COLOR ATLAS OF

FORENSIC PATHOLOGY

BRAIN AND SPINAL CORD

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Version 1

BRAIN AND SPINAL CORD

ILLUSTRATIVE CASES

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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.

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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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3. CEREBRAL INFARCTION

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

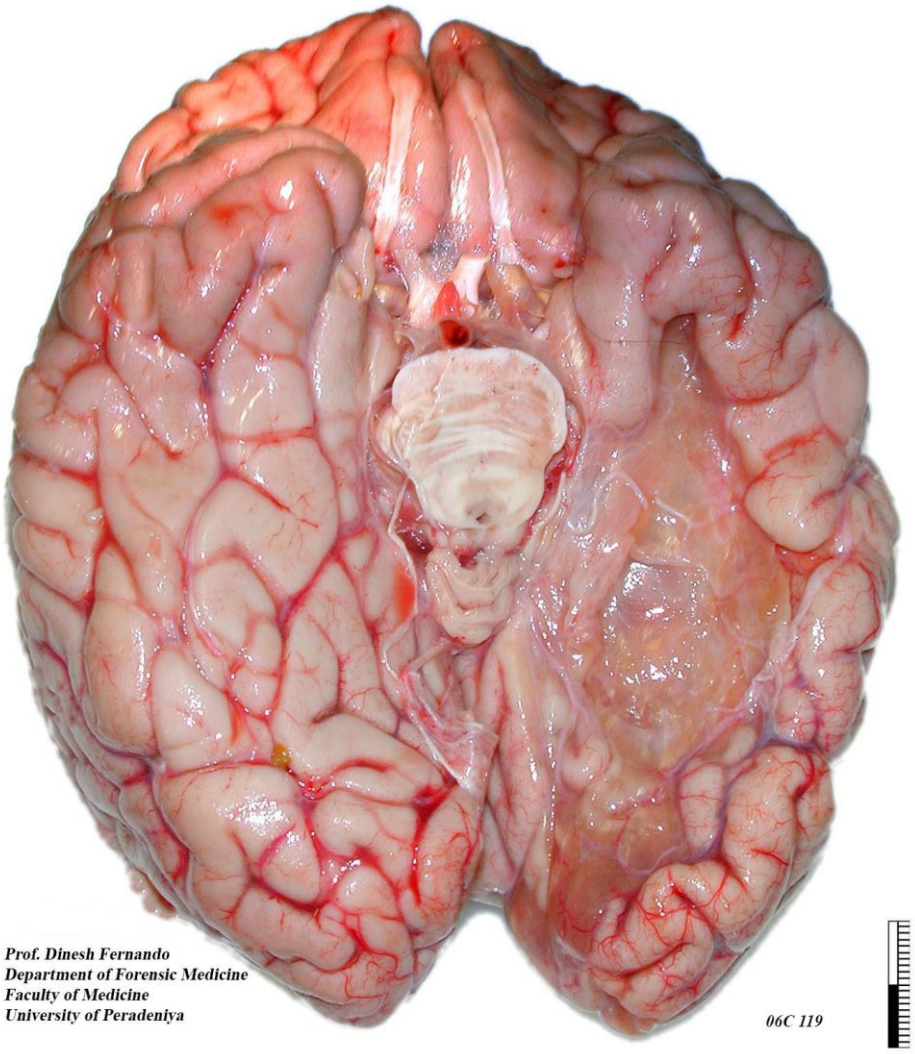
A 77-year-old male who had several strokes over the past six years and had pancreatitis for the past four years presented to the local hospital with a productive cough of a few days and severe abdominal pain, especially in the right upper quadrant. He had vomited bile. Investigations showed raised amylase. 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

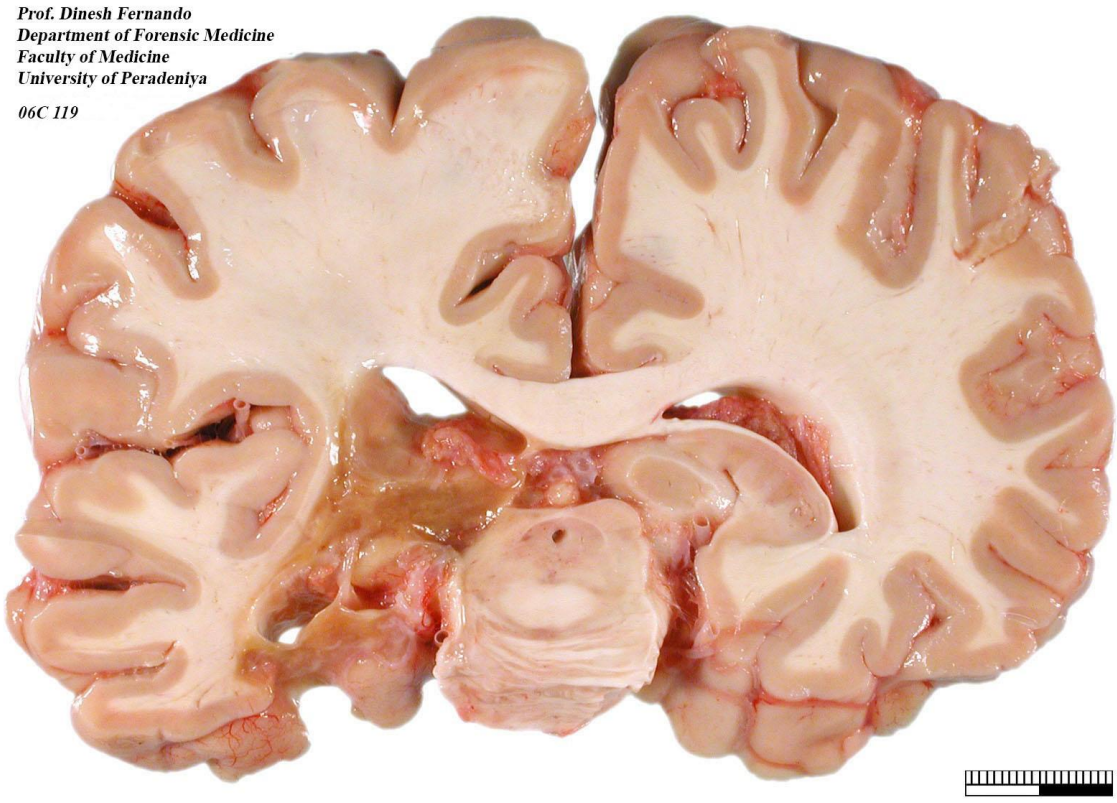
Central Nervous System: A gelatinous area was present on the inferior aspect of the left occipital lobe close to the midline. Multiple atheromatous plaques were present in the Circle of Willis which had dilated vessels. Multiple sections of the cerebral hemispheres revealed an area of old necrosis in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



(a)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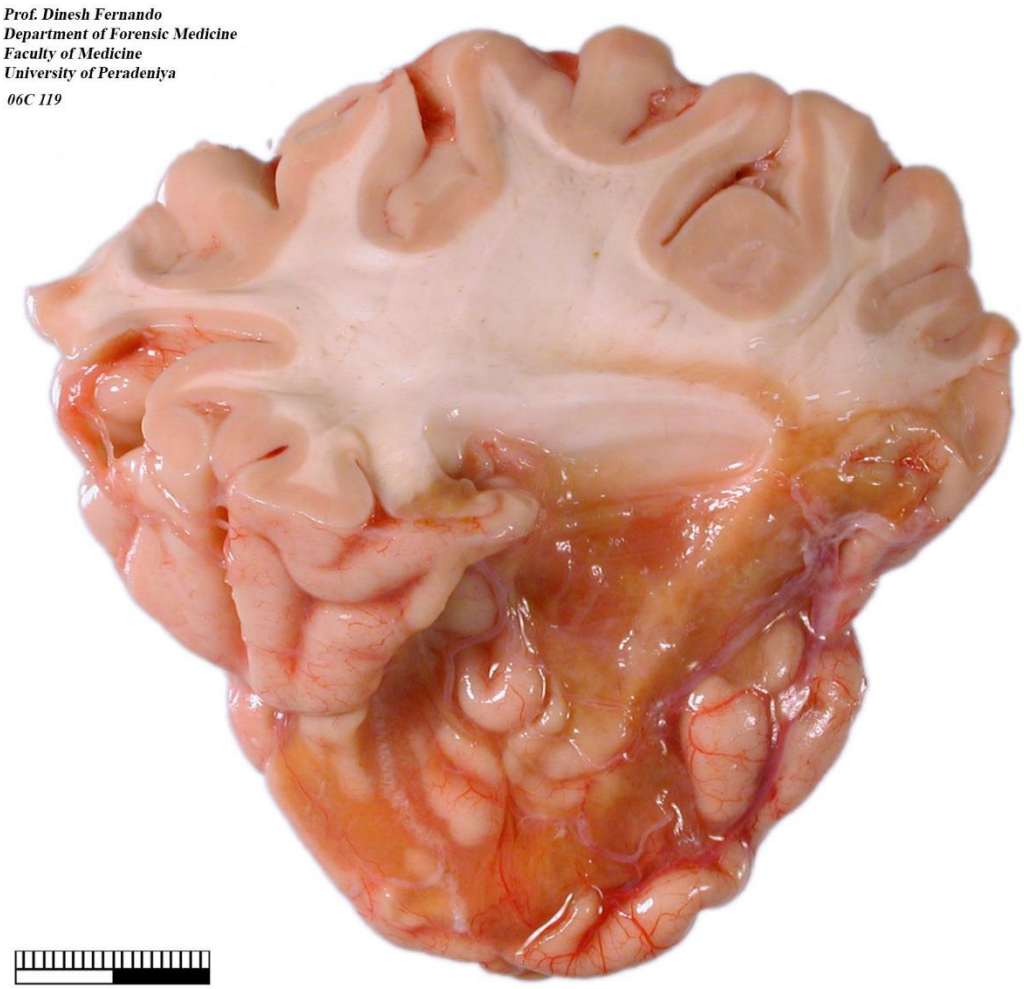
(b)



(c)

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

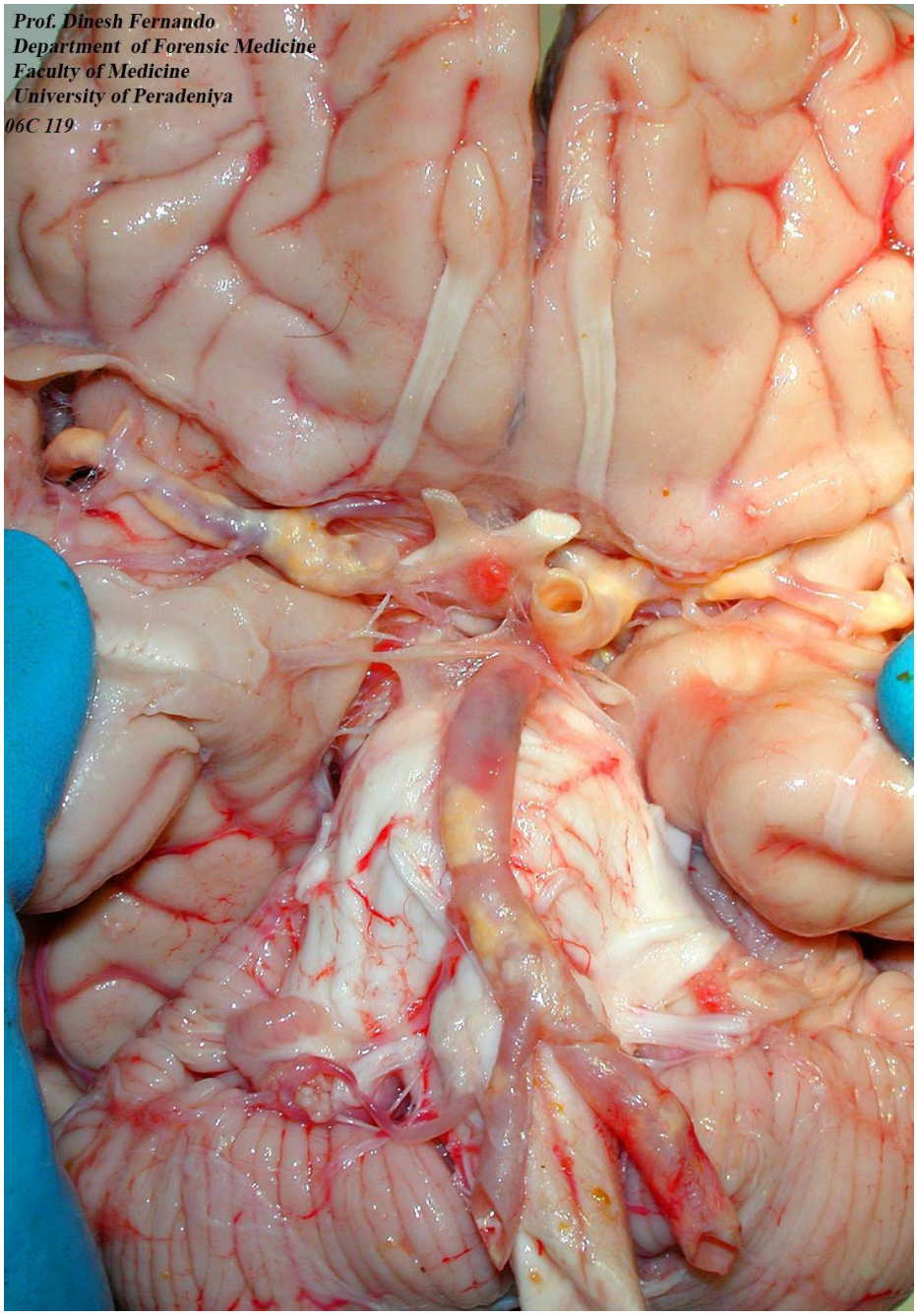


Figure 2: Atheromatous plaques in the circle of Willis

Cause of death

Acute pancreatitis in a person with previous cerebral infarction

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