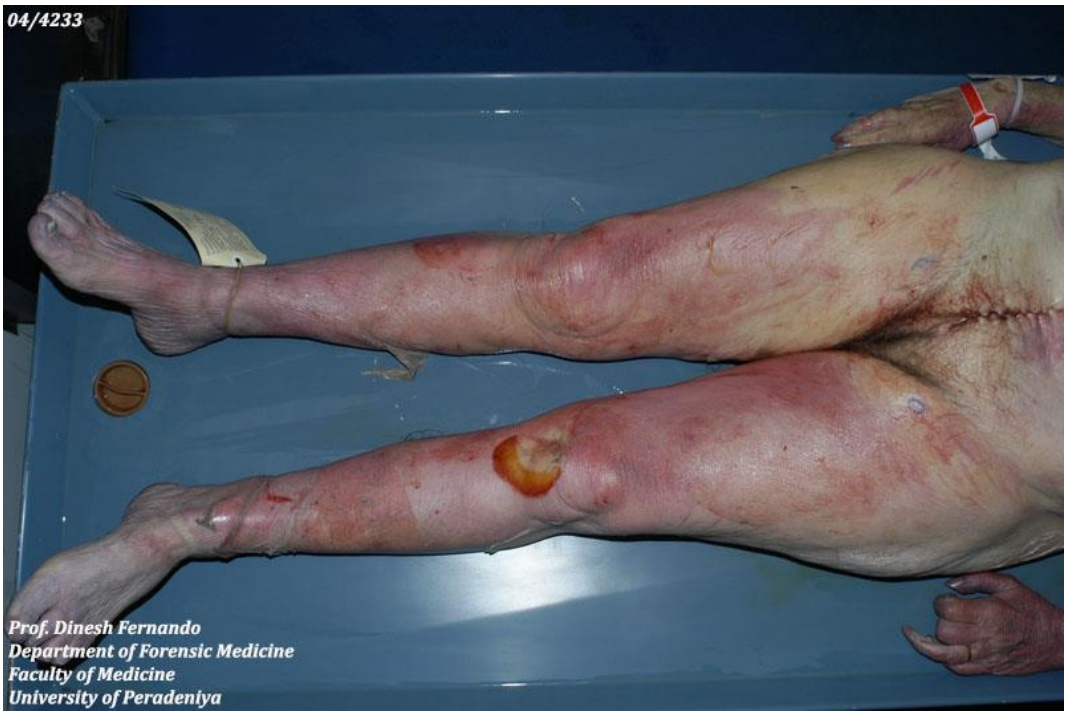

Figure 22: Peeling of the skin and hypostasis on anterior aspect of lower limbs

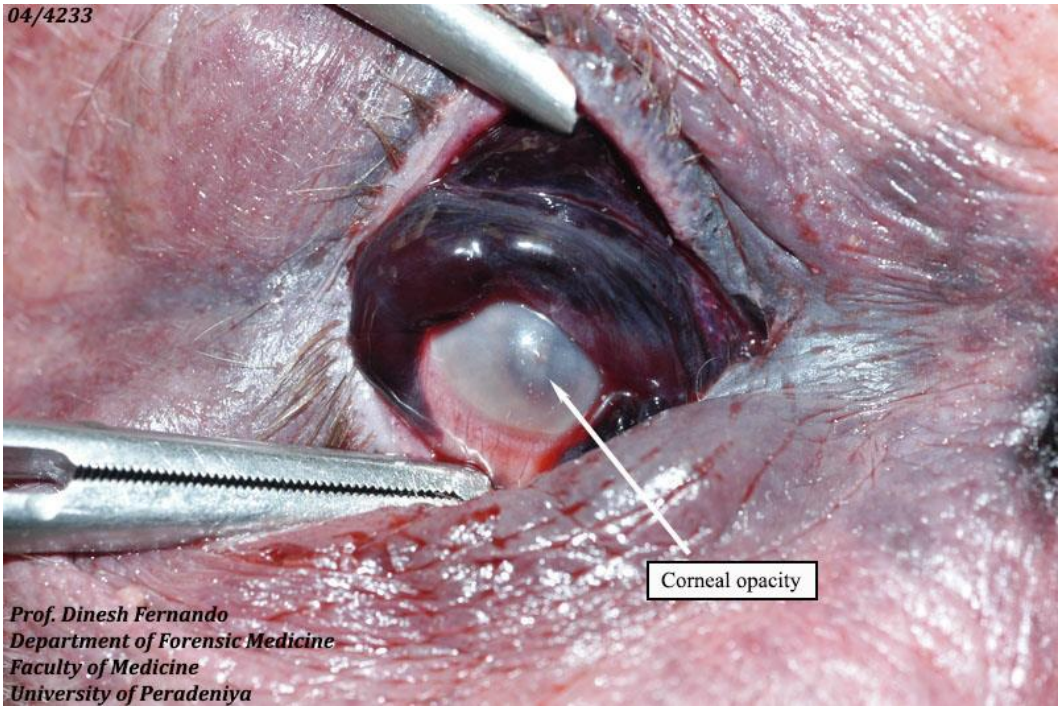

Figure 23: Intense congestion in the conjunctiva and sclera of the eye due to the prone position: Note the post mortem opacification of the cornea.

Figure 24: Contusion on the inner aspect of the lower lip.

Internal Examination

Figure 25: A contusion measuring 3 cm x 2 cm on the inner aspect of the scalp on the left side of the forehead.

Cardiovascular system: The pericardial sac was intact. The pericardial cavity contained 350 ml of clotted and fluid blood. The thoracic and abdominal aorta had multiple ulcerated atheromatous plaques. There was a transverse intimal tear of 2 cm in the ascending part of the aorta, causing a dissection that had opened into the pericardial cavity causing a haemopericardium.

In order to demonstrate haemopericardium, Figures 26 to 28 are from a different case.

Haemopericardium

Haemopericardium is an accumulation of blood in the pericardial sac. Normally, the pericardial sac contains 30 to 50 ml of clear, serous fluid. When a large volume collects in this space, the ventricular filling of the heart is compromised, causing cardiac tamponade. The common causes of haemopericardium are rupture of the myocardium following a myocardial infarction, ruptured aortic dissection, traumatic perforation, and rarely, infective endocarditis.

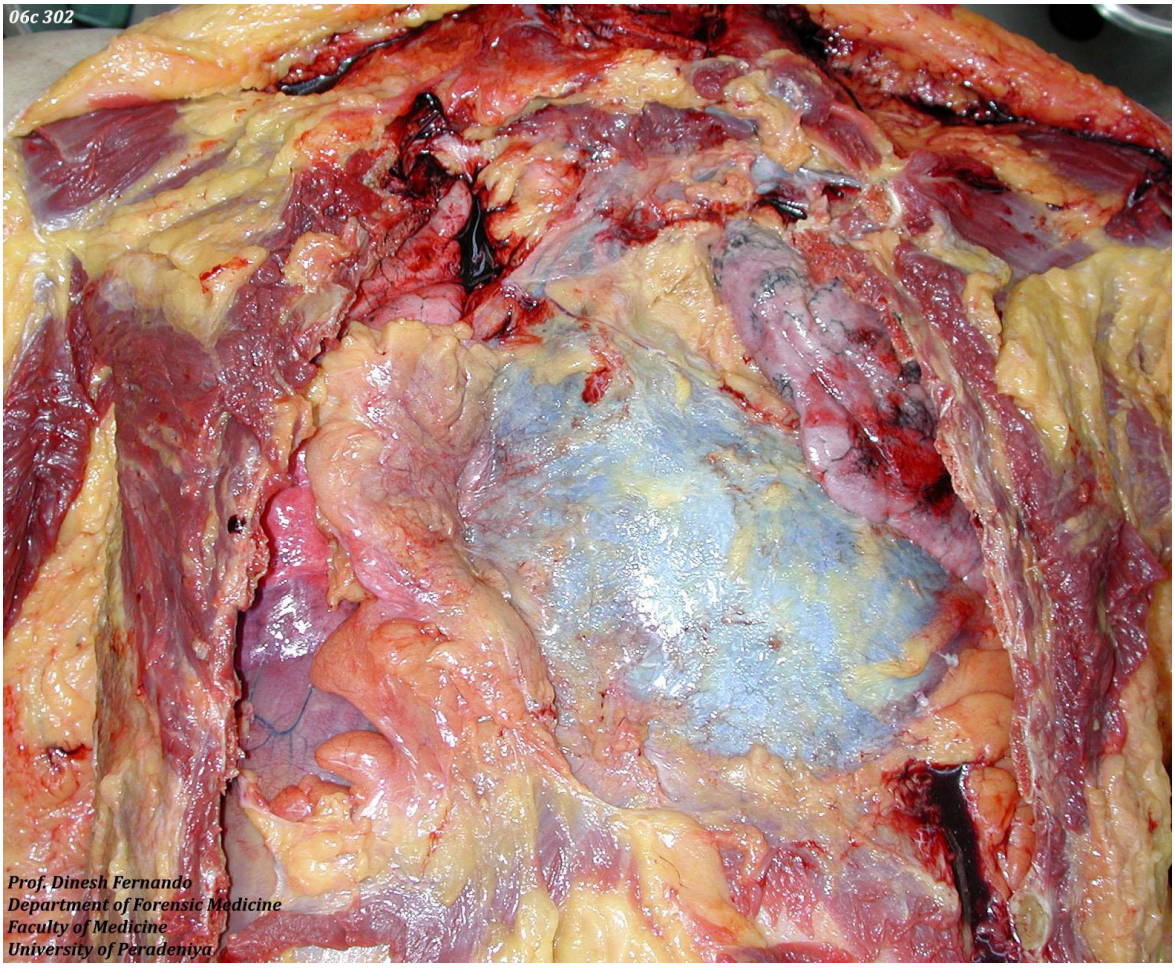

Figure 26: Opened chest cavity: note the bluish colour of the pericardium

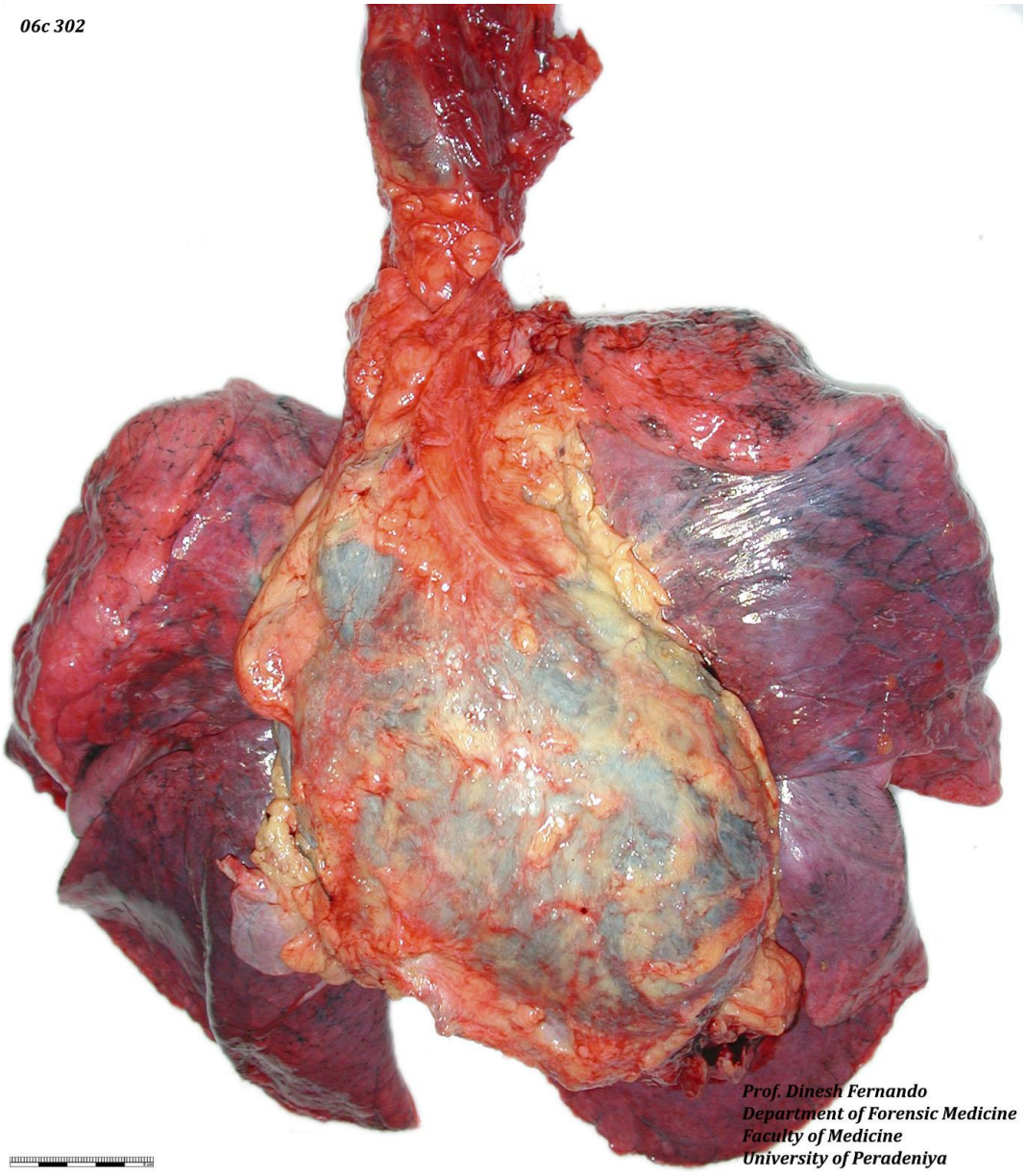

Figure 27: Thoracic pluck with intact pericardium: note bluish colour due to the blood in pericardial space

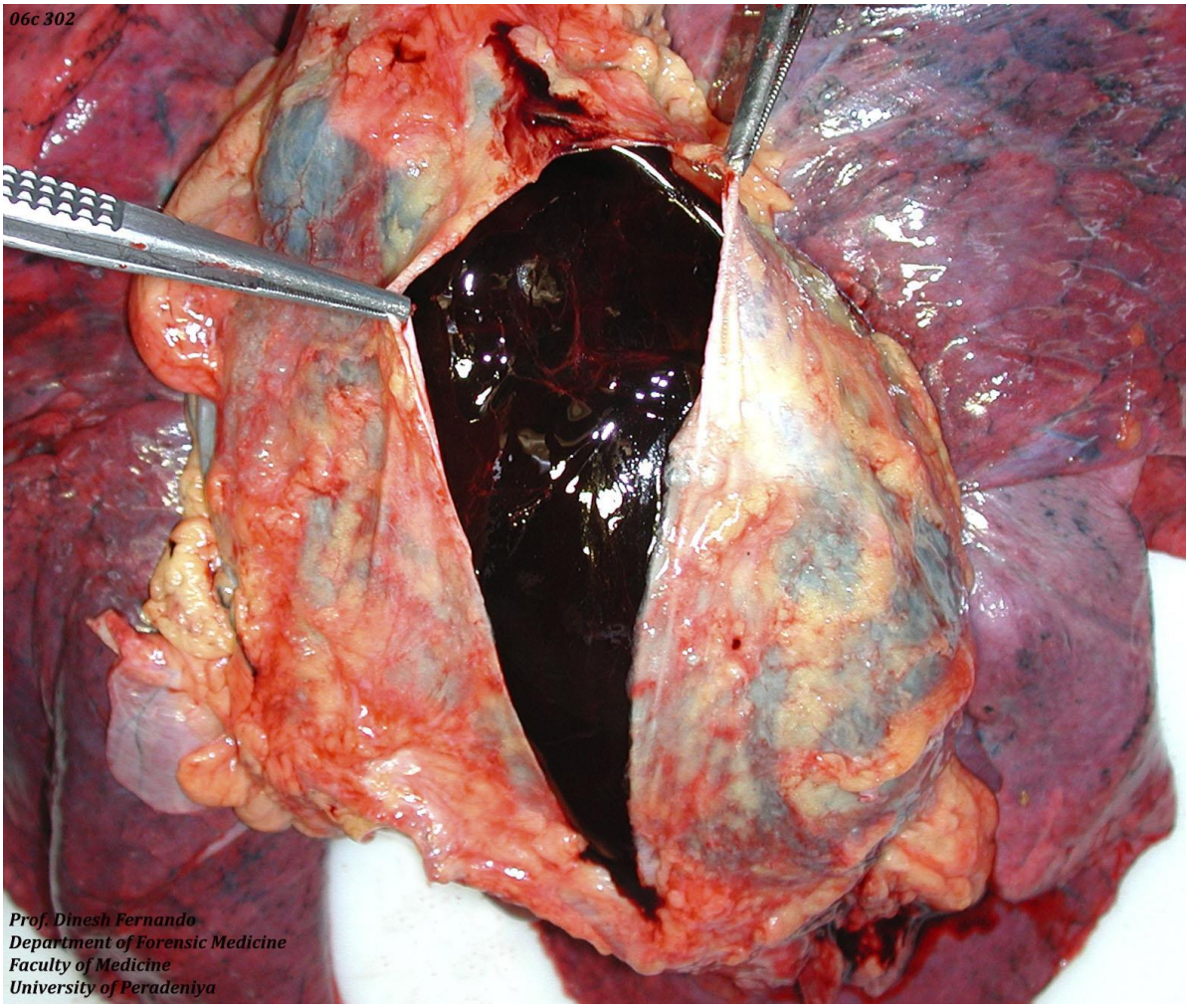

Figure 28: Open pericardium showing clotted and liquid blood in the pericardial cavity

Microscopic Examination

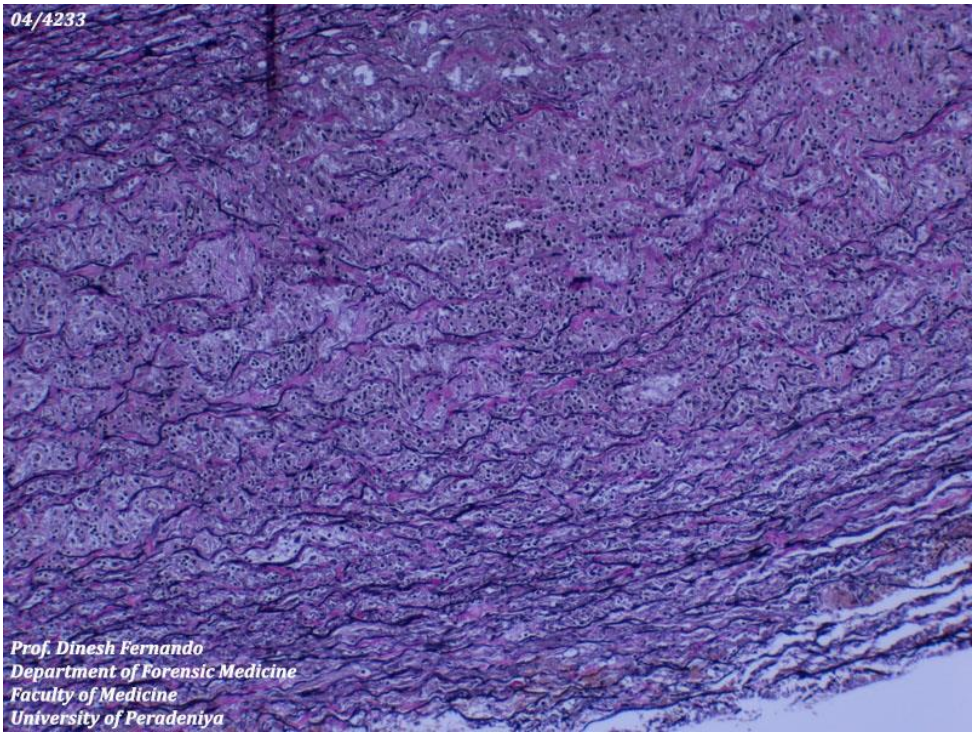

Figure 29: Disruption and fragmentation of elastic fibres (H&Ex100)

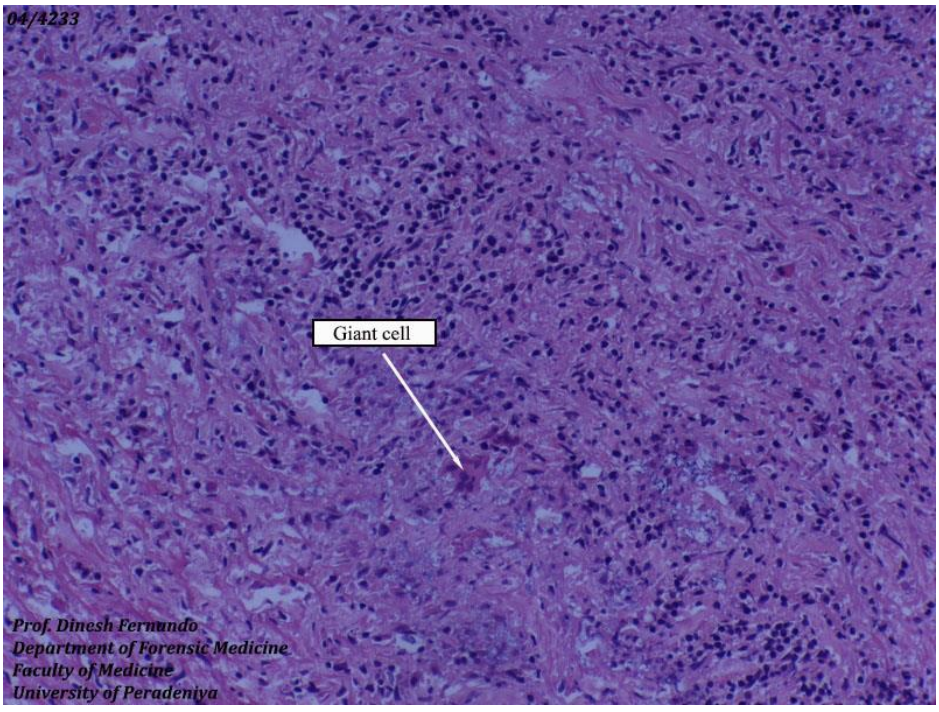

Figure 30: Media of the aorta showing infiltration with mononuclear and Langhans type multinucleated giant cells (H&Ex200)

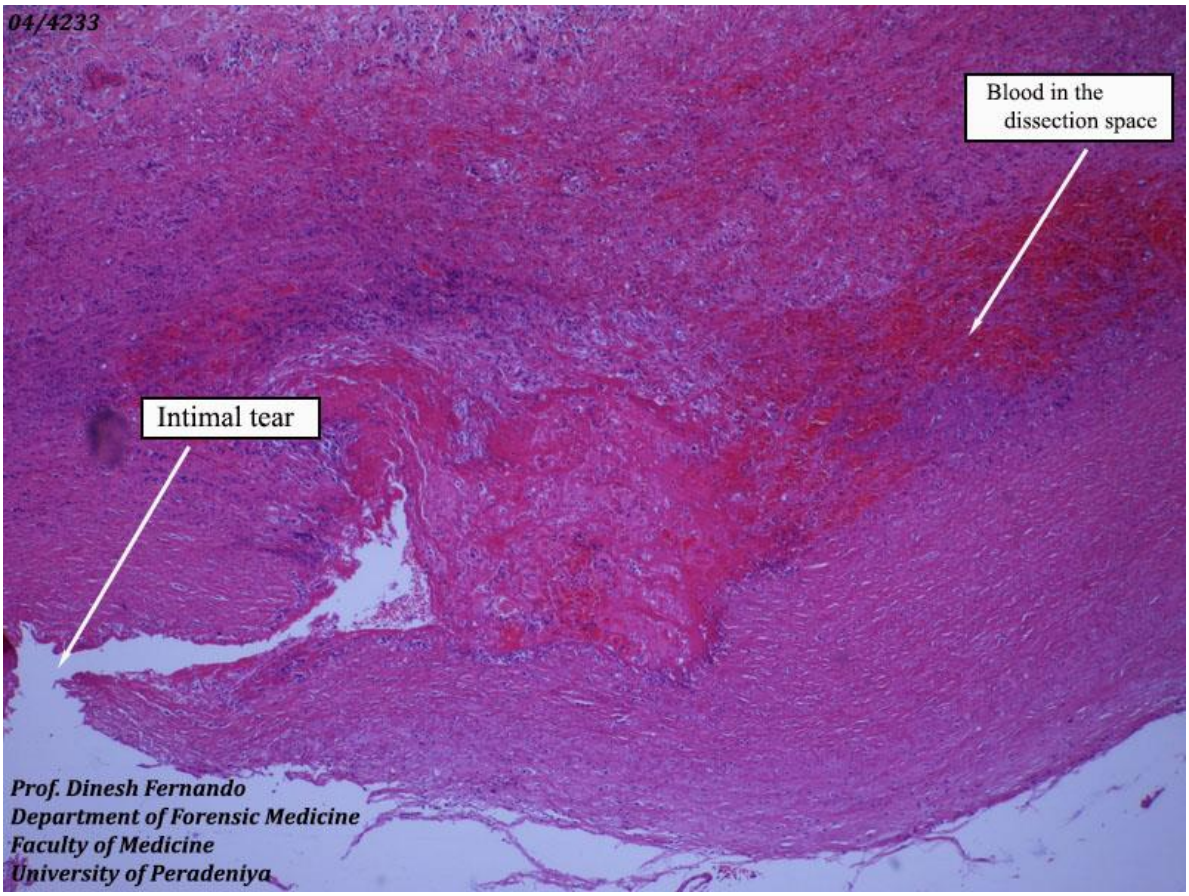

Figure 31: Intimal tear with aortic dissection (H&Ex40)

Cause of death

Haemopericardium due to ruptured dissection of the ascending aorta caused by giant cell aortitis.

Aorto-enteric fistula

Aorto-enteric fistula, a rare cause of massive upper gastrointestinal bleeding, occurs when the wall of the aorta erodes into the adjacent gastrointestinal system. Primary aorto-enteric fistulae occur in the native aorta, whereas, secondary aorto-enteric fistulae occur following placement of synthetic aortic graft material. Primary fistulae occur spontaneously and are less common than the secondary ones. The most common site of a fistula is the duodenum, particularly the third and fourth segments, which are anatomically closest to the aorta.

Aorto-oesophageal fistulae constitute less than 10% of all aorto-enteric communications. Common causes of aorto-oesophageal fistula are rupture of a thoracic aortic aneurysm, aortic dissection, foreign bodies, oesophageal stenting for oesophageal malignancy and thoracic surgery. Foreign bodies in the gastrointestinal tract can range from a coin to sharp objects including bone pieces, pins, needles, and tooth picks. Non sharp objects such as coins are more common in children, whereas, bone pieces are common in adults. Aorto-oesophageal fistulae caused by foreign bodies have a very low long term survival rate.

Most aorto-oesophageal fistulae are diagnosed post-mortem after a massive exsanguinating haemorrhage. The classic symptoms of aorto-oesophageal fistulae consist of dysphagia or mid-thoracic pain, followed by a sentinel or herald bleed, and then an exsanguinating haemorrhage after a variable asymptomatic period (Chiari's triad). The interval between the sentinel haemorrhage and exsanguination is more than 24 hours. Presentation with the classic Chiari's triad is usually diagnostic.

Aorto-oesophageal fistulae should be managed surgically, following circulatory support by volume replacement and transfusion of blood.

History

A 36-year-old male presented complaining of epigastric pain, discomfort in his retrosternal area and feeling of something sticking in his throat. He had ingested fish the previous day while consuming alcohol. He was discharged after four days of inward management with a diagnosis of gastritis. The same evening, he was re-admitted with an episode of haematemesis. About 12 hours later he developed massive hematemesis, for which he underwent emergency anterior gastrectomy. Three litres of blood was found in the stomach during surgery but no bleeders were identified. He succumbed, 30 min after admission to the intensive care unit.

Internal Examination

Thoracic dissection at autopsy revealed ulceration 2 cm below the aortic arch with an impacted fish bone creating a fistula tract with the oesophagus. The fish bone was triangular in shape measuring 3.5x2.5 cm with sharp pointed ends. The junction of the upper and middle third of the oesophagus was ulcerated and showed inflammation around the fish bone. There was clotted blood in the stomach. There were sub endocardial flame shaped haemorrhages.

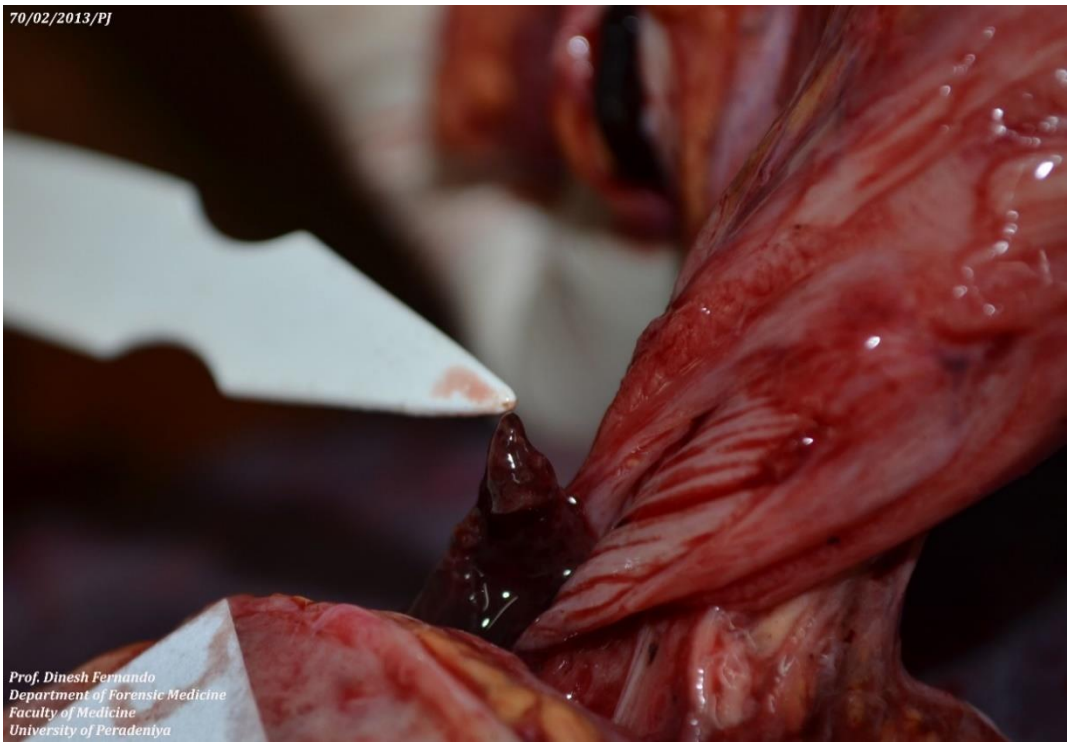

Figure 32: Pointed fish bone found within the lumen of the oesophagus

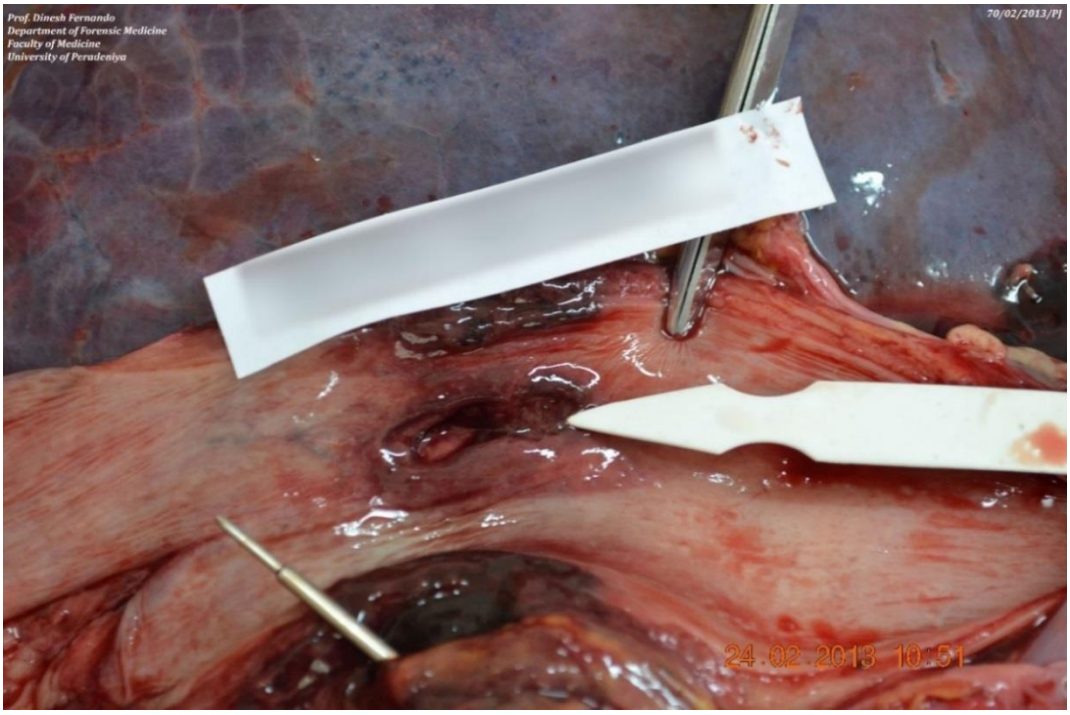

Figure 33: A 2x0.5 cm ulceration with bleeding and inflammation on the oesophageal mucosa

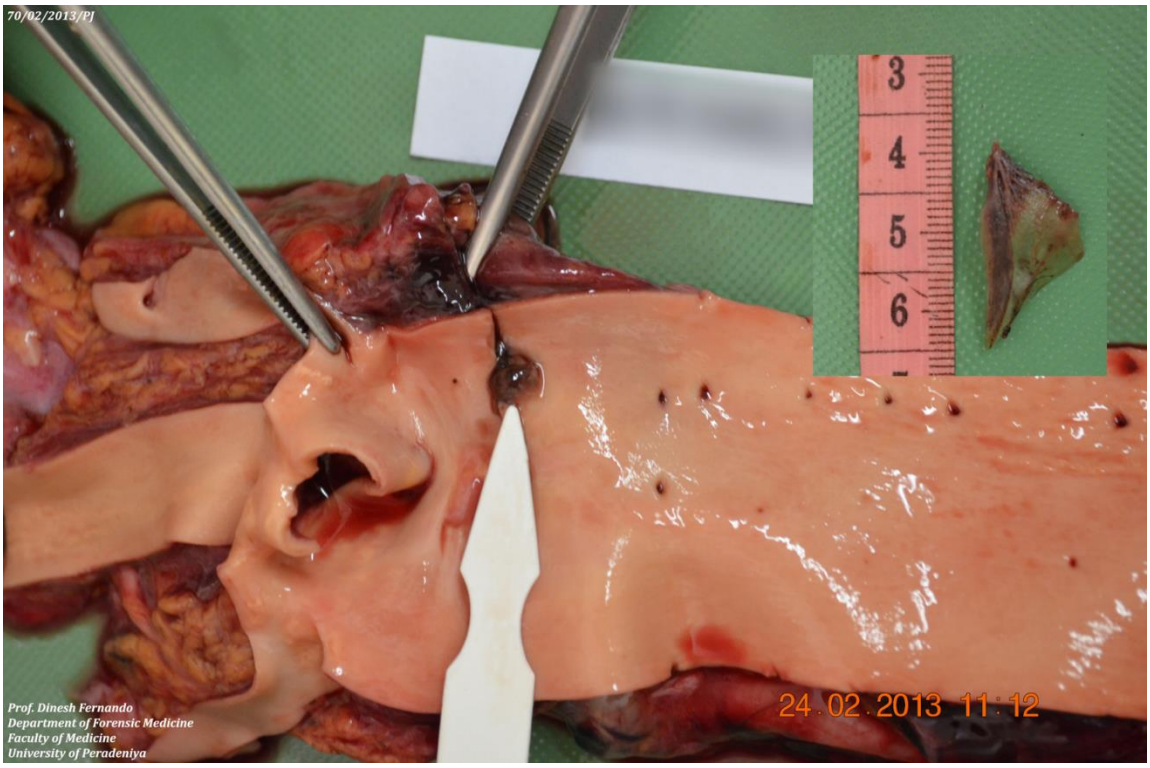

Figure 34: Fish bone protruding into the aortic lumen. Inset: fish bone that caused the fistula

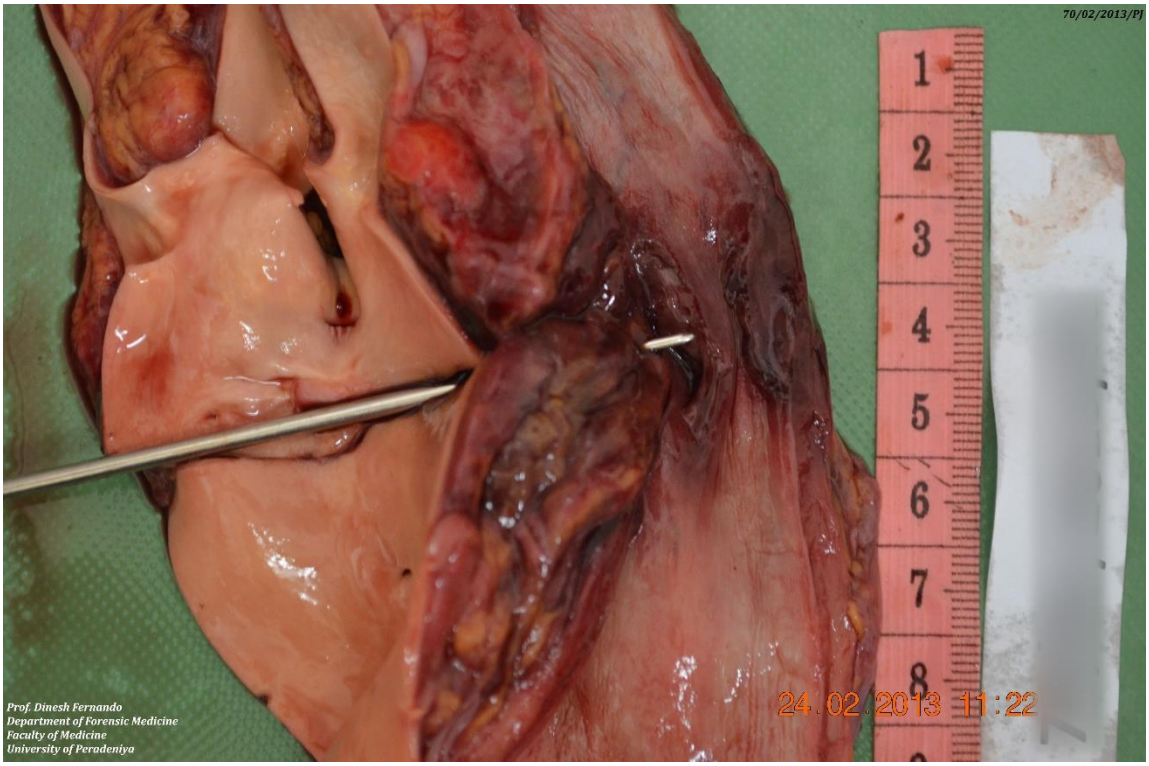

Figure 35: Fistula tract extending from junction of upper and middle third of oesophagus to thoracic aorta

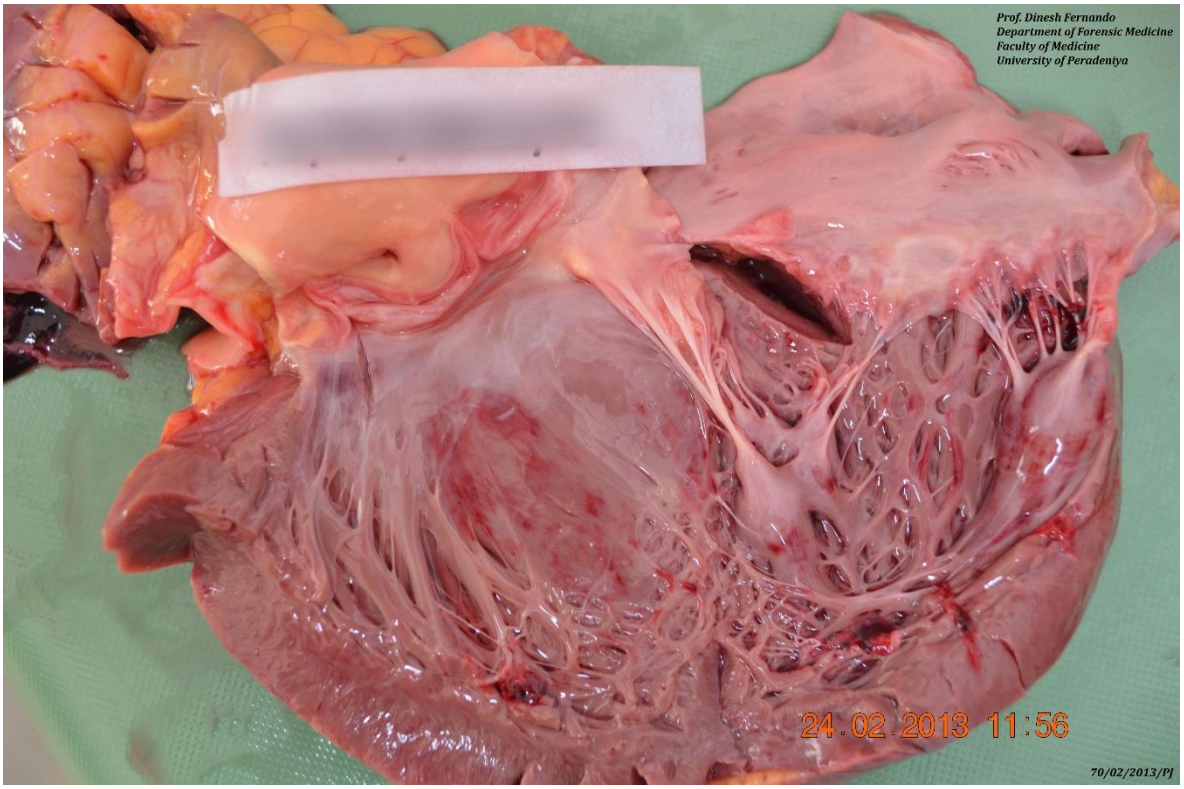

Figure 36: Sub endocardial flame shaped haemorrhages

Sub endocardial haemorrhages occur after cardiac injuries, head trauma, infections, intoxications, abdominal trauma, hypovolaemic shock and resuscitation.

Cause of death

Exsanguination due to haemorrhage from an aorto-oesophageal fistula caused by impaction of a fish bone in the oesophagus.

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