

COLOR ATLAS OF  
**FORENSIC  
PATHOLOGY**

**BRAIN AND SPINAL CORD**

# **COLOR ATLAS OF FORENSIC PATHOLOGY**

**Version 1**

**BRAIN AND SPINAL CORD**

**ILLUSTRATIVE CASES**

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**ISBN: 978-624-96229-0-6**

Last updated on 10/07/2020

## 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**

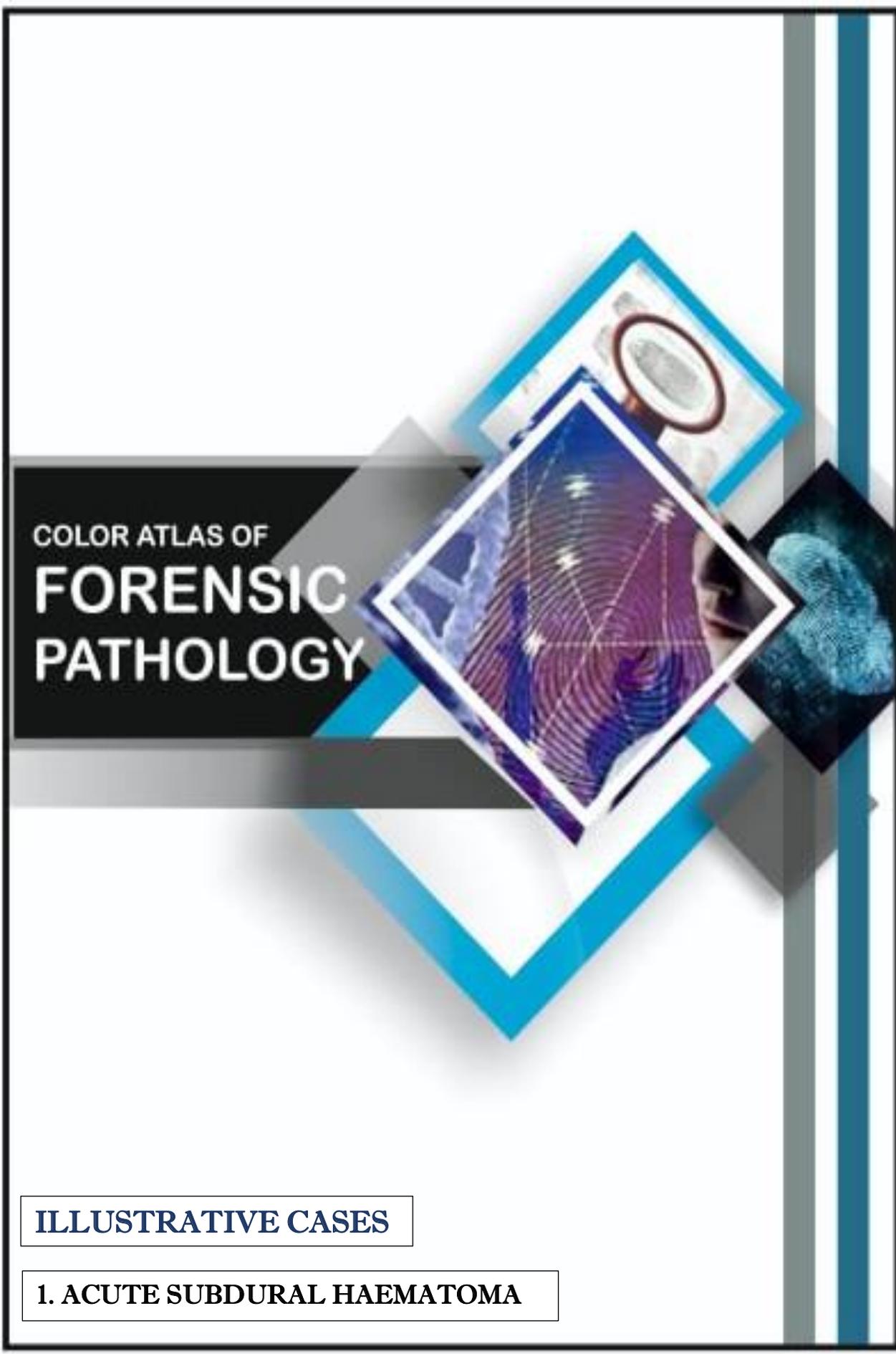
Forensic Medicine in Sri Lanka encompasses, both, examination of patients for medico-legal purposes and conducting autopsies in all unnatural deaths, in addition to those that the cause of death is not known. In the eyes of the justice system in Sri Lanka, all MBBS qualified medical officers are deemed 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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**ILLUSTRATIVE CASES**

**1. ACUTE SUBDURAL HAEMATOMA**



## 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 2<sup>nd</sup> 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.

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 uncus region of the hippocampal gyrus. Haemorrhage was seen along the imprint of the free margin of the tentorium cerebelli which extended from the uncus 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.

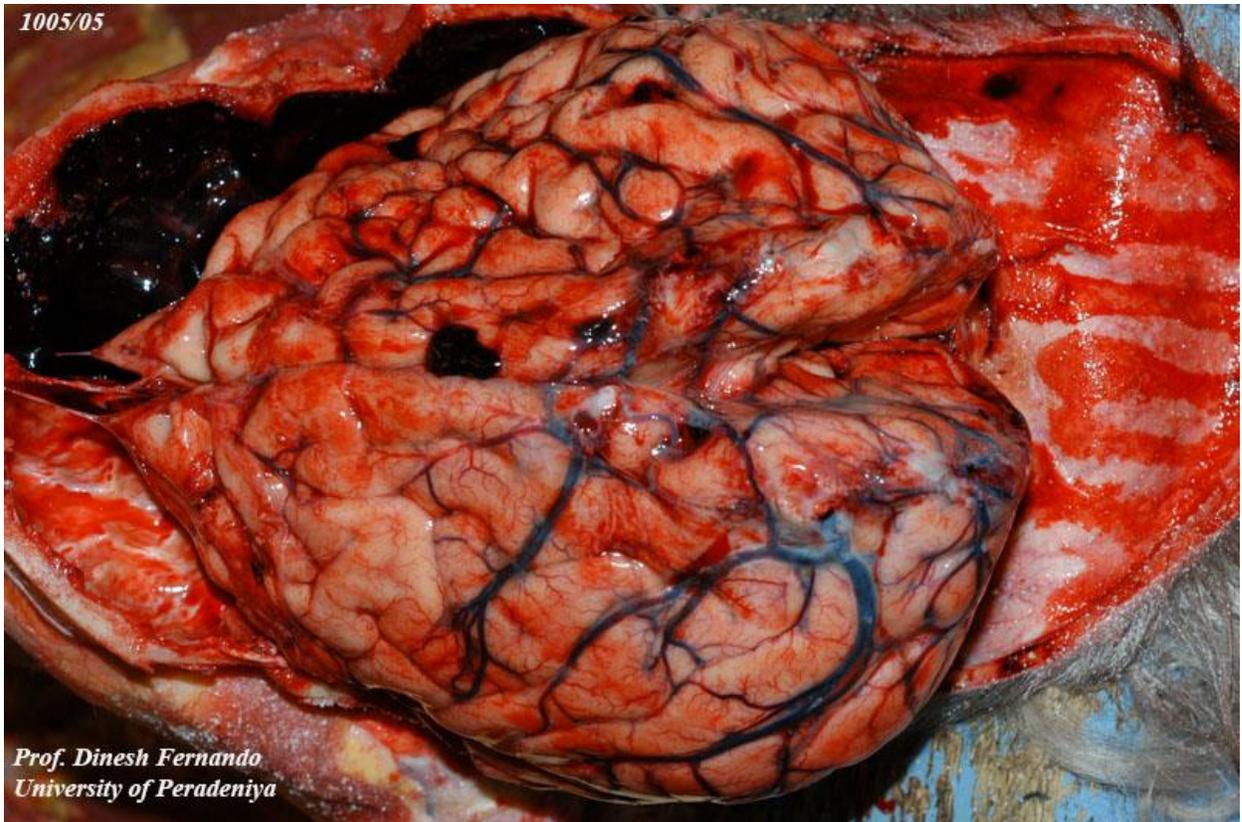


Figure 1: Acute subdural haematoma. Note: congestion of blood vessels

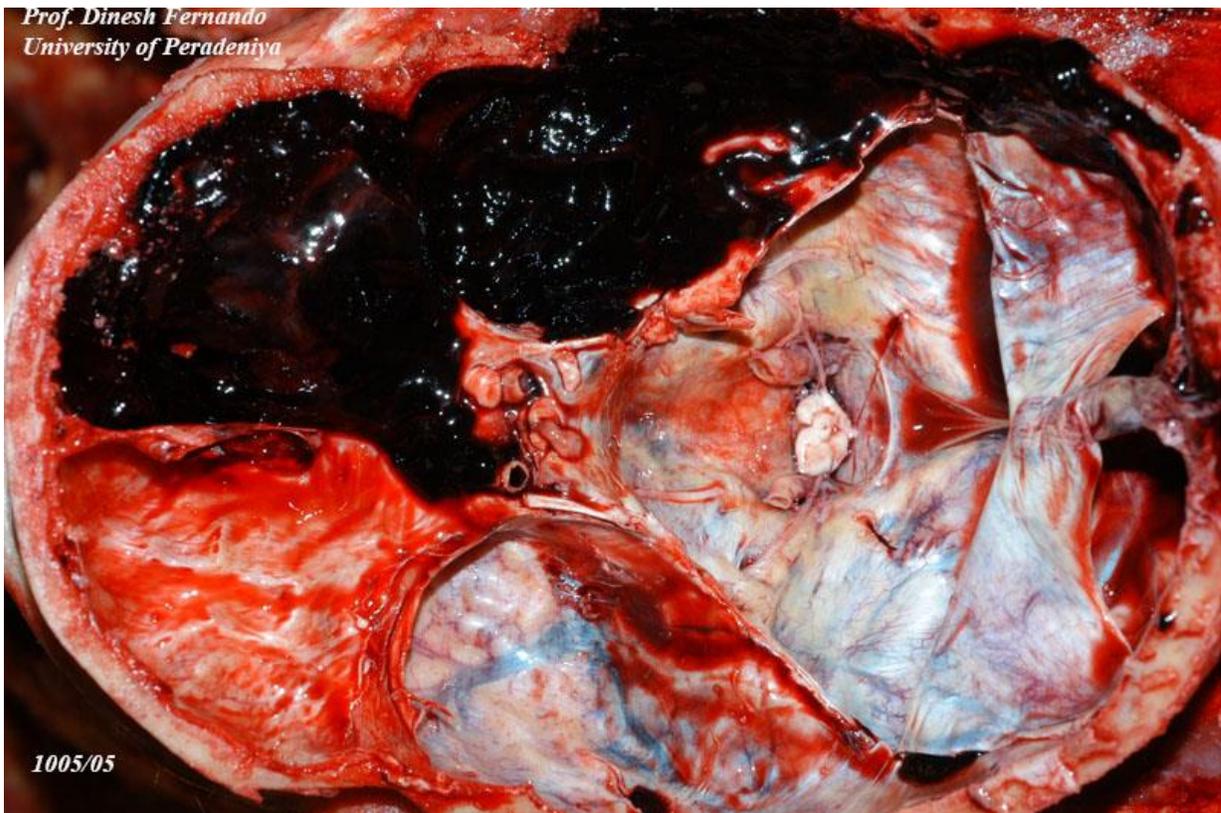


Figure 2: Acute subdural haematoma after removal of the brain

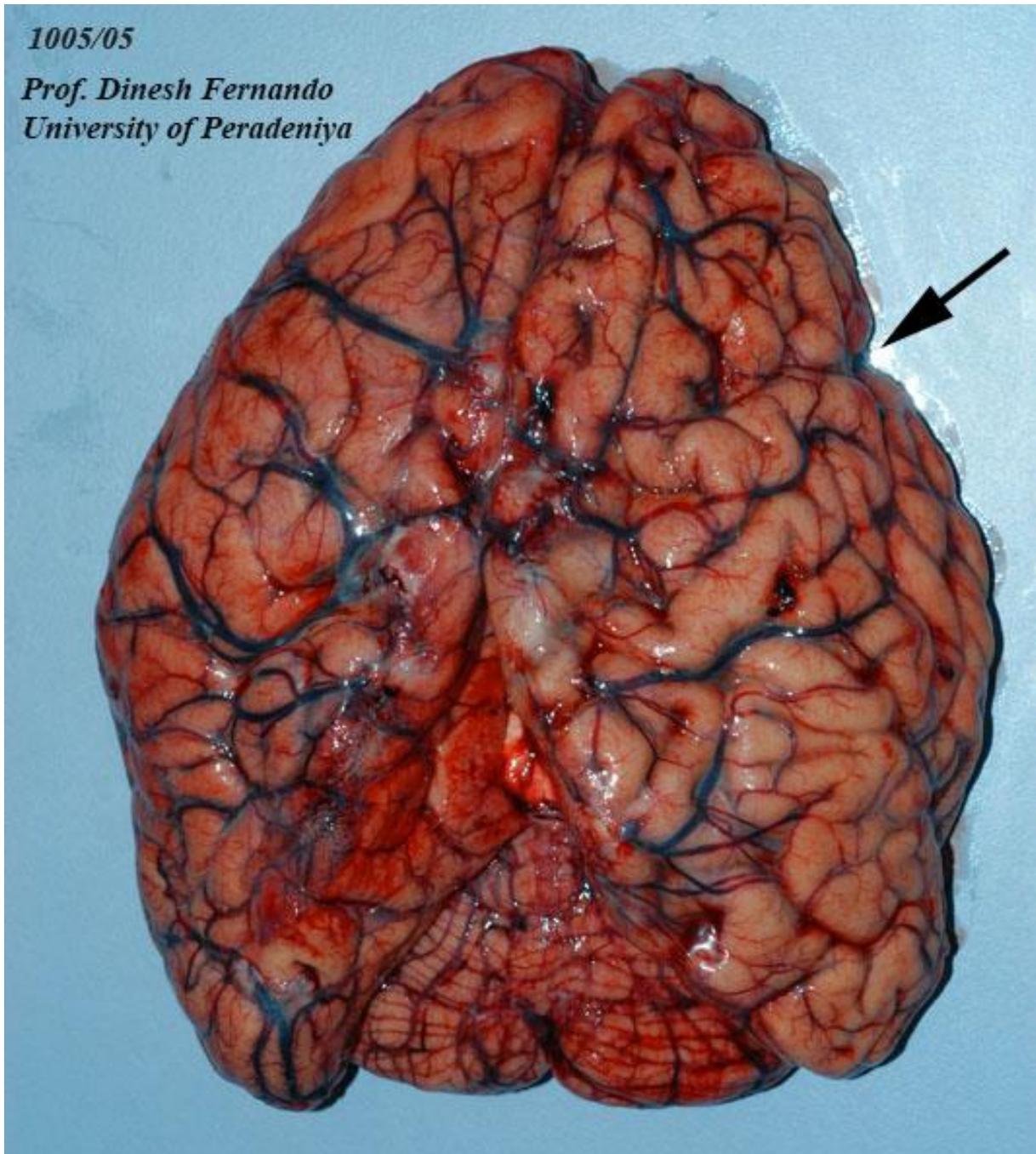
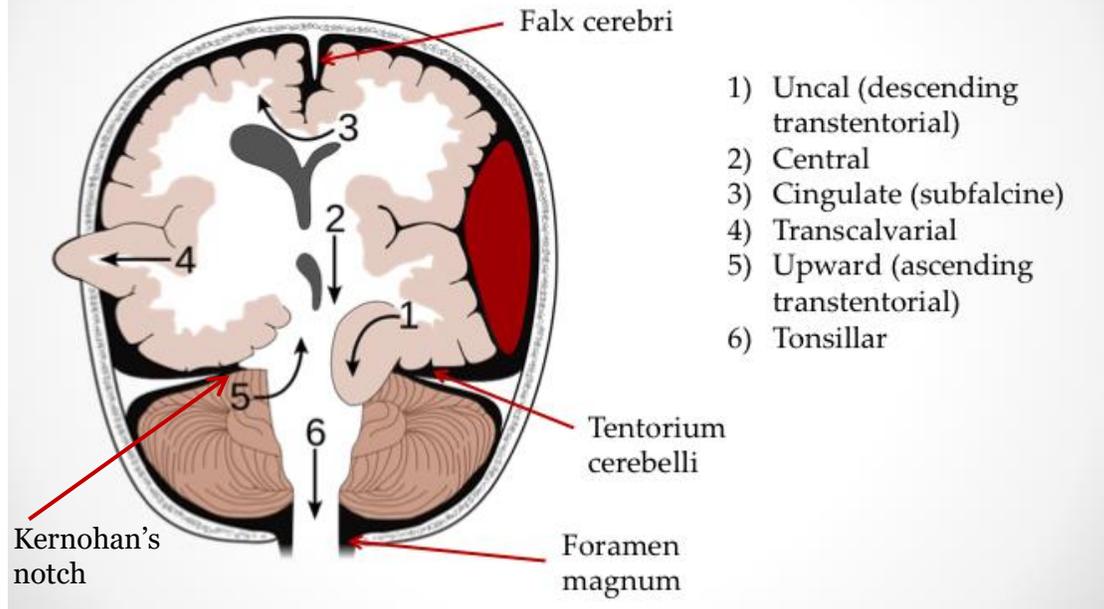


Figure 3: Cerebral oedema. Note: compression of the right cerebral hemisphere associated with midline shift to the left (arrow)

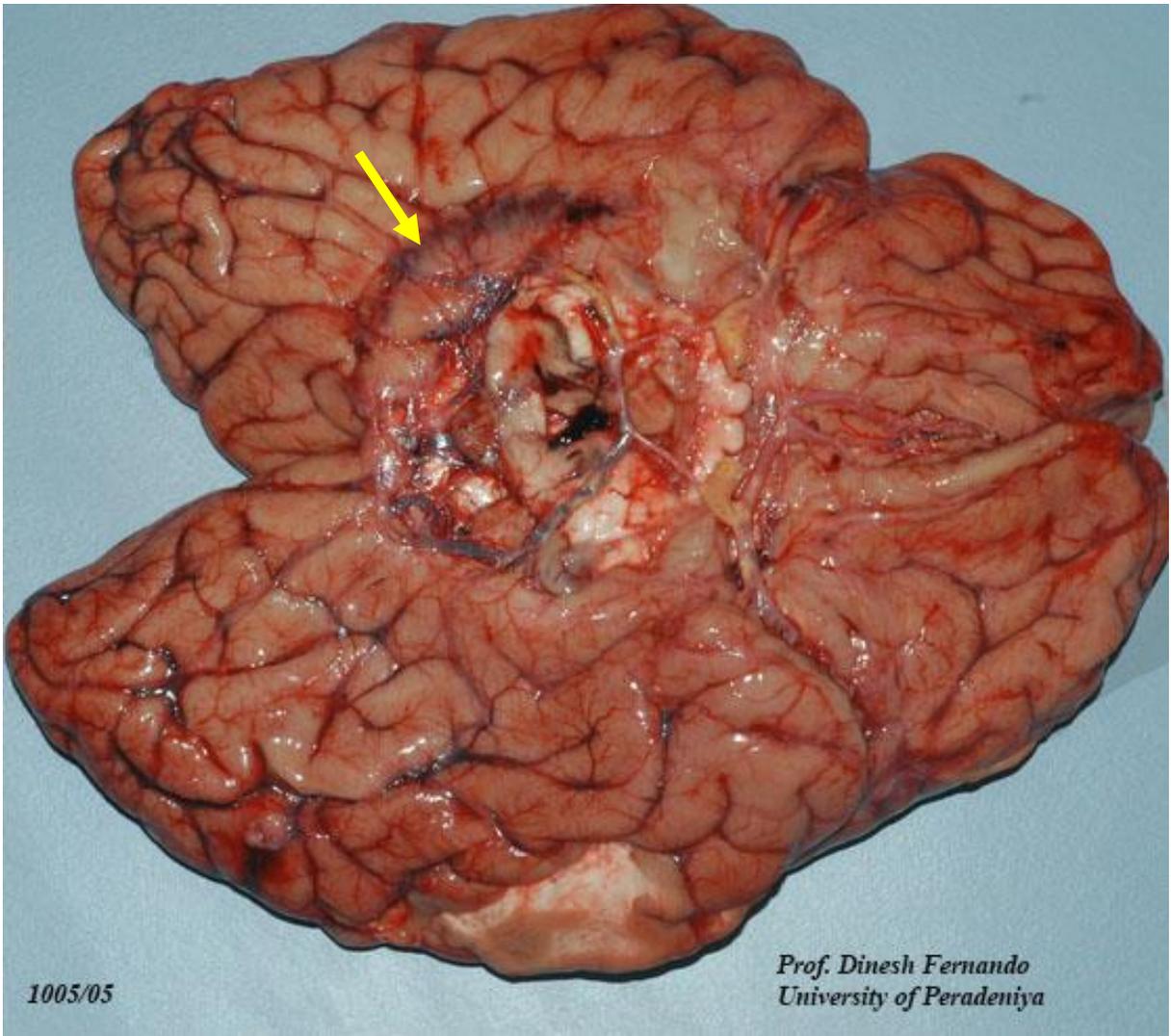


# Herniation



(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 1<sup>st</sup> July 2020)

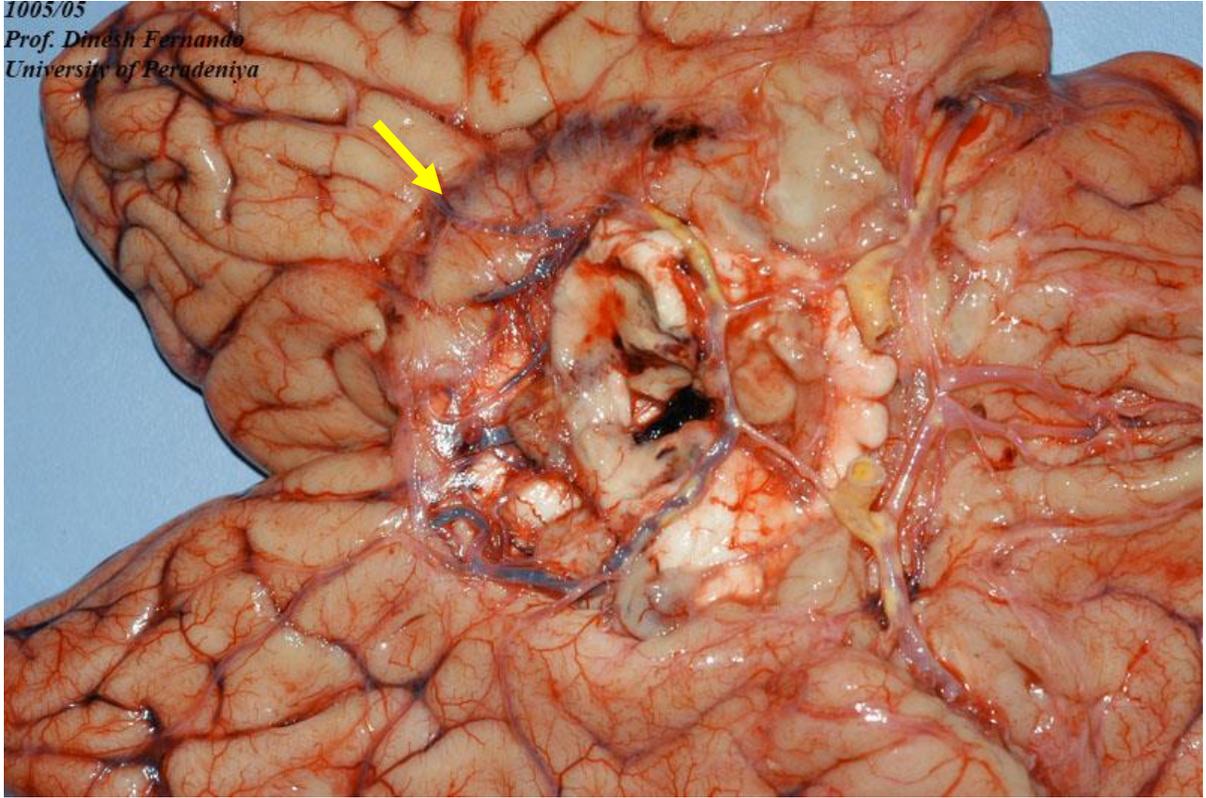
Figure 4: Schematic representation of Kernohan's notch. Demonstrated here are a subdural hematoma and uncal herniation on the same side. Notching of the midbrain is seen on the opposite side (Kernohan's notch). This damages the contralateral pyramidal tract fibres.



(a)



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(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)

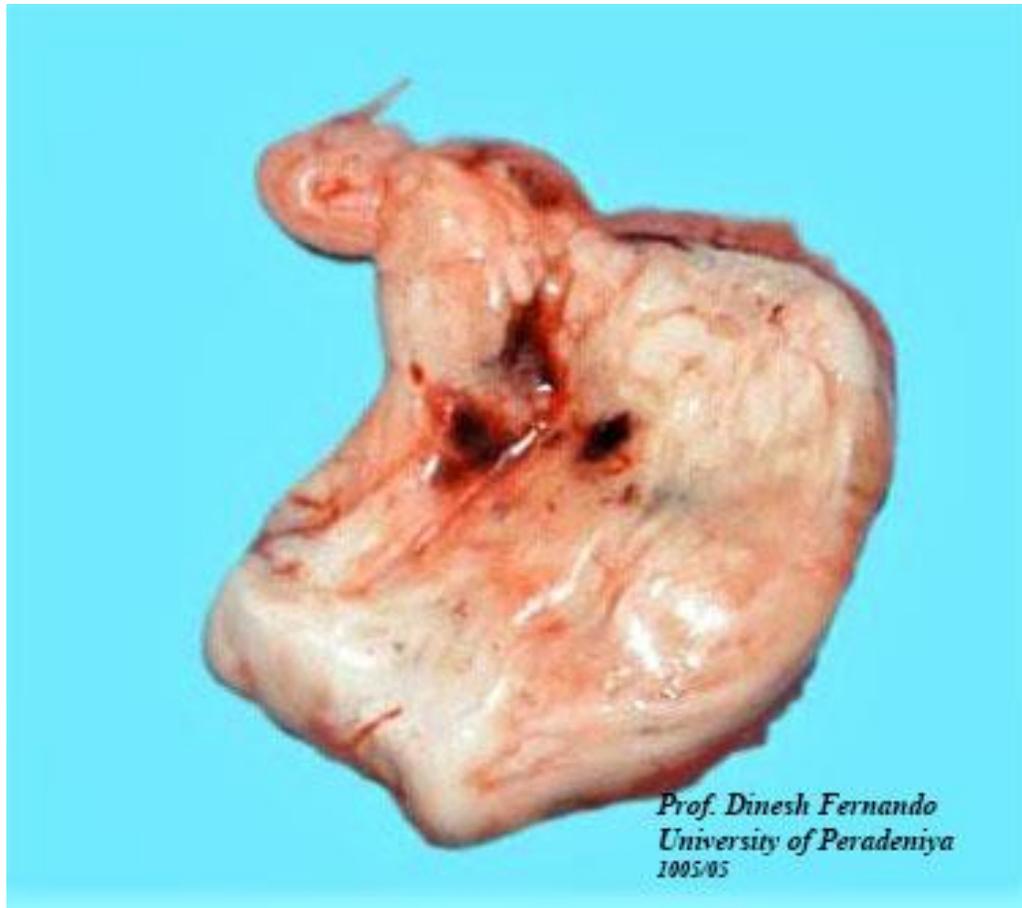


Figure 6: Brain stem haemorrhage



## Microscopic Examination

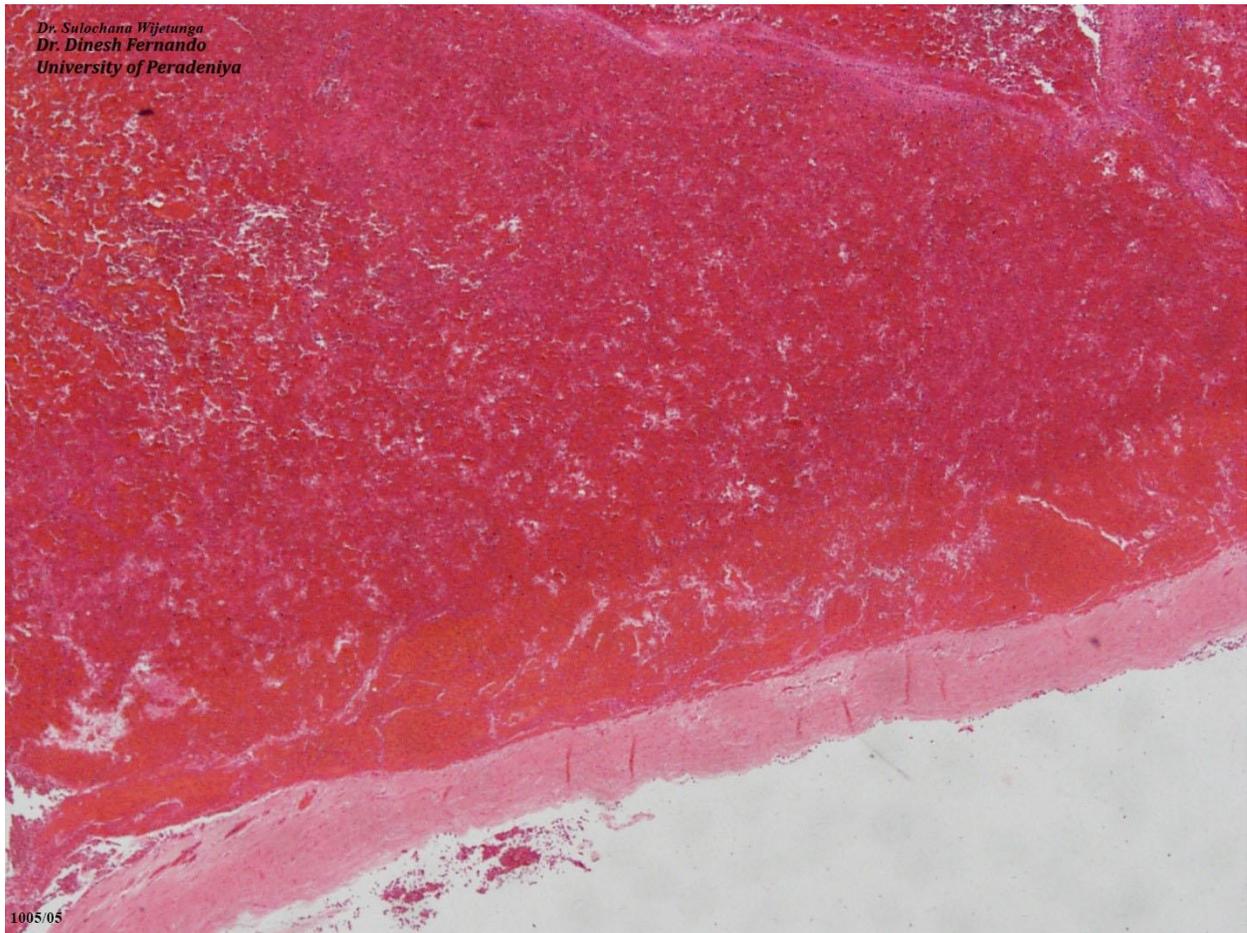


Figure 7: Acute subdural hematoma. Blood collects between the dura and the arachnoid membrane. Sometimes arachnoid membrane may also be torn due to trauma.

## Cause of death

Acute subdural haematoma

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