COLOR ATLAS OF

FORENSIC PATHOLOGY

CARDIOVASCULAR DISEASE

CORONARY ARTERY DISEASE

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

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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.

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

Prof. Dinesh Fernando
Dr. Sulochana Wijetunge

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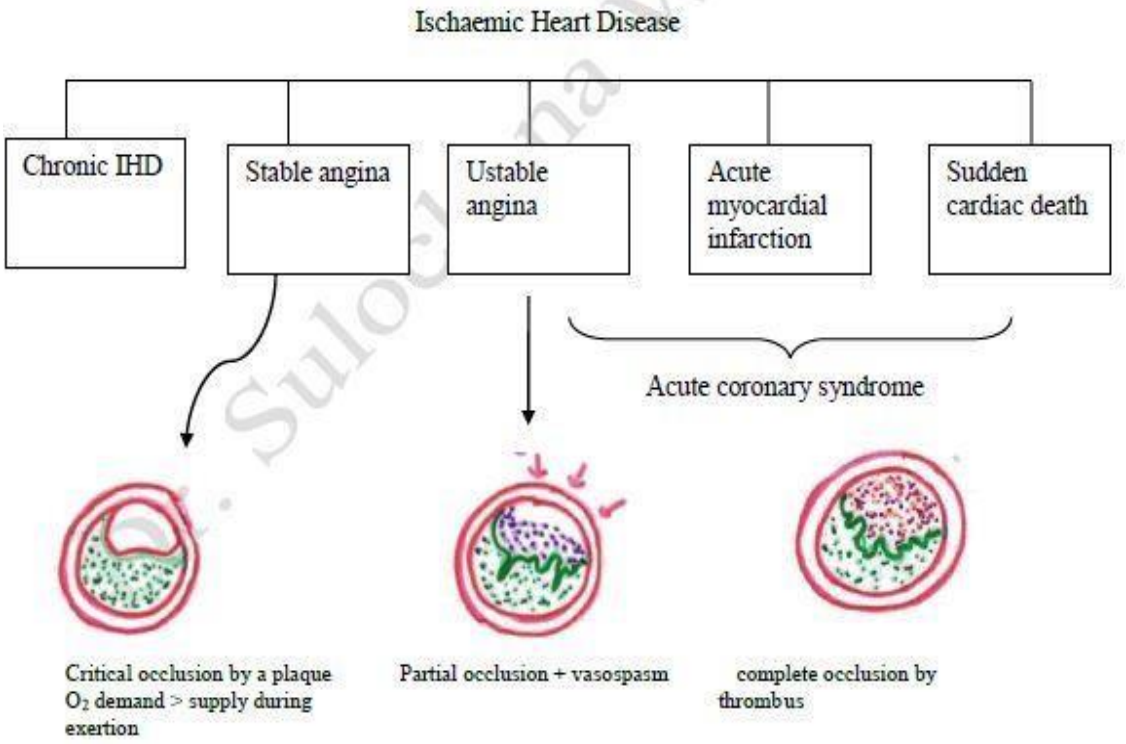
CORONARY ARTERY DISEASE

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 oftime the patient may develop Chronic IHD and congestive cardiac failure.

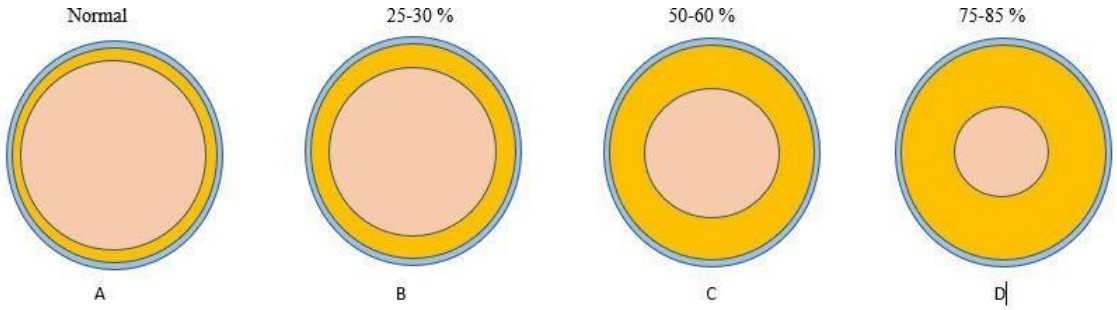


Figure 1: Schematic representation of occlusion by concentric atheroma

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).

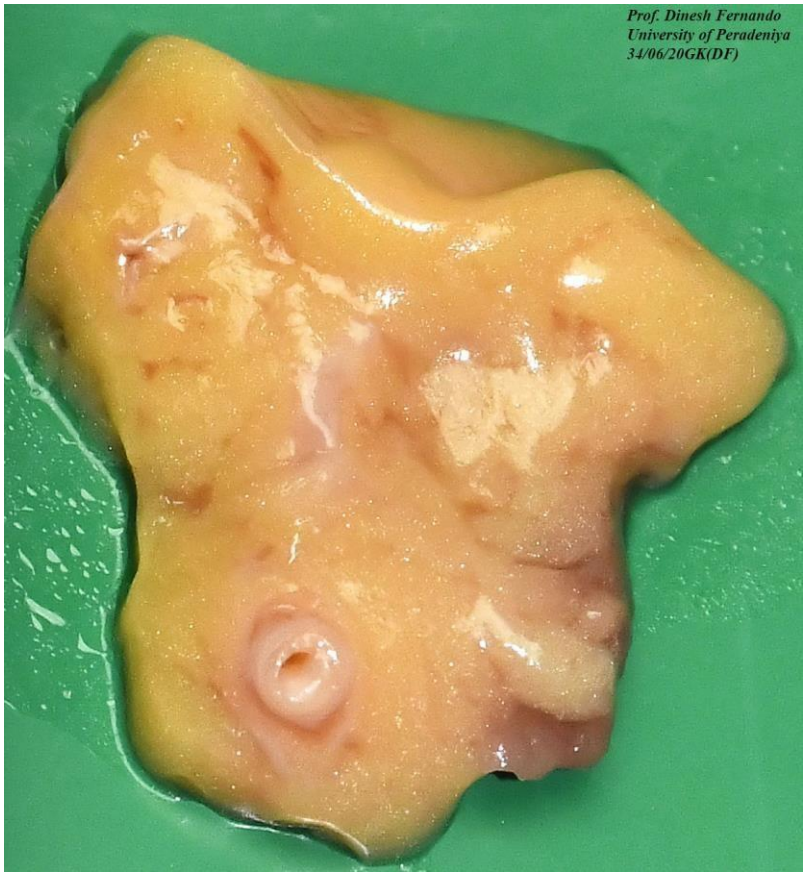


Figure 2: Concentric Atheroma

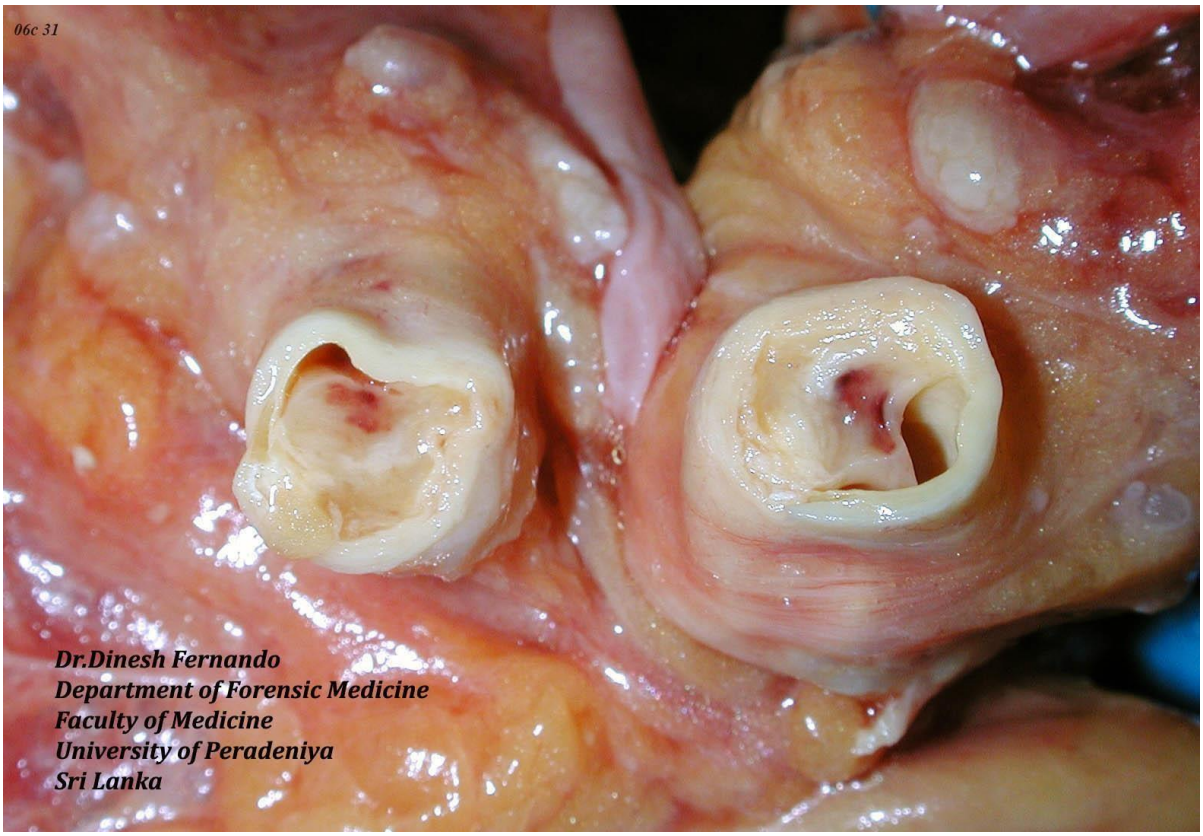


Figure 3: Haemorrhage into an eccentric atheromatous plaque in the coronary artery

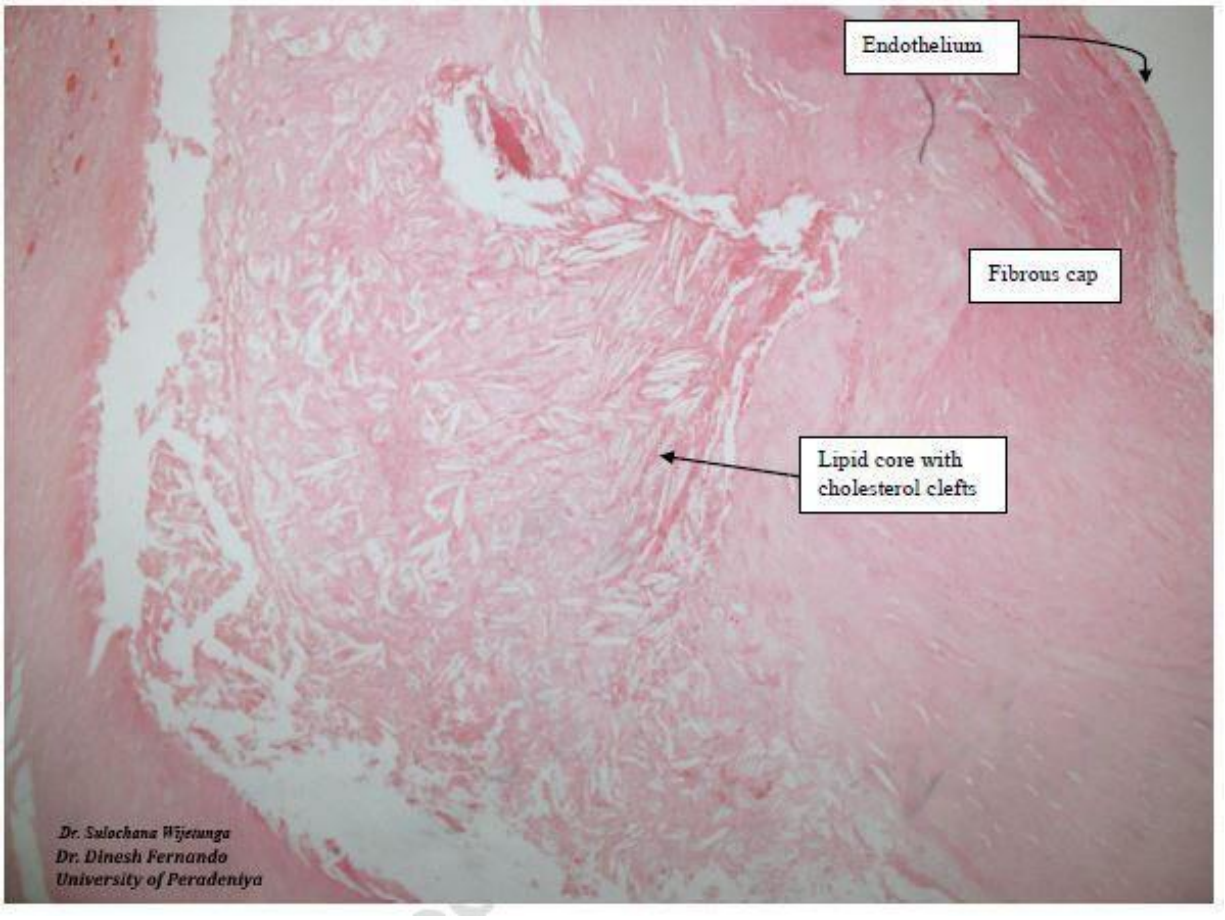


Figure 4: An uninterrupted atheromatous plaque narrowing the coronary artery lumen. Atheromatous plaque is an intimal collection of lipids. The picture shows a typical atheromatous plaque covered with a fibrous cap and a central lipid core. The lipid core can be identified by presence of cholesterol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

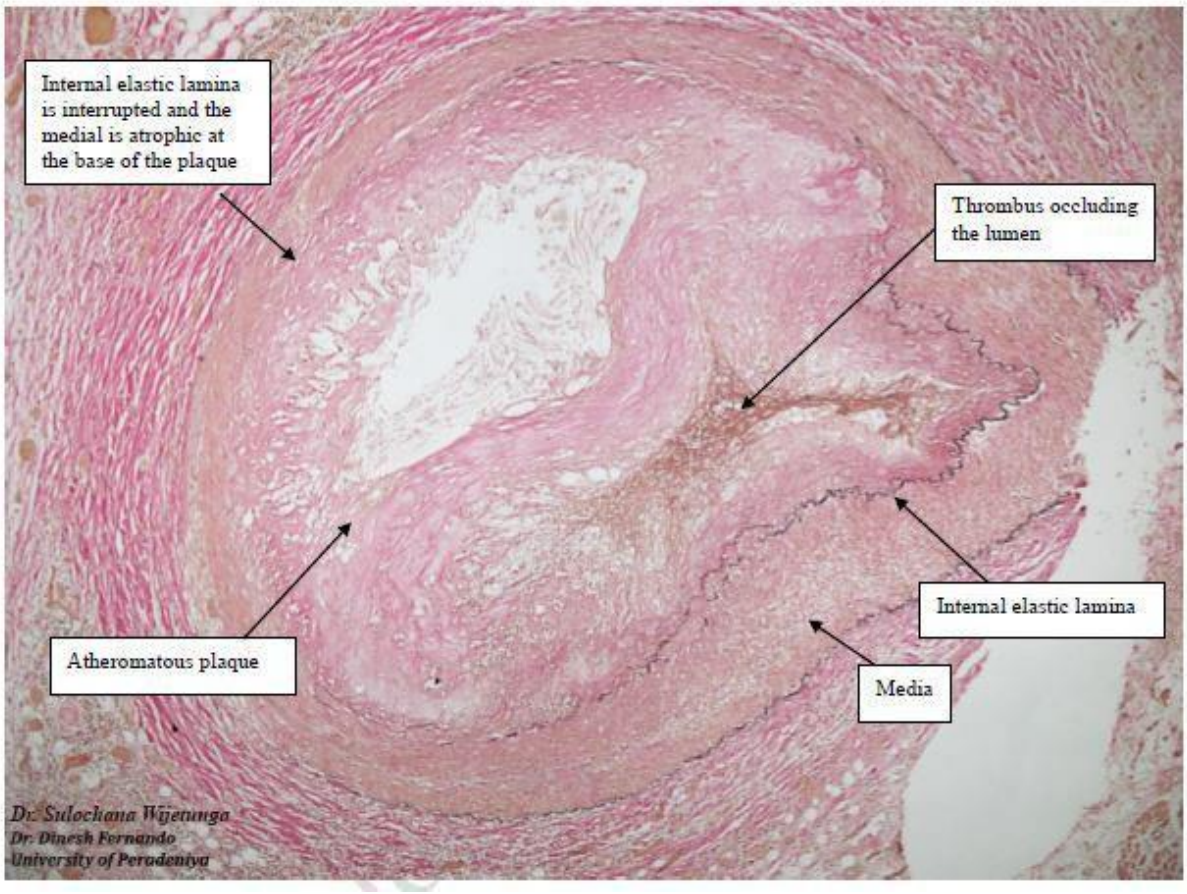


Figure 5: Ulcerated atheromatous plaque with superimposed thrombosis, completely occluding the arterial lumen (elastic van Gieson stain). Staining of the internal elastic lamina shows the intimal location of the plaque. The internal elastic lamina is disrupted and the media is atrophic at the base of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

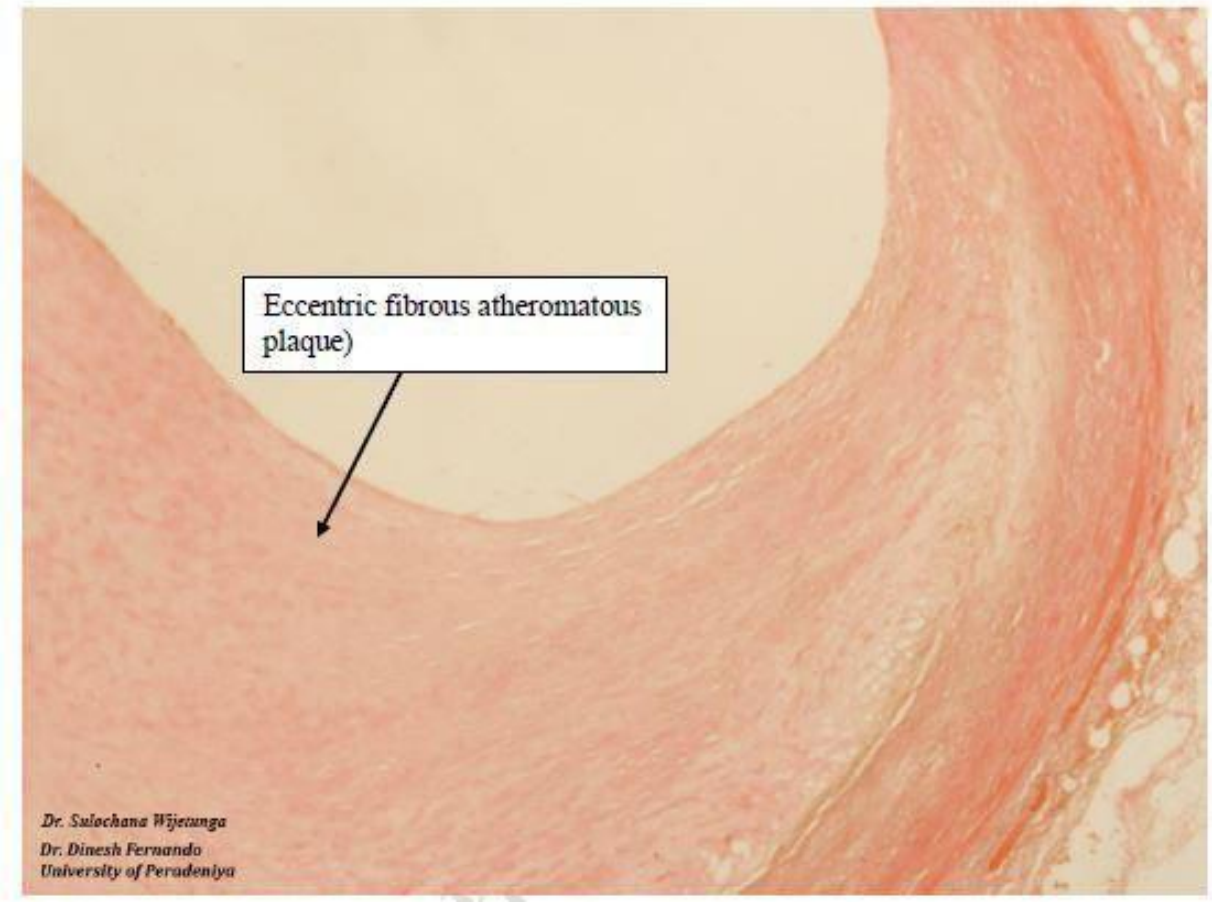


Figure 6: An uninterrupted atheromatous plaque narrowing the coronary artery lumen. Note that this plaque is almost completely composed of fibrous tissue and the typical lipid core is not seen. In long standing plaques, when there is no active deposition of lipids, the plaques get ultimately replaced 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

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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.

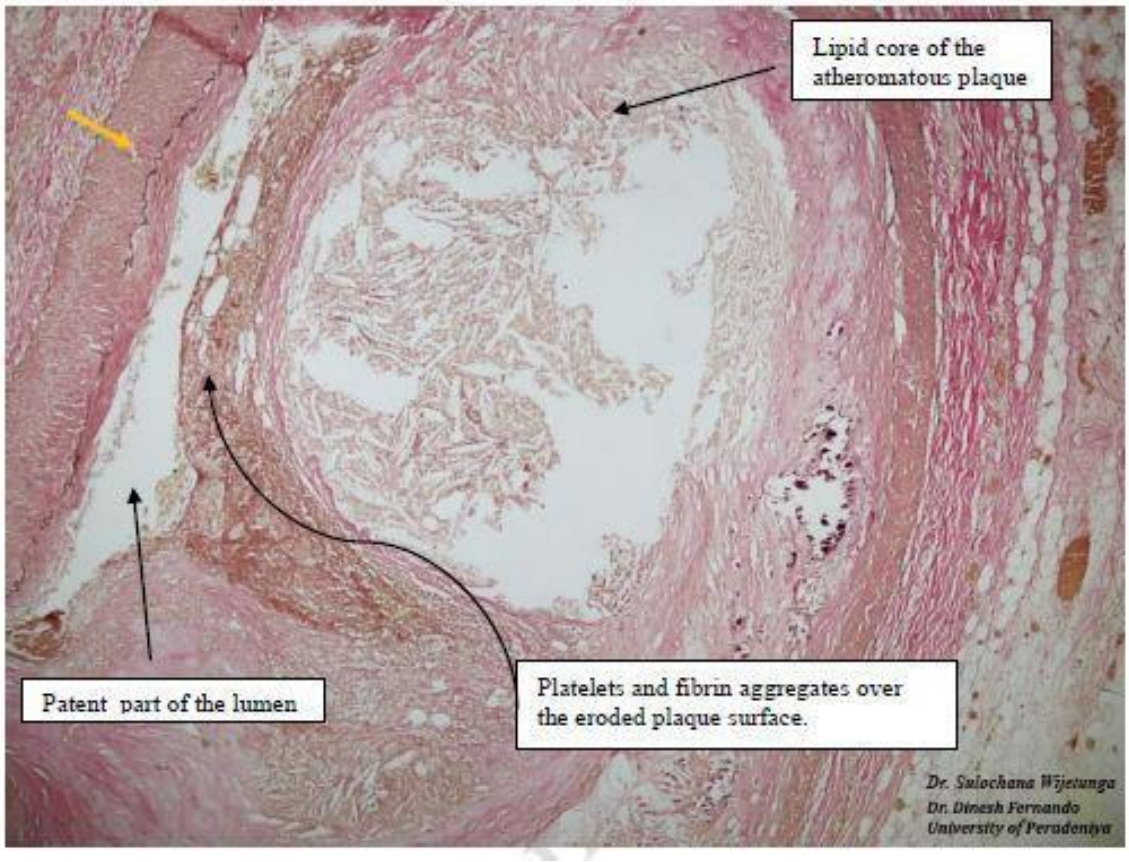


Figure 7: An eroded plaque with platelet aggregations on the surface, without complete obstruction of the arterial lumen. This is a typical plaque that is vulnerable to get acute plaque changes: there is a large lipid core (identified by the presence of cholesterol clefts) with only a thin fibrous cap. 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.

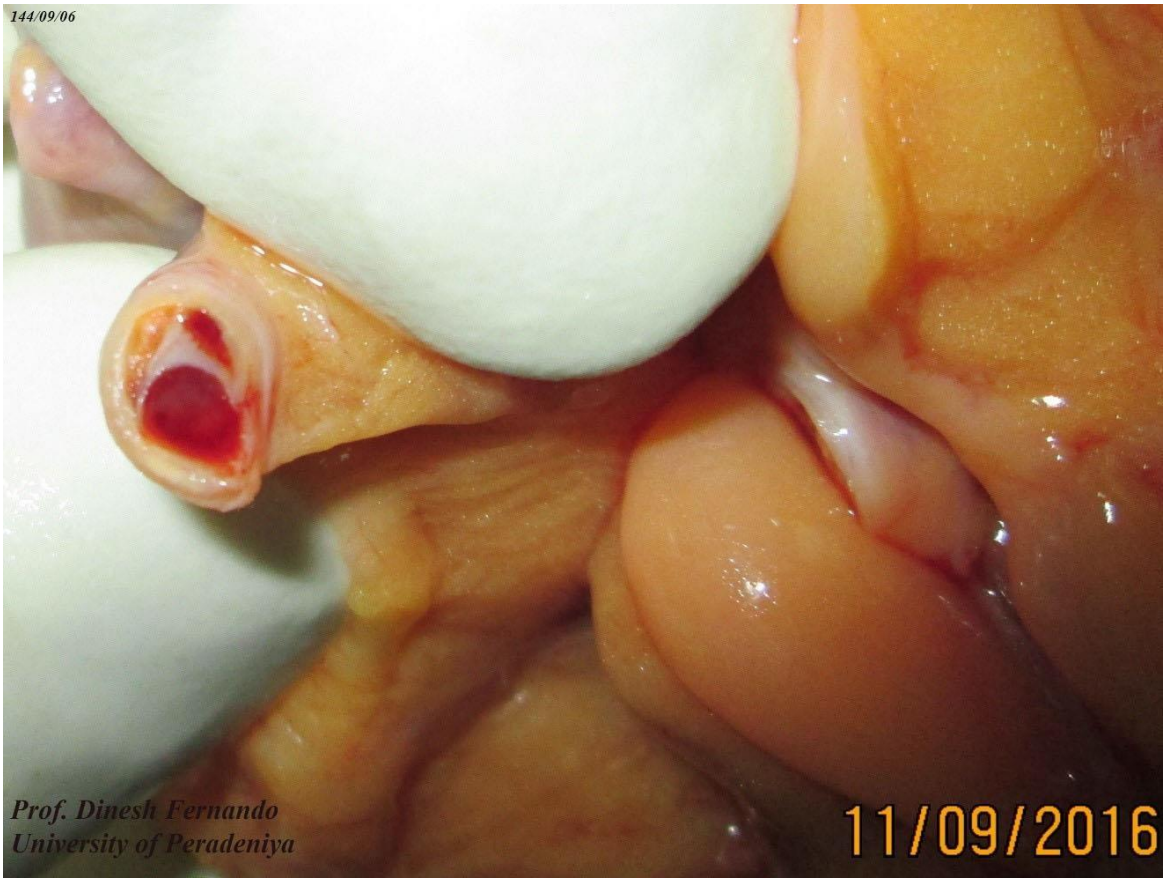


Figure 8: Plaque with superimposed thrombosis

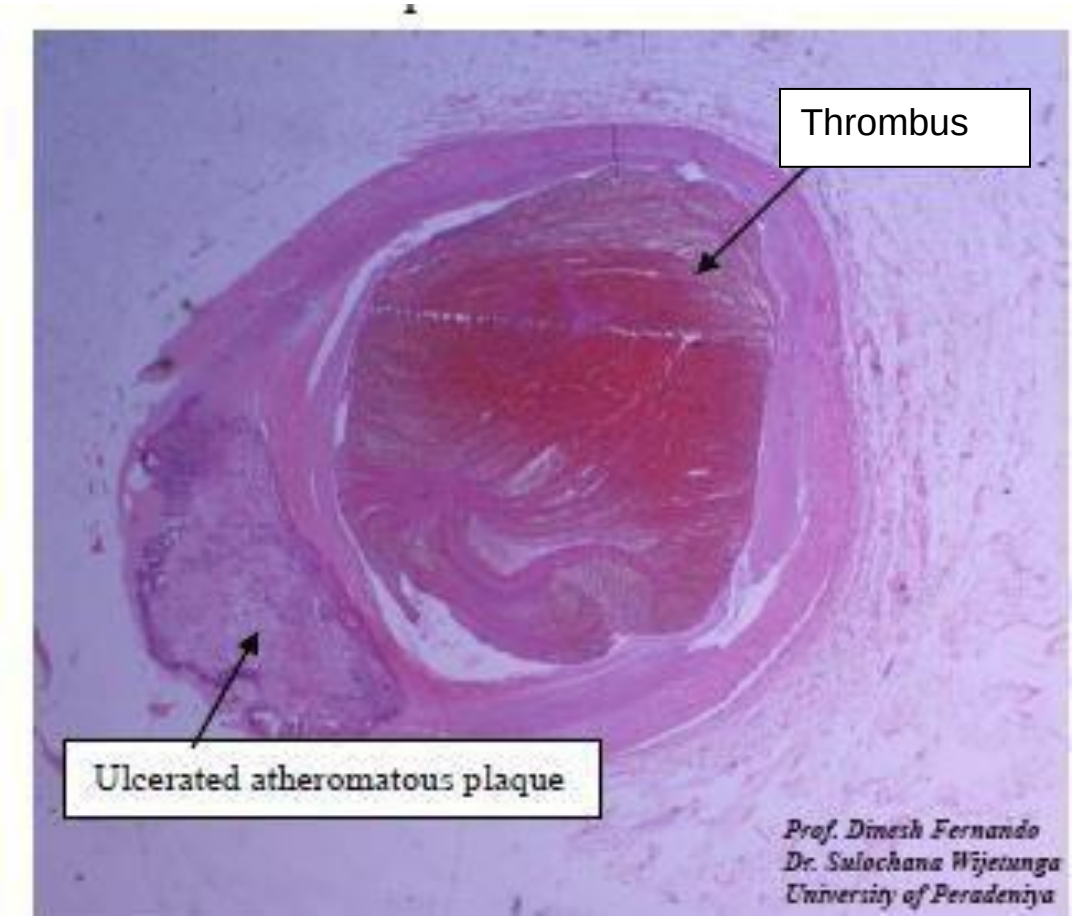


Figure 9: An ulcerated eccentric plaque with superimposed thrombosis, completely occluding the arterial lumen

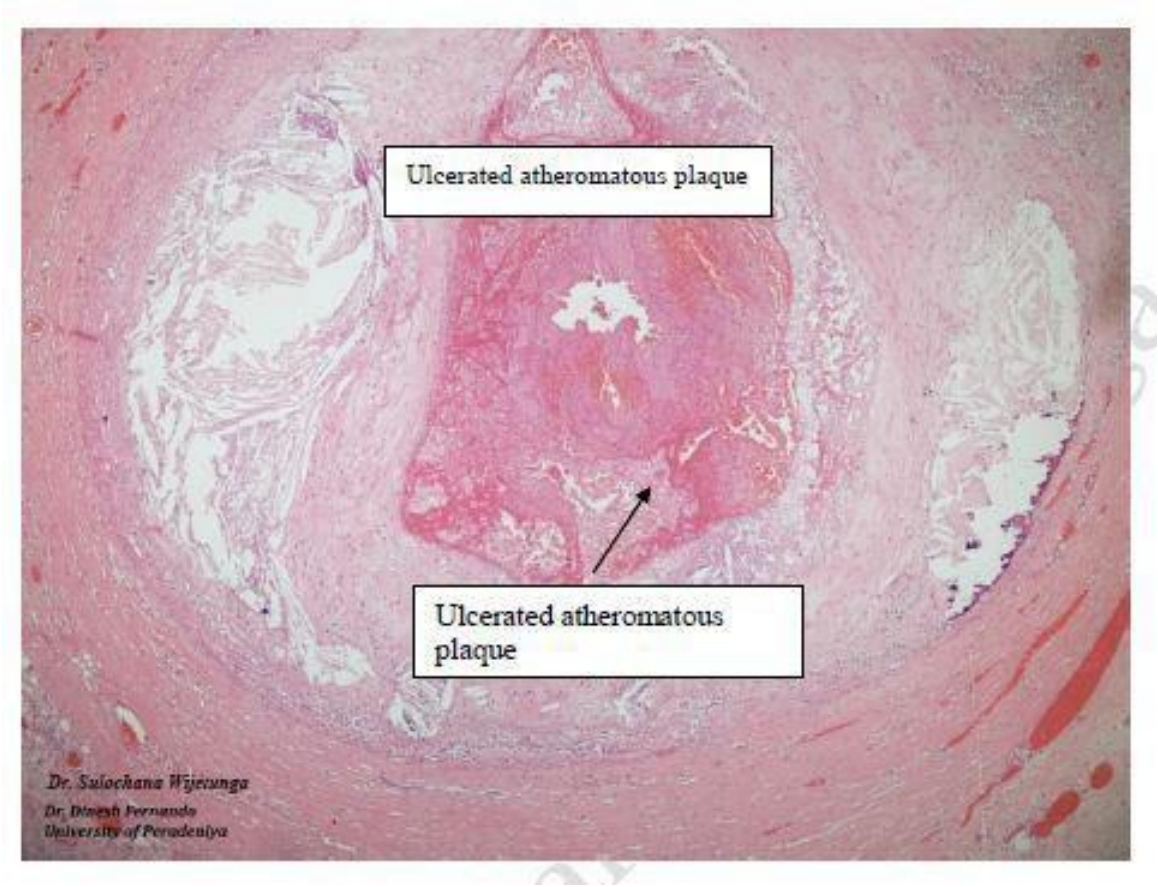


Figure 10: Ulcerated atheromatous plaque with superimposed thrombus, completely occluding the arterial lumen. The atheromatous plaque is more circumferential in this example. The cholesterol clefts in the lipid core as clearly seen.

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.

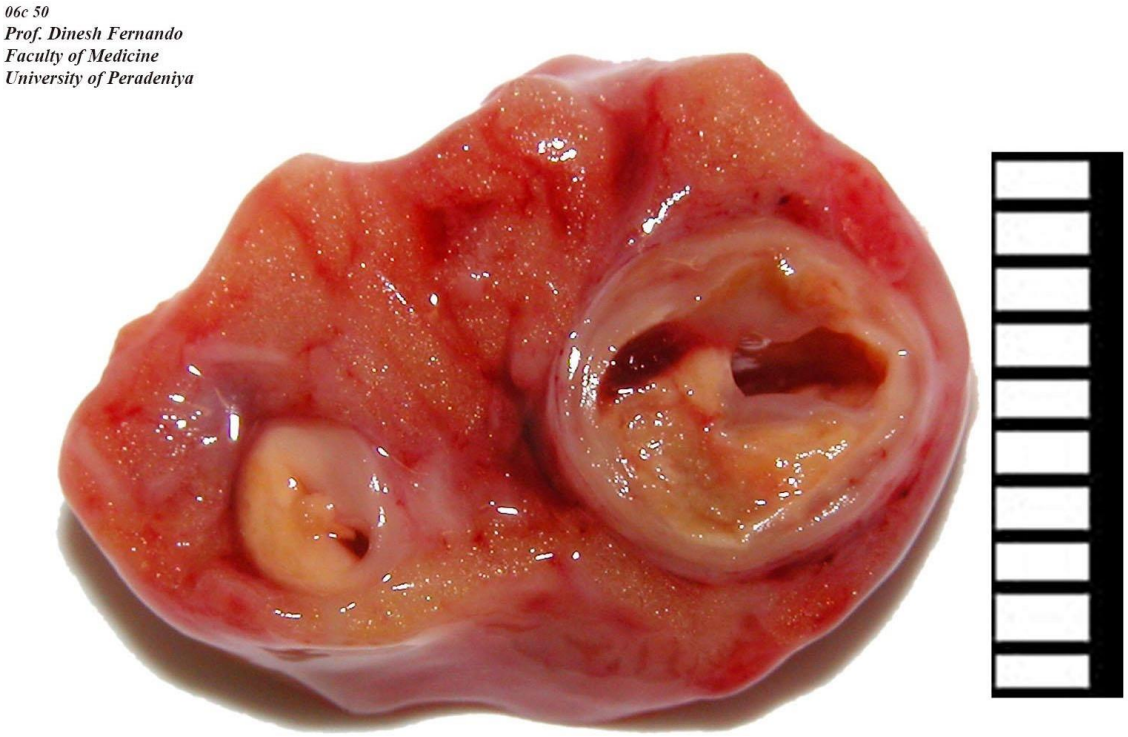


Figure 11: Bleeding into an eccentric atheromatous plaque

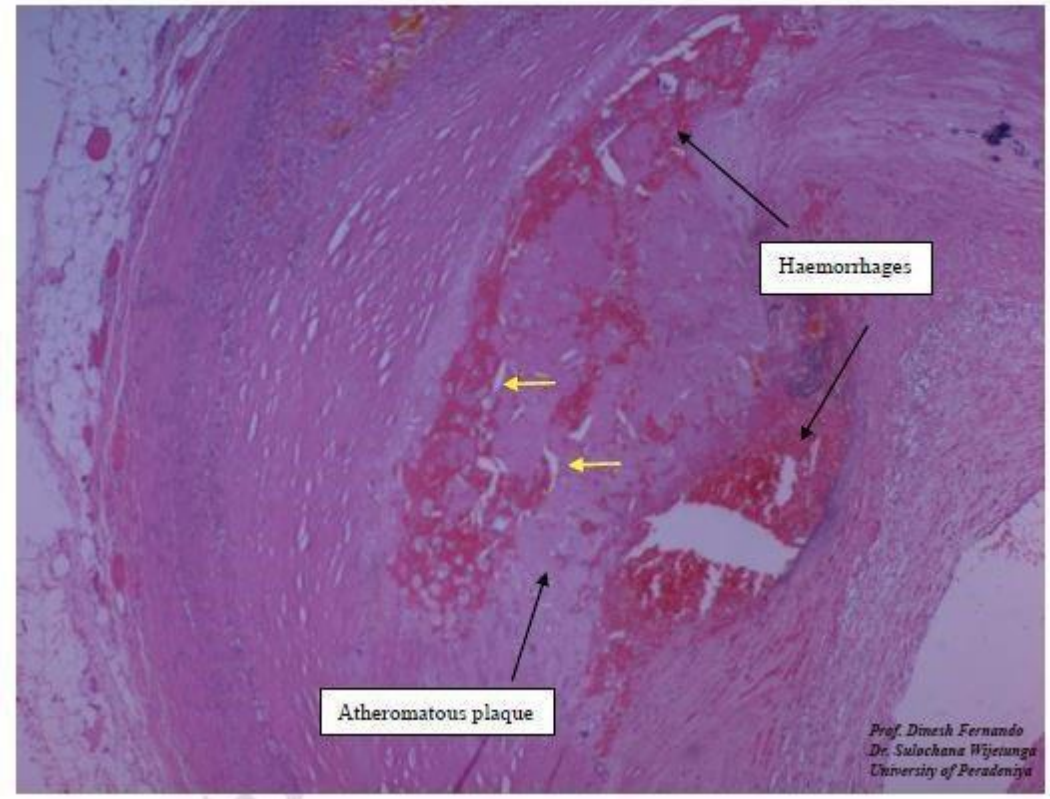


Figure 12: Bleeding into a plaque

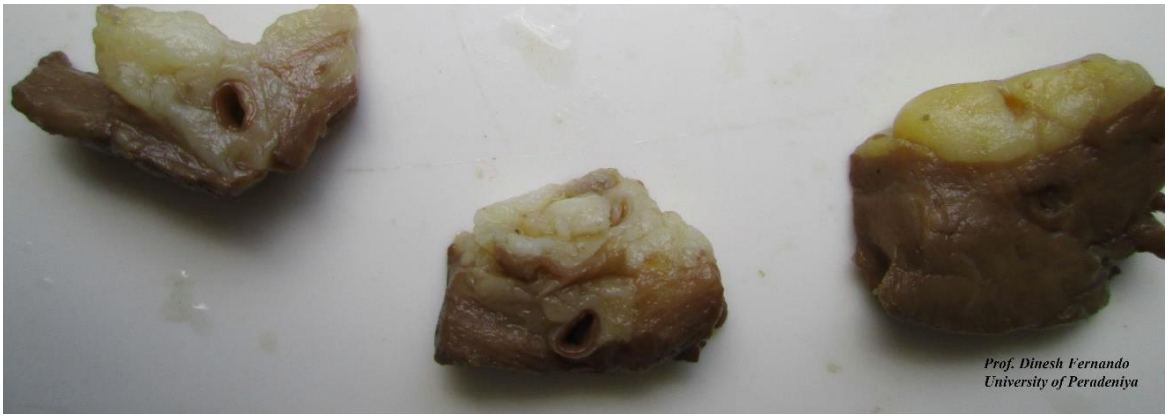
2. Coronary arterial spasms

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.

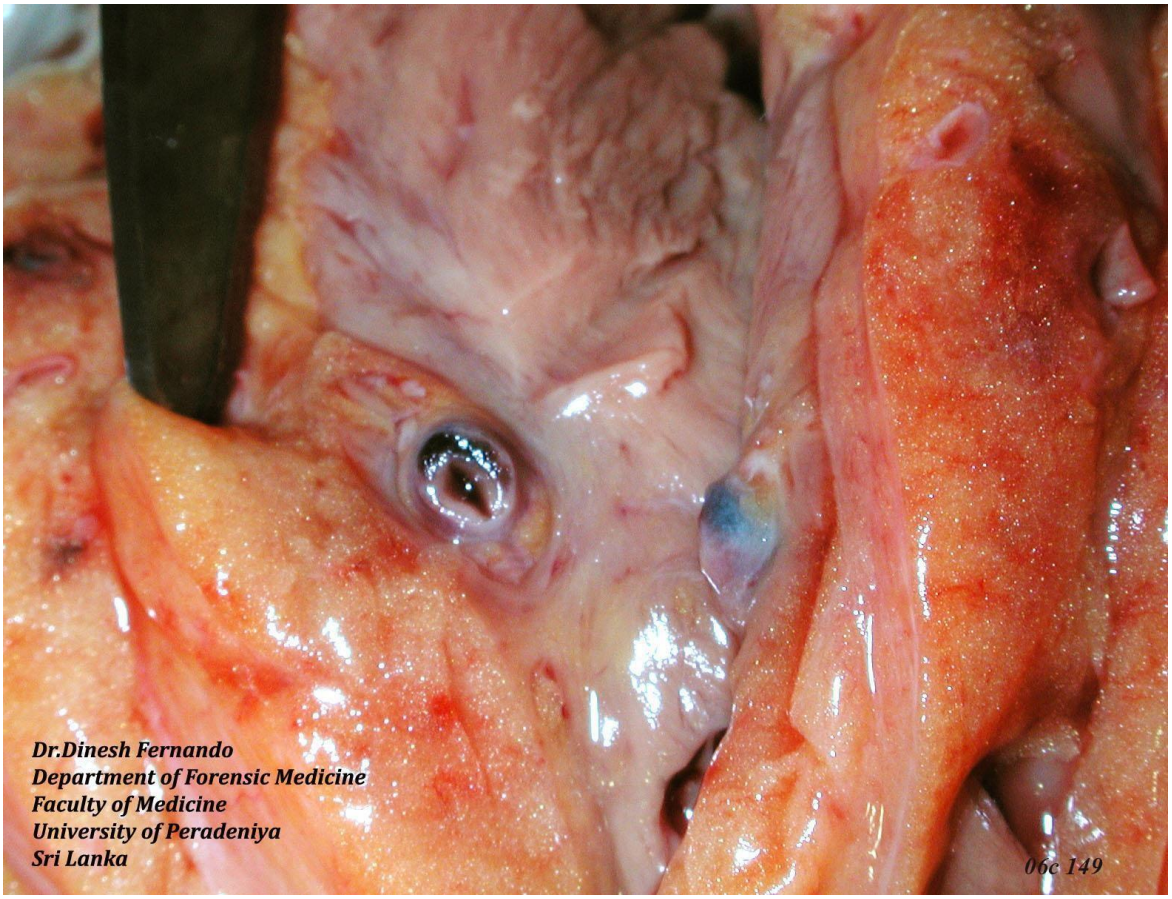
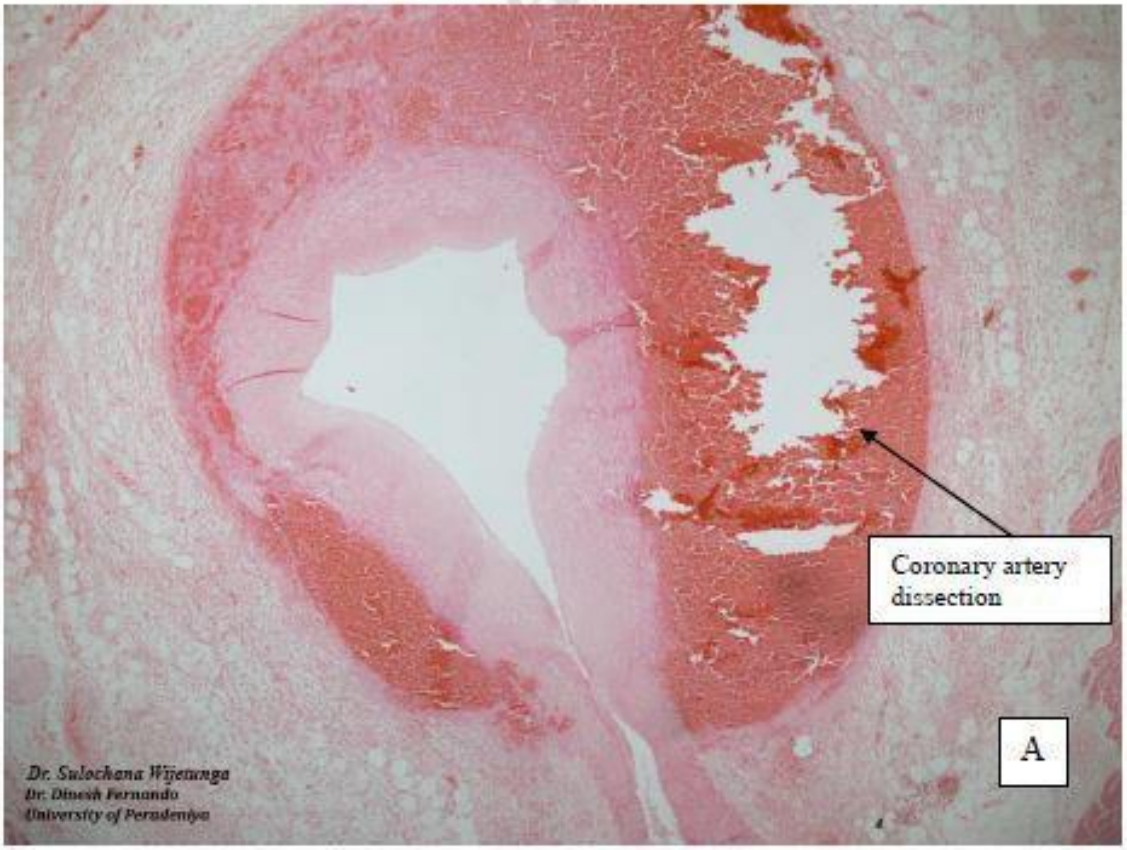


Figure 15: Coronary artery dissection


Coronary artery dissection
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University of Peradeniya

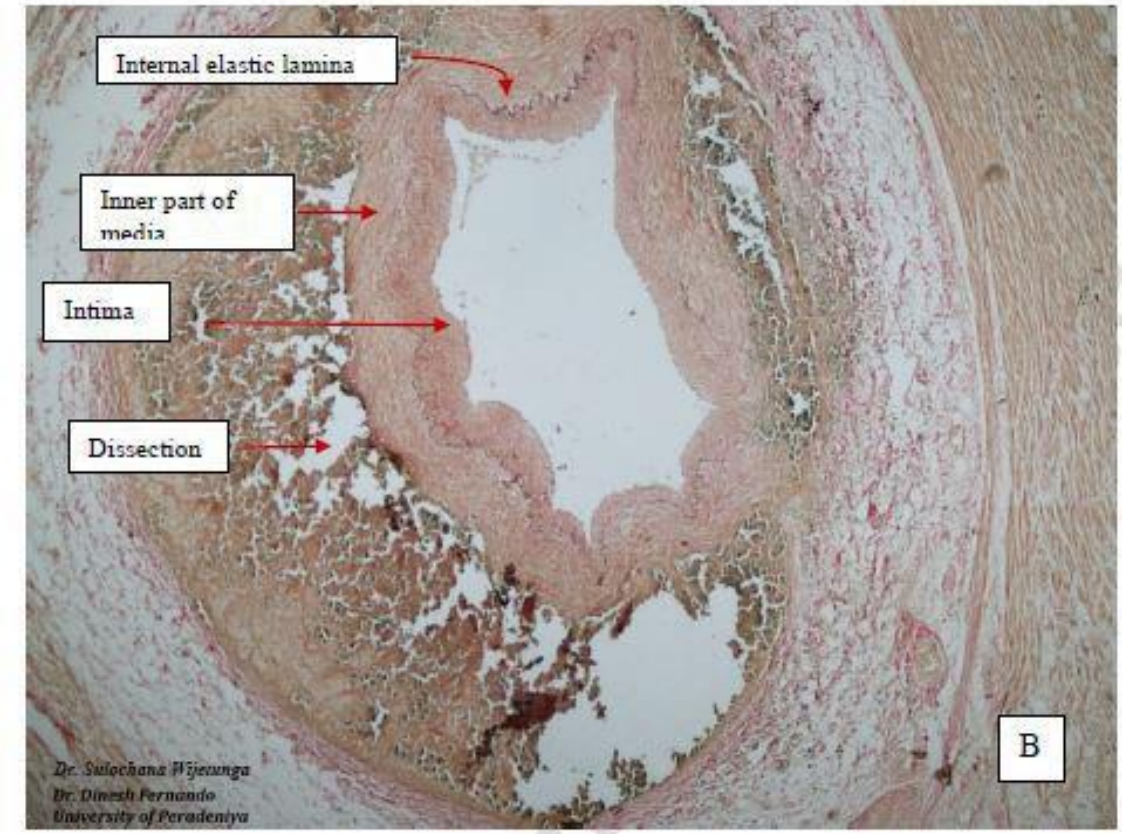


Figure 16: Coronary artery dissection
A) Haematoxylin and eosin stain show a blood-filled channel within the arterial wall
B) Elastin stain demonstrates that the dissection has occurred within the medial planes

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.

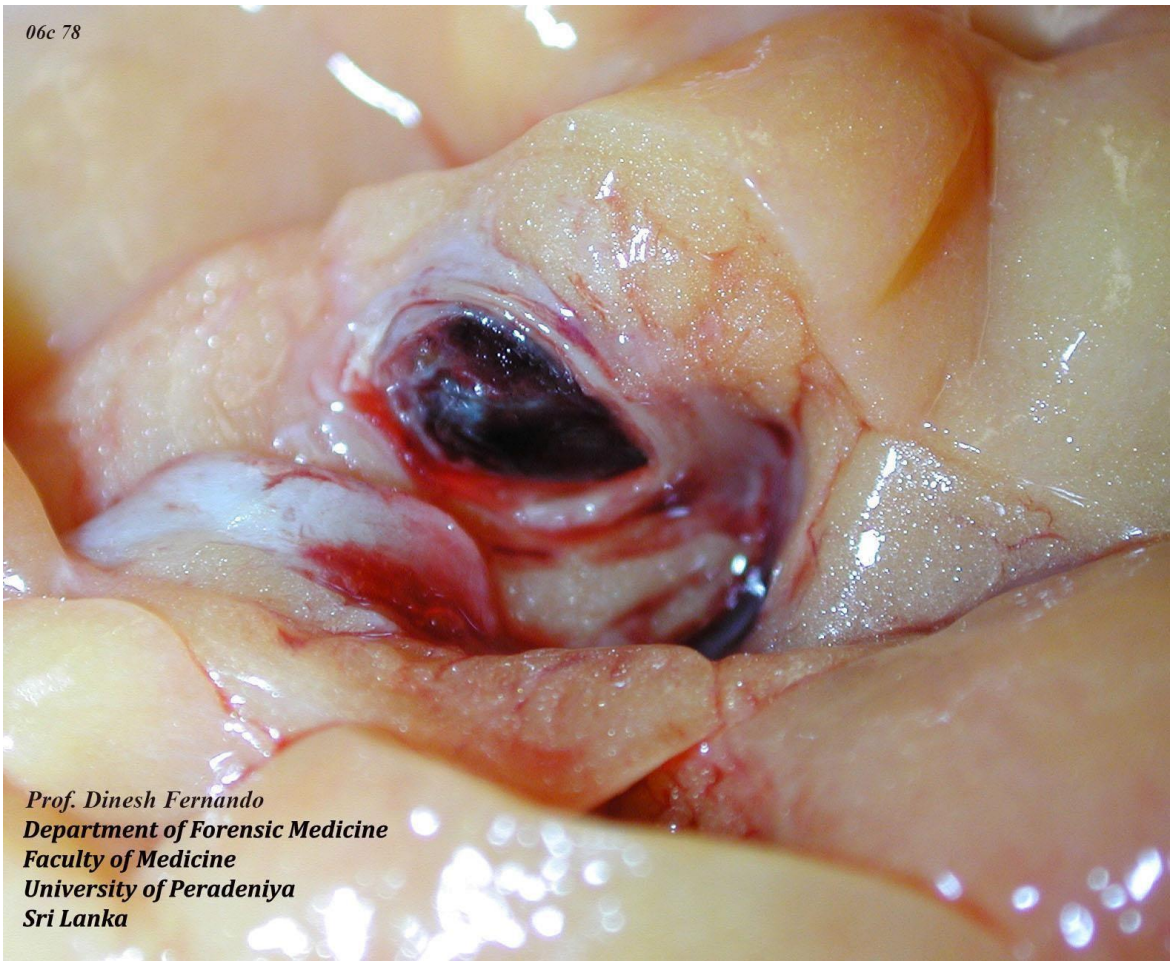
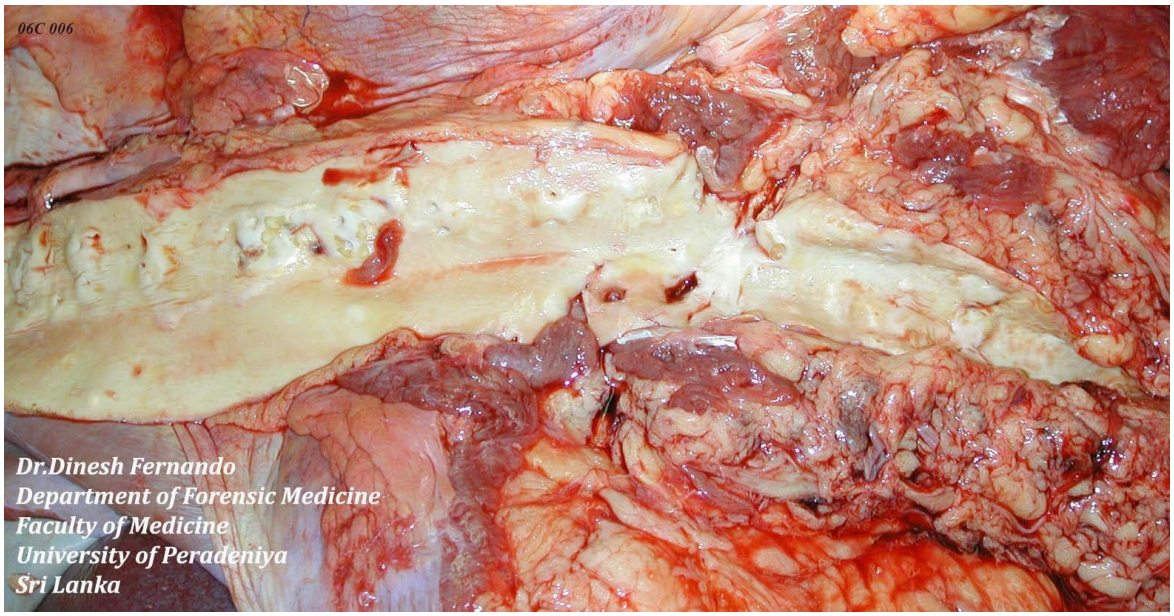
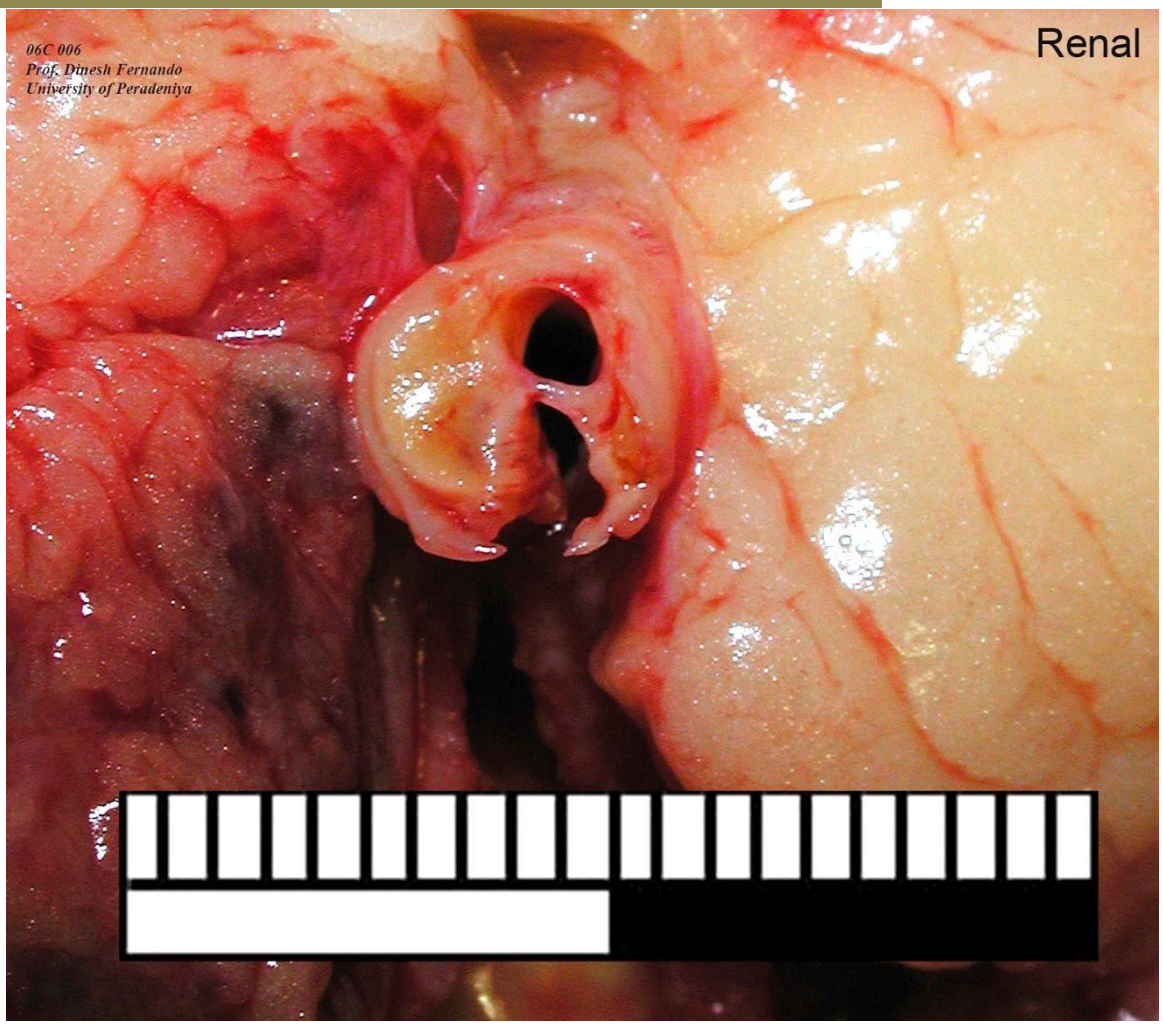


Figure 17: Coronary thrombo-embolism

C) Atheroma in other vessels



(a)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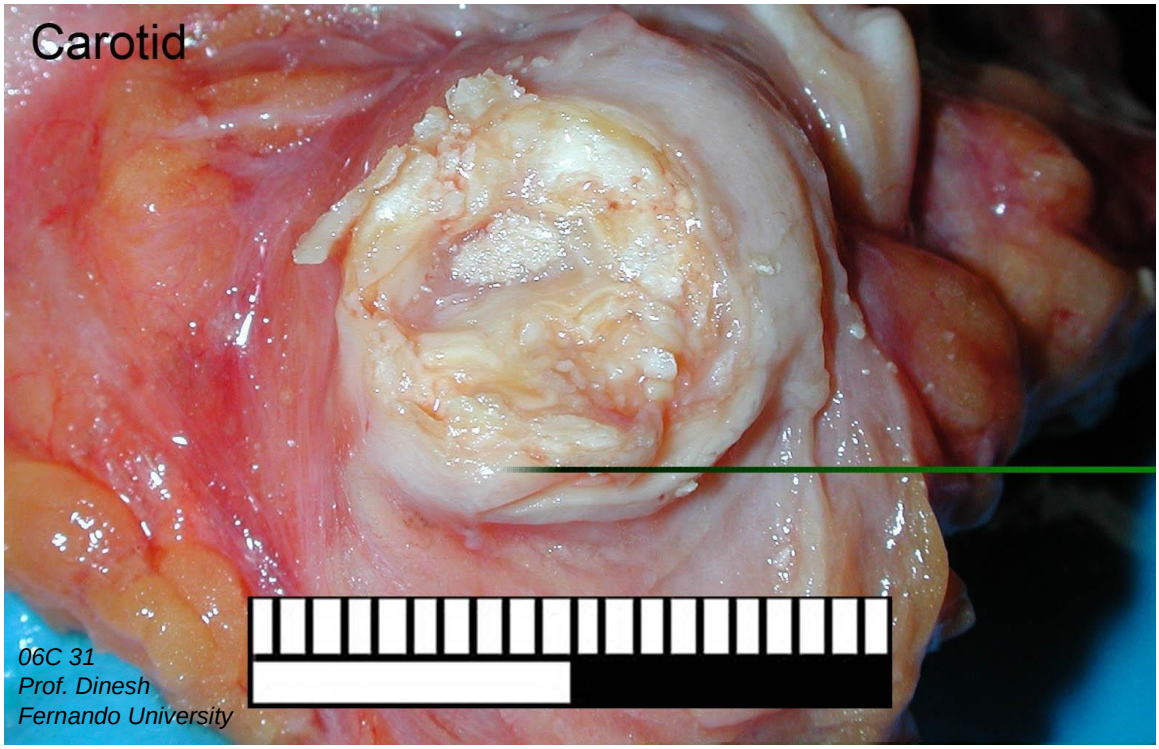
Renal

(b)



(c)

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