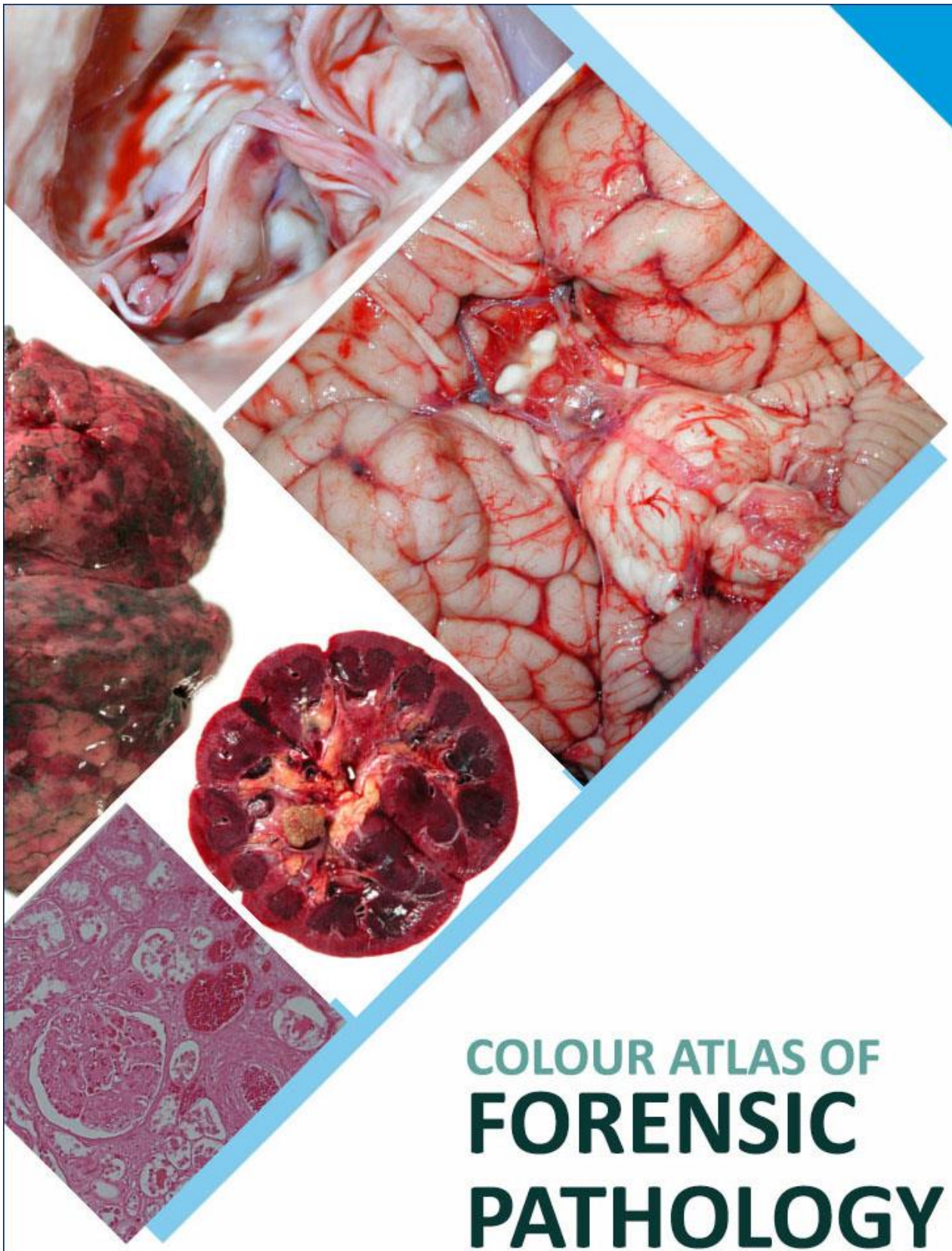


COLOUR ATLAS OF FORENSIC PATHOLOGY

GENITO-URINARY SYSTEM

COLOUR ATLAS OF FORENSIC PATHOLOGY

Version 1

GENITO-URINARY SYSTEM

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

Dr. Deshanee Herath
MBBS
Dept. of Forensic Medicine

Faculty of Medicine,
University of Peradeniya,
Sri Lanka

Copyright reserved by the authors. However, this book can be freely downloaded or reproduced for non-commercial purposes, since it is meant to be used by medical students and post graduate students in forensic medicine and pathology. However, if any images or part of the book is used for educational purposes, due credit should be attributed to the authors.

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

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

Uploaded on 01/04/2022

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. 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. He was elected as a fellow of the College of Forensic Pathologists of Sri Lanka in 2021.

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.

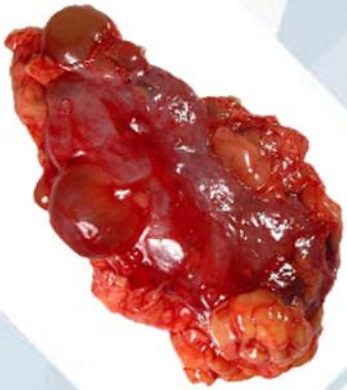
PREFACE

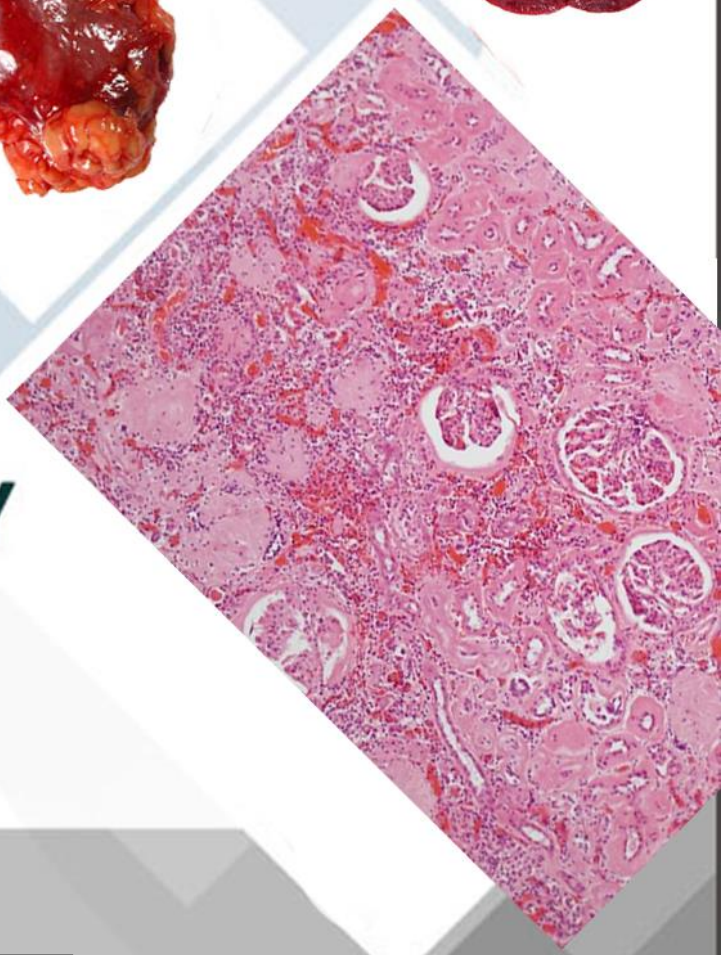
Forensic Medicine in Sri Lanka encompasses, both, examination of patients for medico-legal purposes and conducting autopsies in all unnatural deaths, in addition to those that the cause of death is not known. In the eyes of the justice system in Sri Lanka, all MBBS qualified medical officers are deemed 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. Ms. Chaya Wickramarathne did a yeomen service in the initial designing of lay out and formatting the booklet. The compilation of the case and photographs for publication was done by Dr. Deshanee Herath. This is being continued by Dr. Shashika Weerasinghe.

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

Renal Diseases

RENAL DISEASES

Table of Contents

Renal Diseases

The kidneys are paired retroperitoneal organs, which are located between the transverse processes of the T1-L3 vertebrae. The long axis of each kidney lies parallel to the lateral border of the ipsilateral psoas major. The normal kidney measures about 12x6x3 cm and weighs 130-150 g. The right kidney usually lies in lower position compared to the left kidney, due to the bulk of the right lobe of the liver. The upper pole of the left kidney overlies the eleventh rib, whereas, the upper pole of the right kidney overlies the twelfth rib.

The kidney, covered by its capsule, has a smooth surface. The perinephric fat that lies outside the renal capsule contributes in maintaining the position of the kidney. Perinephric fat is surrounded by the renal fascia (Gerota's fascia), which ascends to cover the suprarenal glands.

The commencement of the ureter, known as the renal pelvis is funnel shaped and is the posterior most structure at the renal hilum. The blood supply to the kidney is via the renal arteries that originate from the abdominal aorta at the level of L2. The kidneys receive approximately 20% of the cardiac output. The usual order of structures in the hilum of each kidney is vein, artery and ureter from front to the back.

The cortex of the kidney is a dark reddish structure that lies beneath the capsule and extends towards the renal pelvis as the renal columns. The darker, triangular, striated areas that lie in between the renal columns are known as renal pyramids, apices of which open into the renal papillae. Each of these papillae opens into a minor calyx. The minor calyces unite to form two or three major calyces which open into the renal pelvis.

The functional and histological unit of the kidney is the nephron, which consists of a glomerulus and a tubular system. This tubular system has 5 parts, namely, Bowman's capsule, proximal convoluted tubule, loop of Henle, distal convoluted tubule and the collecting ducts.

The cortex consists of the glomeruli and convoluted tubules and the medulla contains the loops of Henle and collecting tubules and ducts.

The kidneys serve many functions including, filtration and excretion of metabolic products, regulation of necessary electrolytes, fluid, and acid base balance, and stimulation of red blood cell production.

The renal pathologies include acute and chronic pyelonephritis, chronic kidney disease, renal stones, cysts, tumours and congenital anomalies.

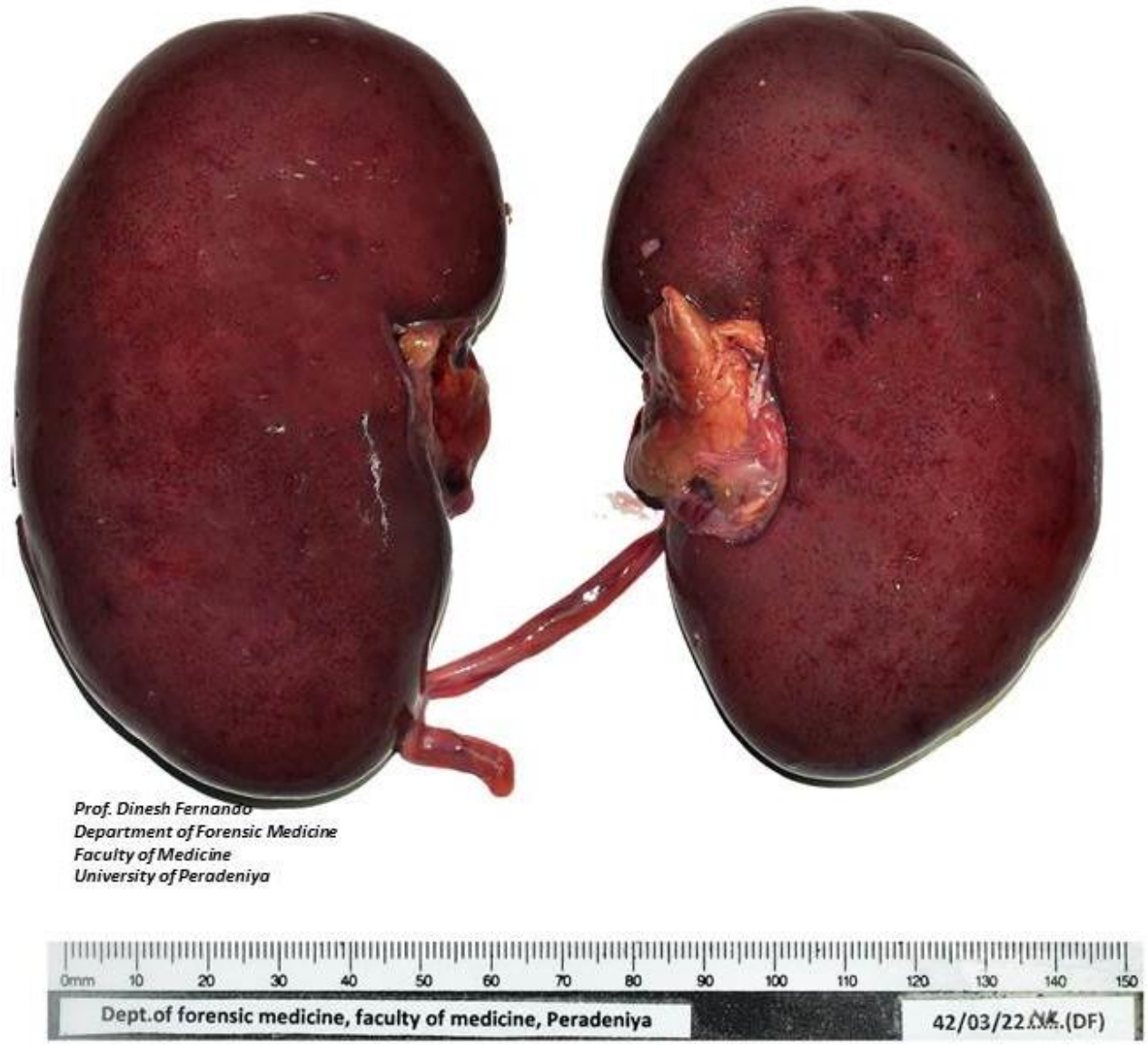
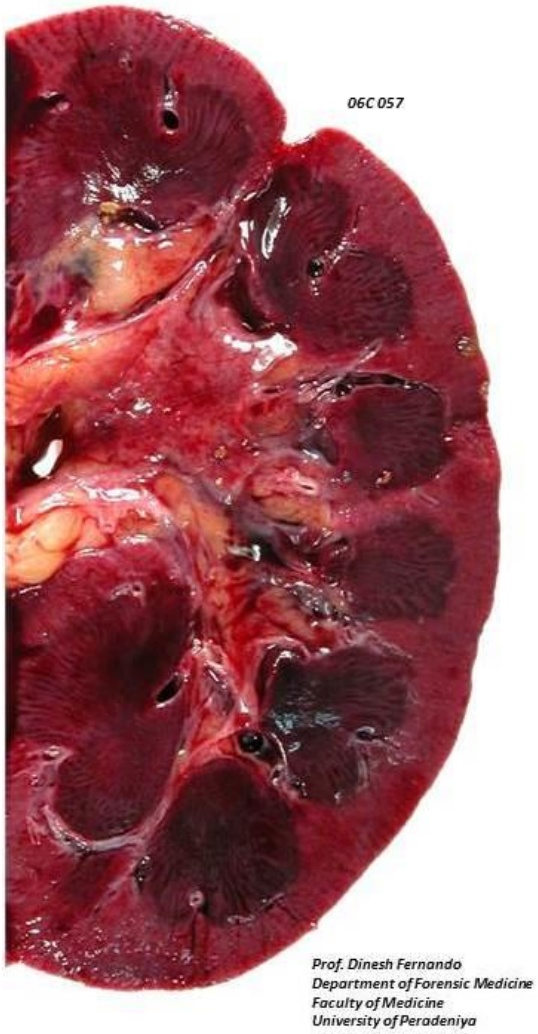


Figure 1: Kidneys external appearance



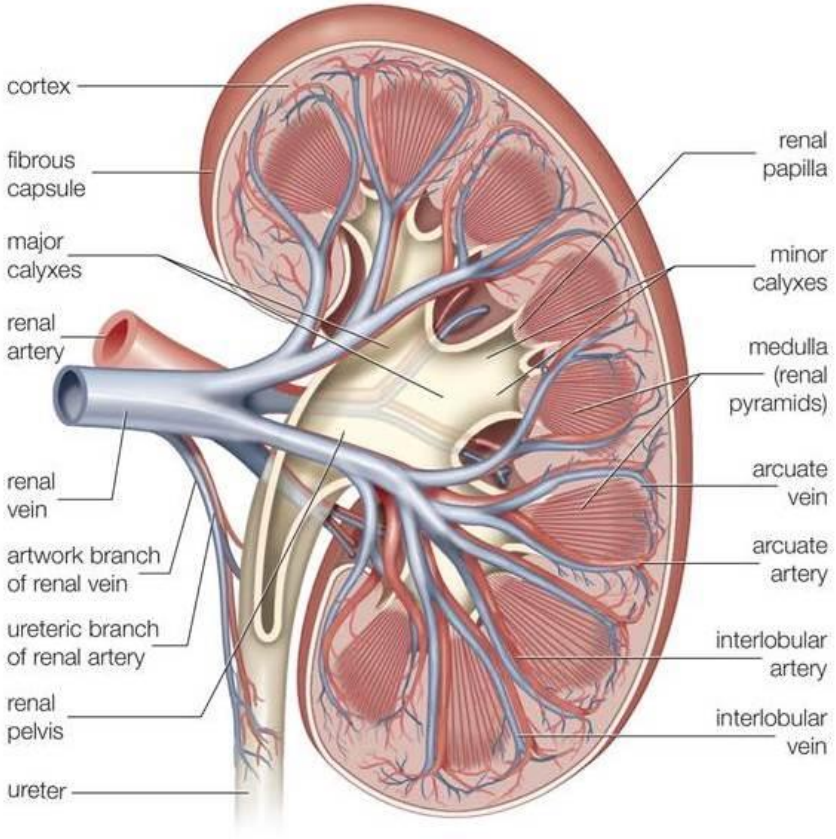


Figure 2: Cut section of a kidney

Acute Pyelonephritis

Acute pyelonephritis is the suppurative inflammation of the kidney and the renal pelvis, caused by bacterial infection. It is a potential life threatening condition that can cause renal scarring. The bacteria can reach the kidney through the bloodstream (haematogenous spread) and the lower urinary tract (ascending infection), latter being the most common route. Acute pyelonephritis is mainly caused by enteric gram-negative rods like *Escherichia coli,* Proteus, Klebsiella, Enterobacter and Pseudomonas. It is commonly seen in patients with predisposing factors like congenital or acquired urinary obstruction, instrumentation of urinary tract, vesicoureteral reflux, pre-existing renal lesions, diabetes and immunodeficiency.

In ascending infections, adhesion and colonization of causative bacteria on the mucosal surface of the distal urethra is followed by contamination of bladder urine. The bacteria ascend along the ureters to infect the renal parenchyma and renal pelvis.

The affected kidney could be normal in size or enlarged. Characteristically, discrete, yellowish, raised abscesses are grossly apparent on renal surfaces. These abscesses can be scattered or coalesce to form a large area of suppuration. Histologically, the renal parenchyma will show liquefactive necrosis with abscess formation. The suppuration that is limited to the renal interstitium initially, can later involve the renal tubules. Presence of large masses of intratubular neutrophils in the collecting ducts will give rise to white cell casts found in the urine. Usually, glomeruli are spared in this condition. If there is a significant obstruction in the urinary tract, the pus may not drain, filling the renal pelvis, calyces and ureters causing pyonephrosis.

Papillary necrosis is a less common form of acute pyelonephritits, which is predisposed by diabetes, urinary tract obstruction and analgesic abuse. This is characterized by well-defined gray-white to yellow necrosis of the apical two thirds of the renal pyramids. There is coagulative necrosis at the tip of the papillae, surrounded by neutrophilic infiltrate.

A patient with acute pyelonephritis can present with sudden onset pain at the costovertebral angle of the affected side, associated with chills, rigors, fever and malaise. The patient can also have localized features of urinary tract infection such as, dysuria, frequency and urgency. Gross haematuria occurs more commonly in young women with pyelonephritis. The diagnosis of the condition is usually clinical, supported by urinalysis, in the outpatient setting. A urine culture is also indicated in patients with pyelonephritis. An episode of acute pyelonephritis can progress into significant renal damage, acute kidney injury, abscess formation, sepsis and multi organ failure.

In the management of the condition, antibiotic therapy is needed to prevent the progression and this is based on the antibiotic sensitivity testing performed along with the urine culture. The patient should be supported with intravenous fluids and pain relief. Surgical intervention maybe needed in renal cortical abscesses, perinephric abscesses and papillary necrosis.

History

A 45-year-old female with a history of gout and urinary incontinence, was found dead in her home.

Internal Examination

Urinary Tract: The right and left kidneys weighed 230 grams and 250 grams respectively. The renal capsules stripped easily to reveal granular scarred cortical surfaces. The renal parenchyma had a thin cortex with normal cortical medullary demarcation. Areas of scarring were present. Streaky appearances to the pyramids were noted. The calyces, pelvis, ureters and bladder were unremarkable. The bladder was empty.

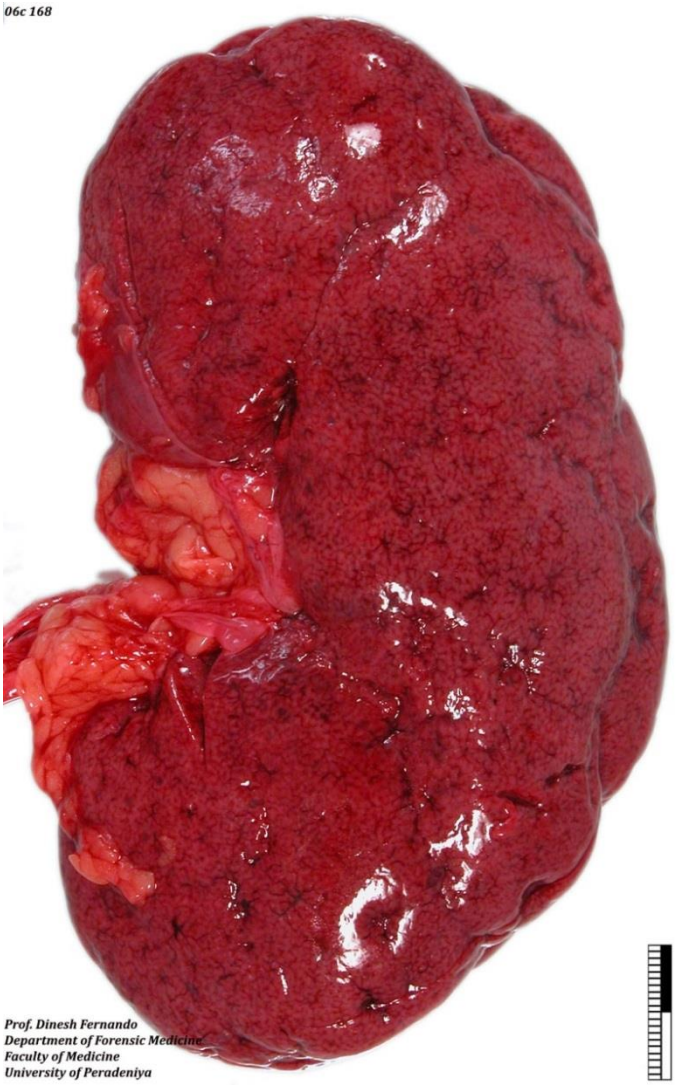


Figure 3: Granular scarred cortical surfaces

Microscopic Examination

Genitourinary system: Sections of kidneys showed acute on chronic pyelonephritis. Hyaline is present in the vessels.

In order to demonstrate acute pyelonephritis, the following histological images are taken from another case.

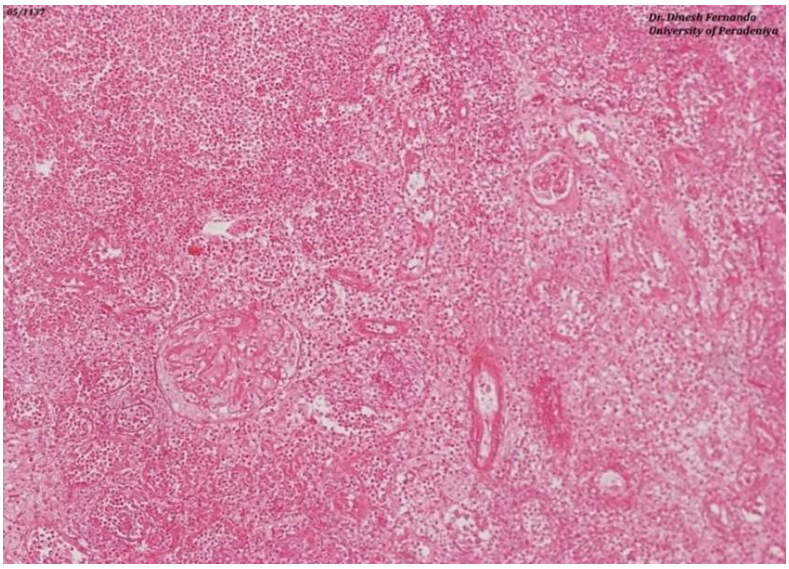


Figure 4: Renal parenchyma in acute pyelonephritis (low power)

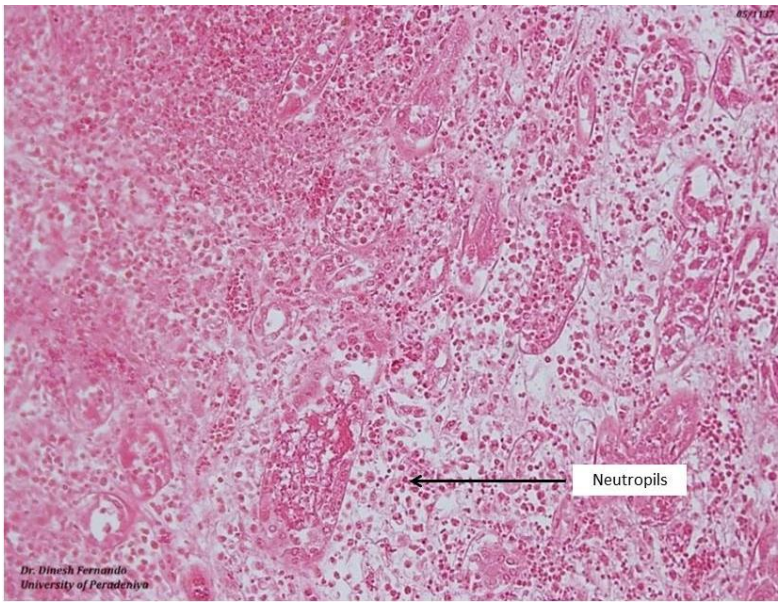


Figure 5: Note the neutrophil infiltration (arrow) of the renal parenchyma (high power)

Chronic Pyelonephritis

Chronic pyelonephritis, an important cause of chronic renal failure, is a form of renal interstitial inflammation and scarring of the renal parenchyma. This is associated with grossly visible scarring and deformity of the pelvicalyceal system. It can be divided into two forms, namely, chronic obstructive pyelonephritis and chronic reflux associated pyelonephritis. In chronic obstructive pyelonephritis, diffuse or localized obstructive lesions associated with recurrent infections lead to recurrent episodes of renal inflammation and scarring. Chronic reflux associated pyelonephritis (reflux nephropathy), is more common and occurs when urinary tract infection is associated with congenital vesicoureteral reflux and intrarenal reflux. This can affect one or both kidneys, eventually leading to chronic renal insufficiency.

The involvement of renal parenchyma could be diffuse or patchy. This leads to uneven scarring of the kidneys, making it easier to distinguish from the more symmetrically contracted kidneys that are formed in vascular sclerosis and chronic glomerulonephritis. The factors that affect the pathogenesis of chronic pyelonephritis include sex of the patient, pregnancy, genetic factors, bacterial virulence factors and neurogenic bladder dysfunction. The hallmark of chronic pyelonephritis is scarring involving the pelvis or calyces, or both, leading to papillary blunting and marked calyceal deformities.

Microscopically, the findings are nonspecific. There may be uneven interstitial fibrosis associated with inflammatory infiltrate of lymphocytes, plasma cells and neutrophils. The renal tubules can be dilated or contracted, with atrophy of the lining epithelium. These dilated tubules can contain colloid casts, which give the tubules an appearance similar to thyroid tissue. The calyceal mucosa and the wall will be fibrosed and will have chronic inflammatory cell infiltrate. The condition can be frequently associated with hypertension that can cause arteriolosclerosis. Nephron loss caused by the infection can lead to secondary glomerulosclerosis.

Patients with chronic pyelonephritis present relatively late, due to the gradual onset of the renal insufficiency. They can present with abnormal renal function results in routine laboratory investigations or with the development of hypertension. Radiologically it is characterized by asymmetrically contracted affected kidney and blunting and deformity of calyceal system. The disease can manifest as polyuria and nocturia, if the disease is bilateral and progressive causing tubular dysfunction. Some patients may develop secondary glomerulosclerosis leading to proteinuria. Other complications of chronic pyelonephritis include xanthogranulomatous pyelonephritis and pyonephrosis. Urinalysis may show pyuria and the urine culture may isolate gram negative bacteria like *Escherichia coli* and Proteus. Serum creatinine and blood urea levels are elevated in this condition. Management of the condition can be achieved by continuous antibiotic prophylaxis and surgically relieving the obstruction or correcting the reflux. Progressive renal injury can be prevented by dietary protein restriction and blood pressure control.

Microscopic Examination

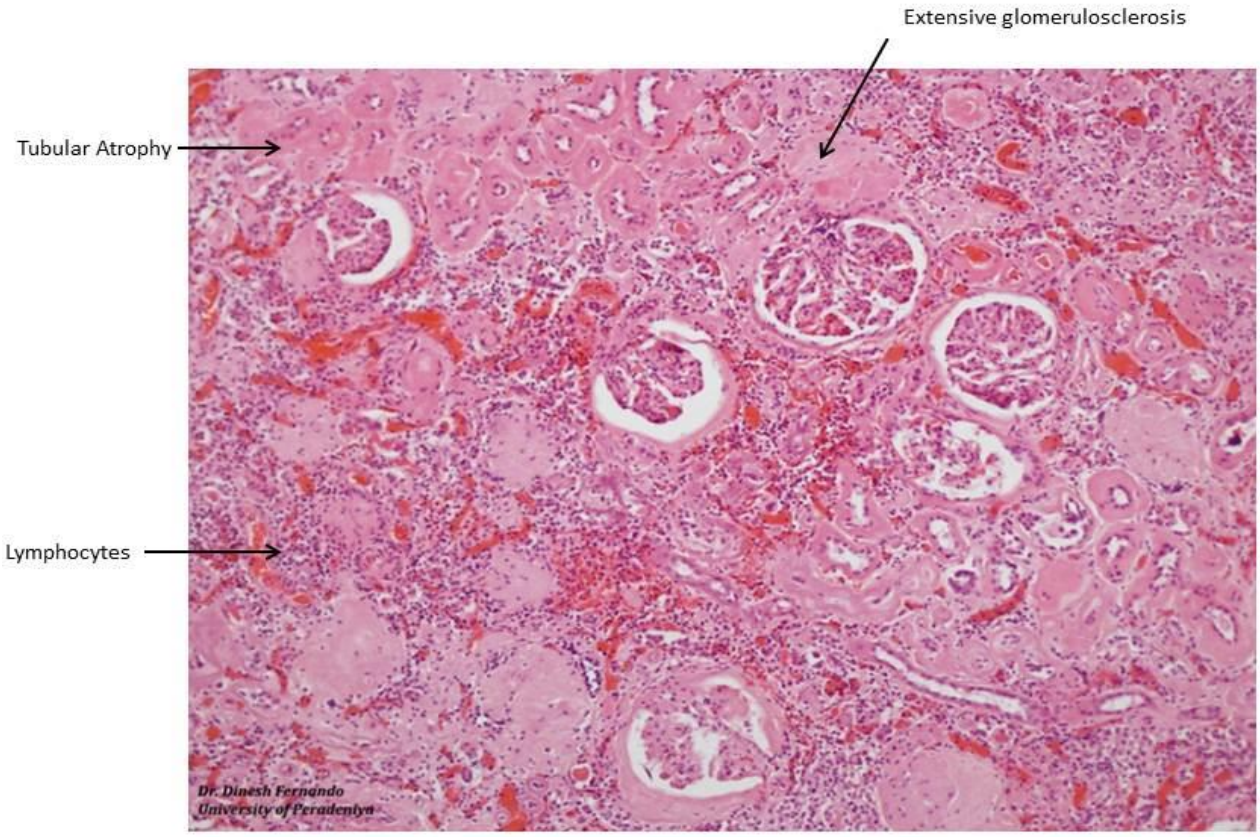


Figure 6: Note the tubular atrophy and extensive glomerulosclerosis of the renal parenchyma

Diabetic Nephropathy

Diabetic nephropathy is a part of diabetic macrovascular disease. Diabetes mellitus exerts a damaging effect on the kidneys, at the level of glomeruli and the microcirculation. Three lesions are encountered in diabetic nephropathy, namely, glomerular lesions, renal vascular lesions and pyelonephritis. The most important glomerular lesions are capillary basement membrane thickening, diffuse mesangial sclerosis and nodular glomerular sclerosis. A diffuse increase in mesangial matrix associated with mesangial cell proliferation can be seen in diffuse mesangial sclerosis. It is directly induced by hyperglycaemia which leads to glycosylation of matrix proteins. It is observed in patients with disease of more than 10 years duration and it can progress into nephrotic syndrome which is characterized by proteinuria, hypoalbuminemia and oedema.

Nodular glomerular sclerosis is characterized by a glomerular lesion made by ball-like deposits of laminated matrix located in the periphery of the glomerulus. These nodules contain trapped mesangial cells and are known as Kimmelstiel-Wilson lesions. This is a major contributor to morbidity and mortality. Glomerular sclerosis leads to ischaemia which causes significant scarring which is seen as a finely granular cortical surface of the kidneys.

Renal vascular disease consists of renal atherosclerosis and arteriolosclerosis. Both acute and chronic forms of pyelonephritis are more common in patients with diabetes, compared to the general population. Necrotizing papillitis, a specific form of acute pyelonephritis is commonly seen in patients with diabetes.

Even though the exact cause of diabetic nephropathy is not known, hyperglycaemia induced hyperfiltration and renal injury, advanced glycation products and activation of cytokines are considered to be mechanisms responsible for the development of the condition.

Clinically, diabetic nephropathy is characterized by persistent albuminuria, progressive decline in glomerular filtration rate and elevated blood pressure. The patient may present with passage of foamy urine, unexplained proteinuria, fatigue, oedema and associated diabetic retinopathy and peripheral vascular occlusive disease. Natural history of the condition consists of five stages, namely, hyperfunction and hypertrophy, silent stage, incipient stage, overt diabetic nephropathy, and uraemia. End stage renal disease is the major cause of death in patients with diabetic nephropathy.

In the diagnosis of diabetic nephropathy, 24-hour urinalysis for urea, creatinine and protein, blood tests for estimation of glomerular filtration rate, renal ultrasound and renal biopsy are used.

The management of the condition include, glycaemic control, management of hypertension and reducing salt intake. Renal replacement therapy may be needed in patients with end stage renal disease.

Microscopic Examination

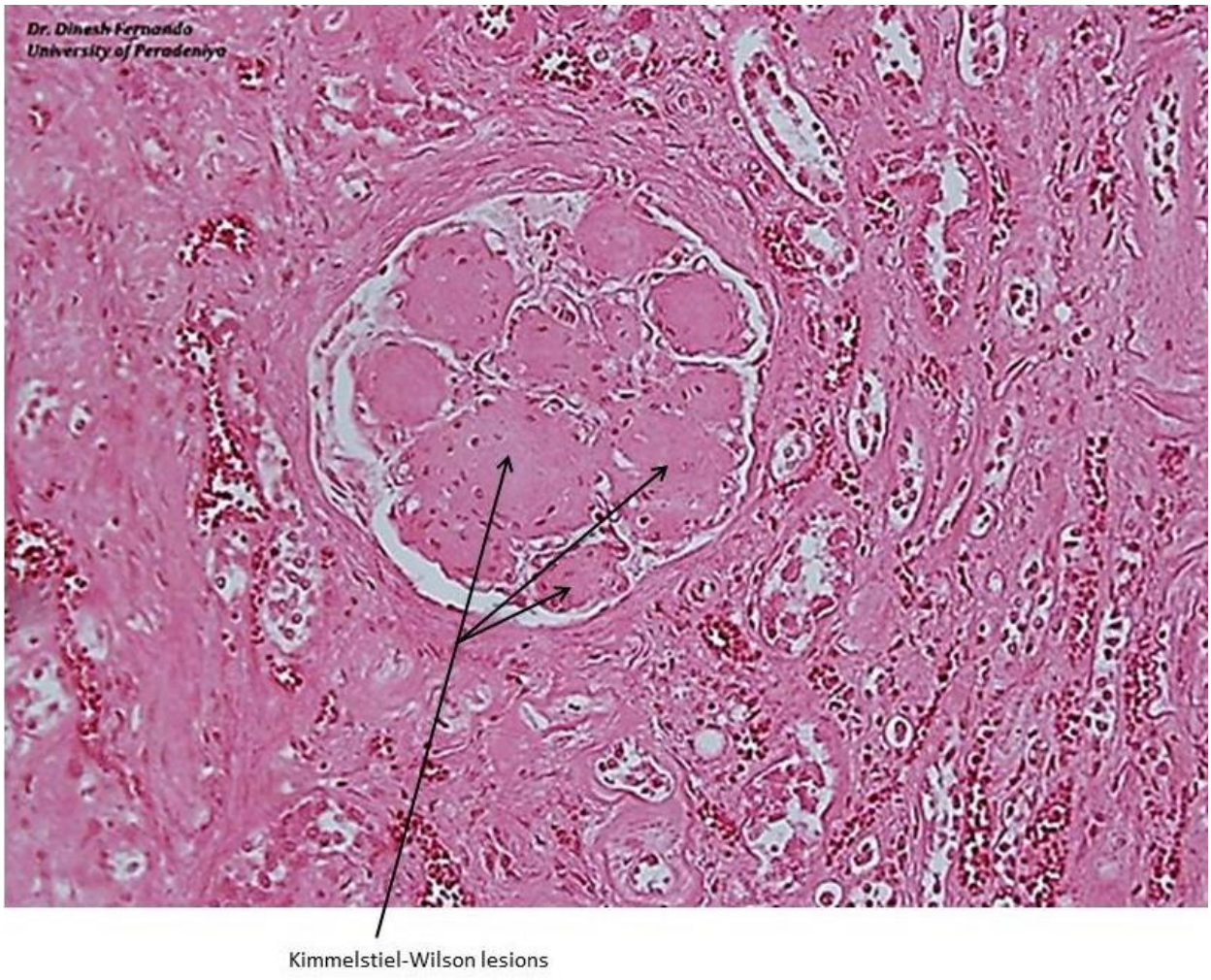


Figure 7: Kimmelstiel-Wilson lesions in the renal parenchyma

Chronic Kidney Disease

Chronic kidney disease (CKD) is the longstanding (>3 months), progressive impairment of the renal function due to any underlying cause. These underlying renal diseases lead to progressive renal scarring, that eventually results in sclerosis of glomeruli, tubules, interstitium and blood vessels. CKD is commonly seen in the elderly population and is associated with increased risk of cardiovascular disease and end stage renal disease. It is defined as either kidney damage or reduced glomerular filtration rate of less than 60 ml/min/1.73m^2. It is divided into 5 stages based on the glomerular filtration rate.

Presence of albuminuria, urine sediment abnormalities, electrolyte abnormalities, histological abnormalities, structural abnormalities detected by imaging, and history of renal transplantation establish the diagnosis of chronic kidney disease.

In renal injury due to any cause, the kidneys maintain the glomerular filtration rate, despite the progressive destruction of nephron, by hyperfiltration and compensatory hypertrophy. Therefore, the plasma level of the waste products like urea and creatinine, will show significant increment only after total glomerular filtration rate is decreased by 50%. The increased glomerular capillary pressure can damage the capillaries, causing secondary focal and segmental glomerulosclerosis and eventually lead to global glomerulosclerosis.

Chronic kidney disease may be caused by systemic hypertension, nephrotoxins, reduced perfusion due to severe dehydration or shock, proteinuria, hyperlipidaemia, hyperphosphatemia, smoking and uncontrolled diabetes. The kidneys are symmetrically contracted in chronic kidney disease. The surfaces of the kidneys appear red brown in colour and diffusely granular, since the underlying the disease may involve the blood vessels and glomeruli. However, if the underlying cause for the chronic kidney disease is chronic pyelonephritis, the kidneys are unevenly involved and can show deep scars.

Histologically the renal parenchyma shows advanced scarring of the glomeruli, which may have progressed into complete sclerosis of the renal parenchyma. It may also show marked interstitial fibrosis, associated with atrophy of the tubules in the cortex and diminution and destruction of parts of the peritubular capillary network. Vascular involvement in chronic kidney disease may appear in the form of thick walled small and medium sized arteries with narrowed lumina, caused by secondary hypertension. The fibrotic interstitial tissue may be infiltrated with lymphocytes, and rarely, with plasma cells. The markedly damaged kidneys in end stage renal disease, may not show any evidence to distinguish whether the primary lesion was glomerular, vascular, tubular, or interstitial.

Clinically, CKD is discovered later in its course, after the onset of the renal insufficiency. The proteinuria, hypertension and azotaemia discovered incidentally, may reveal the renal disease. Complications of chronic kidney disease include, anaemia, mineral bone disorder, skin, gastrointestinal and endocrine abnormalities and muscle dysfunction.

The management of CKD include, general measures like treating the underlying condition, addressing the cardiovascular risk factors and systematic follow up. It may also require renal replacement therapy.

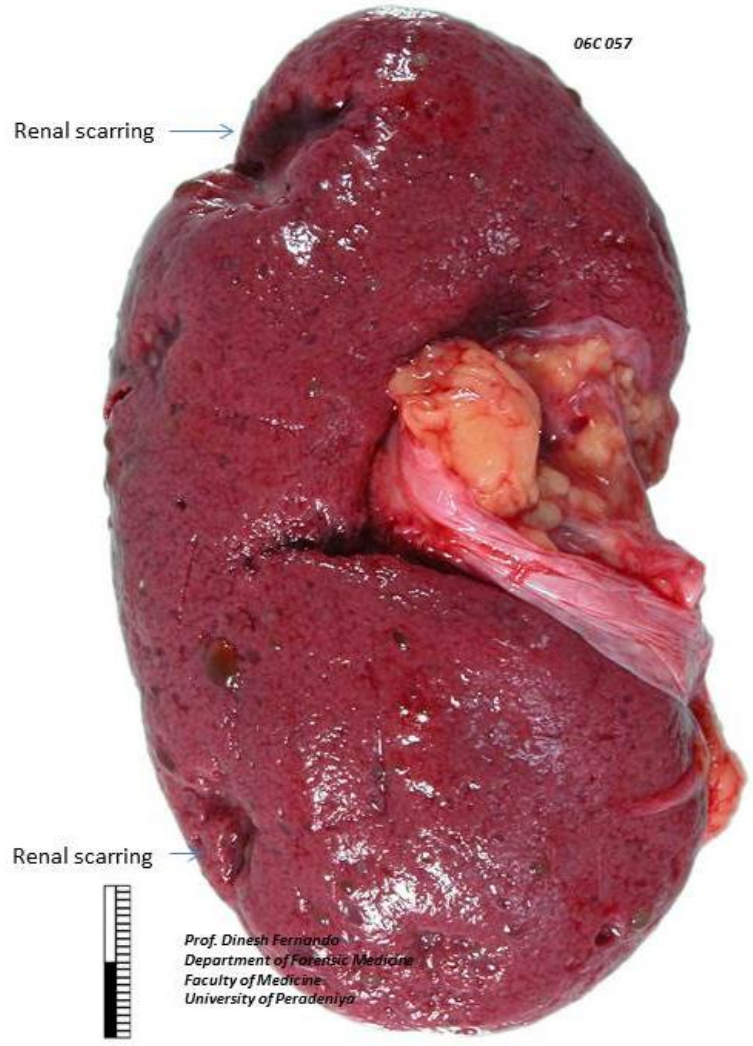


Figure 8: Renal scarring

History

In a 79-year-old female, who died of peritonitis, granular contracted kidneys were found incidentally.

Internal Examination

Urinary Tract: The right and left kidneys weighed 94 grams and 78 grams respectively. The renal capsules stripped easily to reveal granular contracted kidneys. The renal parenchyma revealed a pale cortex and an indistinct cortical medullary junction.

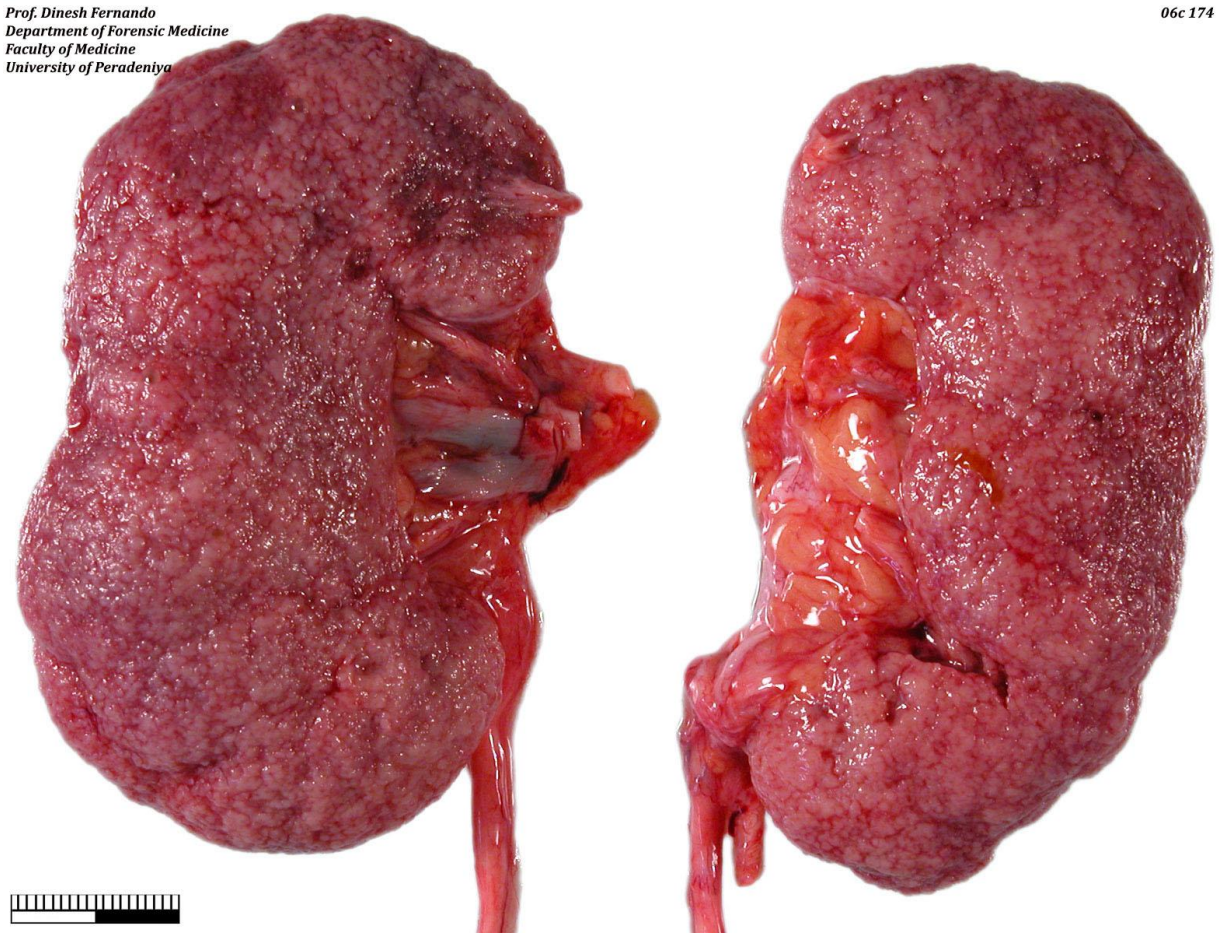


Figure 9: Granular contracted kidneys

Cystic Renal Disease

Cystic renal diseases comprise of hereditary, developmental and acquired diseases. Cystic renal disease consists of simple renal cysts, autosomal dominant polycystic kidney disease, autosomal recessive polycystic kidney disease, and medullary disease with cysts. Simple renal cysts can occur as single or multiple cystic spaces of variable sizes. Usually, they are 1 – 5 cm in diameter, translucent, and are filled with clear fluid. Microscopically, these are lined with membranes composed of a single layer of cuboidal or flattened cuboidal epithelium. These cysts are usually confined to the cortex of the kidneys. Rarely, there can be massive cysts that measure 10 cm in diameter. Simple cysts are a common post-mortem finding with no clinical significance. They become clinically important when they have to be distinguished from renal tumours. Patients with end stage renal disease who have undergone prolonged dialysis can develop dialysis associated acquired cysts. They are present both in the cortex and medulla, and can cause haematuria.

Autosomal dominant (adult) polycystic kidney disease is characterized by multiple expanding cysts that affect both kidneys. They subsequently destroy the intervening renal parenchyma leading to chronic kidney disease. The kidneys may become massively enlarged, manifesting as an abdominally palpable mass extending into the pelvis, on clinical examination. On macroscopic examination, the kidneys consist of a mass of cysts with no intervening parenchyma. These cysts maybe filled with clear, turbid or haemorrhagic fluid. The cysts can have an atrophic lining. There can be associated hypertension induced changes in the renal vessels. Some patients can have associated hepatic cysts. These patients can present with flank pain that may have been caused by acute distension of a cyst, either by intracystic haemorrhage or by obstruction. This condition can also cause intermittent haematuria, hypertension and urinary infection. It can slowly progress into end stage renal disease around 50 years of age. Death usually results from uraemia or hypertensive complications.

Autosomal recessive (Childhood) polycystic renal disease is a rare form of cystic renal disease. In this condition, there can be small multiple cysts in the cortex and medulla, giving the kidneys a sponge like appearance. The cysts are lined with a uniform layer of cuboidal cells that may have originated from the collecting tubules. Both the kidneys are invariably affected in this disease. It is also associated with hepatic cysts and proliferation of portal bile ducts.

Medullary disease with cysts consists of medullary sponge kidney and nephronophthisis-medullary cystic disease complex. There will be small contracted kidneys which have multiple small cysts lined by flattened or cuboidal epithelium at the corticomedullary junction. Other pathological changes include chronic tubulointerstitial nephritis with tubular atrophy and thickened tubular basement membranes and progressive interstitial fibrosis. Patients present with polyuria and polydipsia as a consequence of diminished tubular function. This condition can progress into end stage kidney disease over 5 to 10 years.

History

A 79-year-old male with coronary artery disease, presented with diarrhoea and haematemesis. His condition deteriorated, leading to death.

Internal Examination

Urinary Tract: The right and left kidneys weighed 140 grams and 160 grams respectively. The renal capsules stripped easily to reveal granular cortical surfaces with a clear fluid filled cyst on the right kidney. The renal parenchyma of the left kidney revealed a thin cortex with normal cortical medullary demarcation. The right kidney had a fluid filled cyst which had caused extreme thinning of the cortex and dilatation of the pelvicalyceal system.

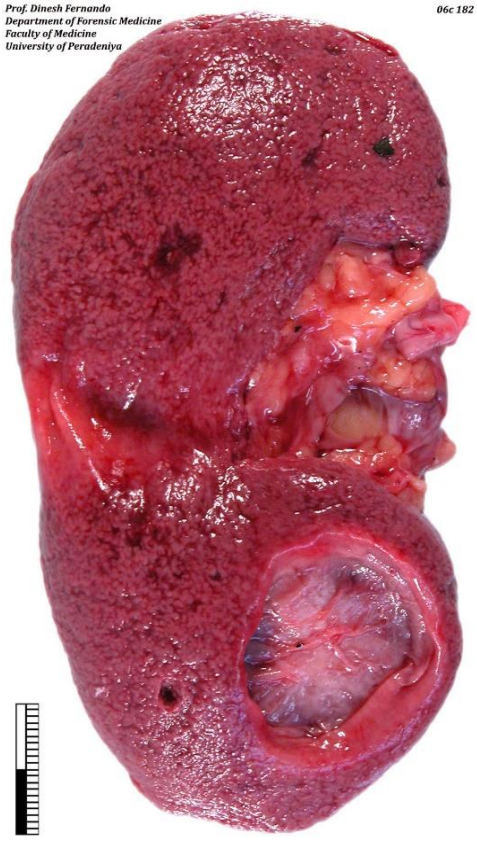


Figure 10: Right kidney - granular cortical surfaces with a clear fluid filled cyst

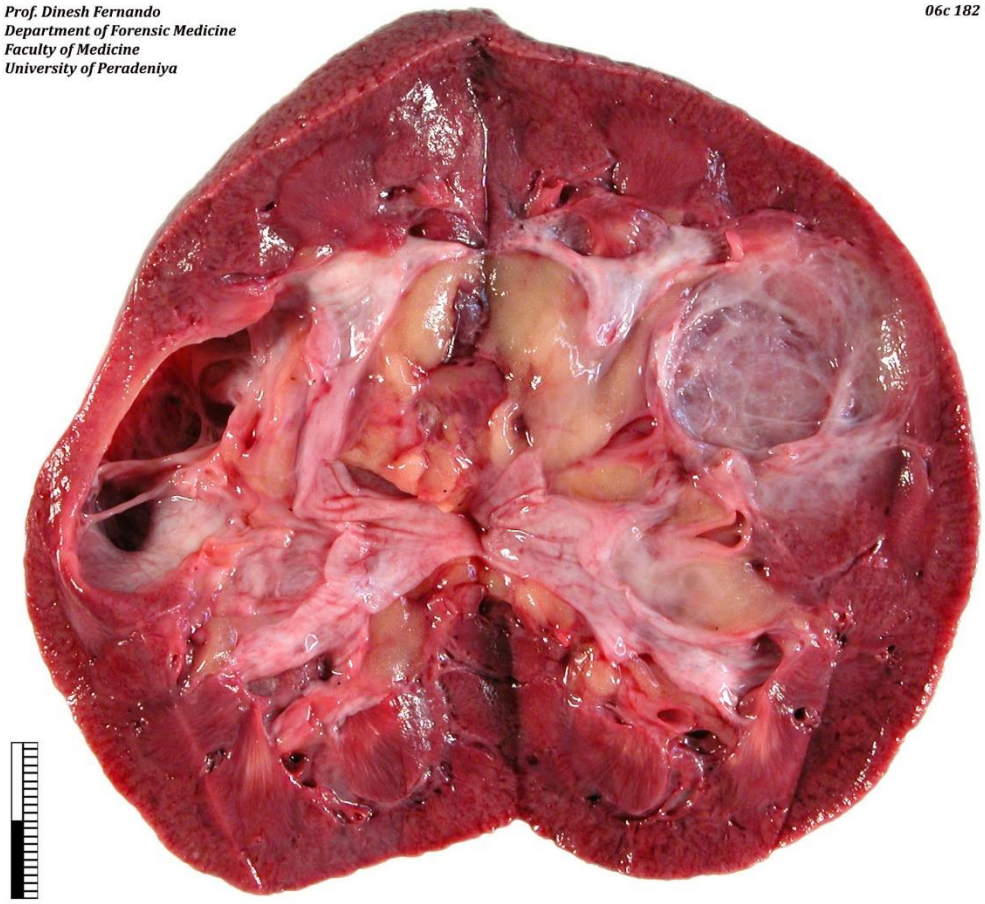


Figure 11: Cross section of the right kidney; note the fluid filled cyst which had caused extreme thinning of the cortex and dilatation of the pelvicalyceal system.

In order to demonstrate cystic renal diseases, the following image is taken from another case.

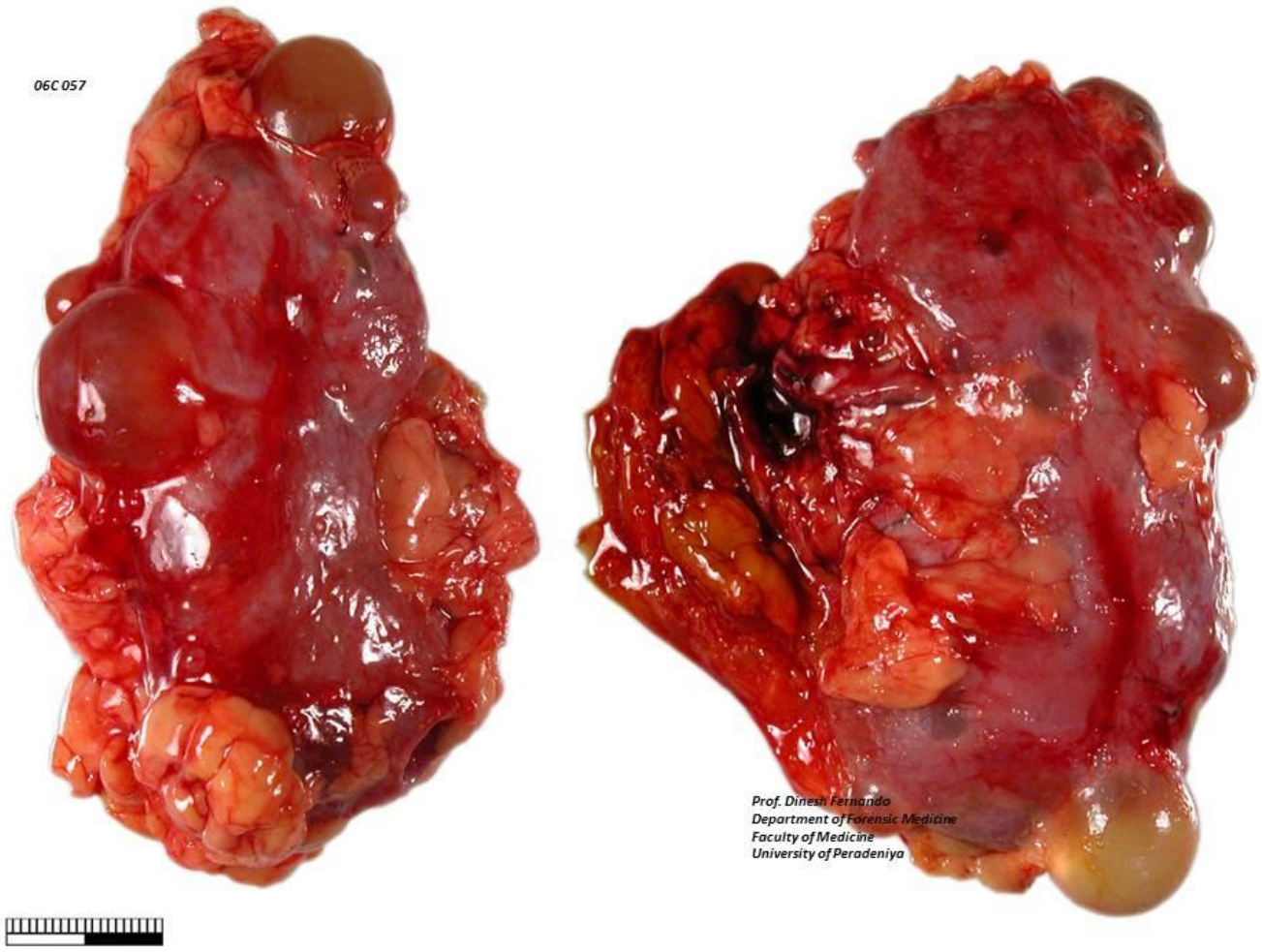


Figure 12: Cystic kidney disease with fluid filled cysts.

Renal stones (Nephrolithiasis)

Nephrolithiasis is defined as calculi in the kidney. Urolithiasis is calculus formation at any level in the urinary collecting system. Symptomatic urolithiasis is more common in males than females. The formation of urinary tract stones can be explained by two major phenomena. The first method is supersaturation of the urine by stone forming constituents, including calcium, oxalate, and uric acid. This can be caused by low fluid intake leading to production of low volume of urine. The ion from the supersaturated urine form microscopic crystalline structures, on the crystals and foreign bodies that act as nidi.

Majority (80%) of renal calculi contain calcium in the form of calcium oxalate or calcium oxalate mixed with calcium phosphate. Ten percent of renal stones are composed of magnesium ammonium phosphate, while 6-9% are either uric acid or cystine stones. The majority of calcium containing renal stones are caused by hypercalciuria without hypercalcaemia. Struvite stones, which are also known as magnesium ammonium phosphate stones, occur in patients with urinary tract infections with urea-splitting bacteria like *proteus vulgaris* and staphylococci leading to alkaline urine. High uric acid level in the blood caused by diseases involving high cell turn over like gout, can lead to production of uric acid stones.

In majority of the patients, renal stones are unilateral. These stones could be smooth or jagged. When the magnesium ammonium phosphate stones take the shape of the branching renal calyces, it is known as a staghorn calculus. These are relatively asymptomatic, but can cause urinary tract infection and haematuria.

A patient with renal calculi can be asymptomatic, or can present with symptoms and renal damage. Large renal stones can get lodged in the renal pelvis. The smaller stones can pass into the ureter, causing a colicky type pain that usually begins in the upper lateral mid back over the costovertebral angle (loin) which radiates towards the groin. Renal colic pain is caused by acute distension, stretching and spasm caused by acute ureteric obstruction. They can also present with haematuria, urinary frequency, urgency, dysuria and suprapubic pain.

Complications of urinary tract stones include, abscess formation, severe pyelonephritis that diminishes renal function, urosepsis, and urinary fistula formation.

The management of renal stones consists of emergency management of renal colic and medical therapy for stone disease. The size of the stone is important in deciding the management option, since it is an important predictor of spontaneous passage. Surgical management options include stent placement, percutaneous nephrostomy, extracorporeal shockwave lithotripsy, ureteroscopy and stone removal and percutaneous nephrostolithotomy.

History

A 46-year old male, a diagnosed patient with dilated cardiomyopathy, end stage renal failure, gout and diabetes mellitus presented with septic arthritis. He had been undergoing dialysis.

Internal Examination

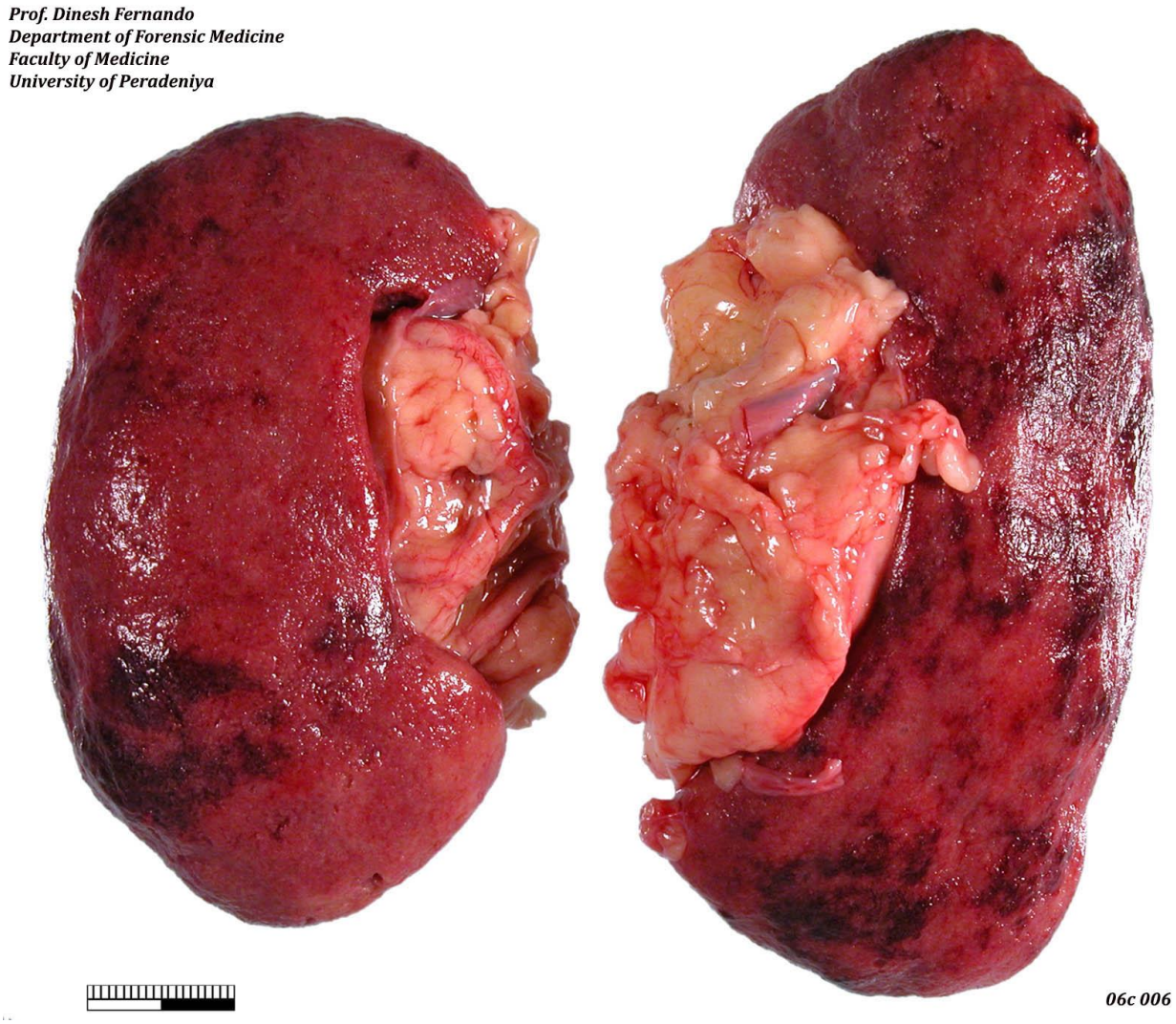


Figure 13: Cortical surfaces of both kidneys; note the black and red colour patches

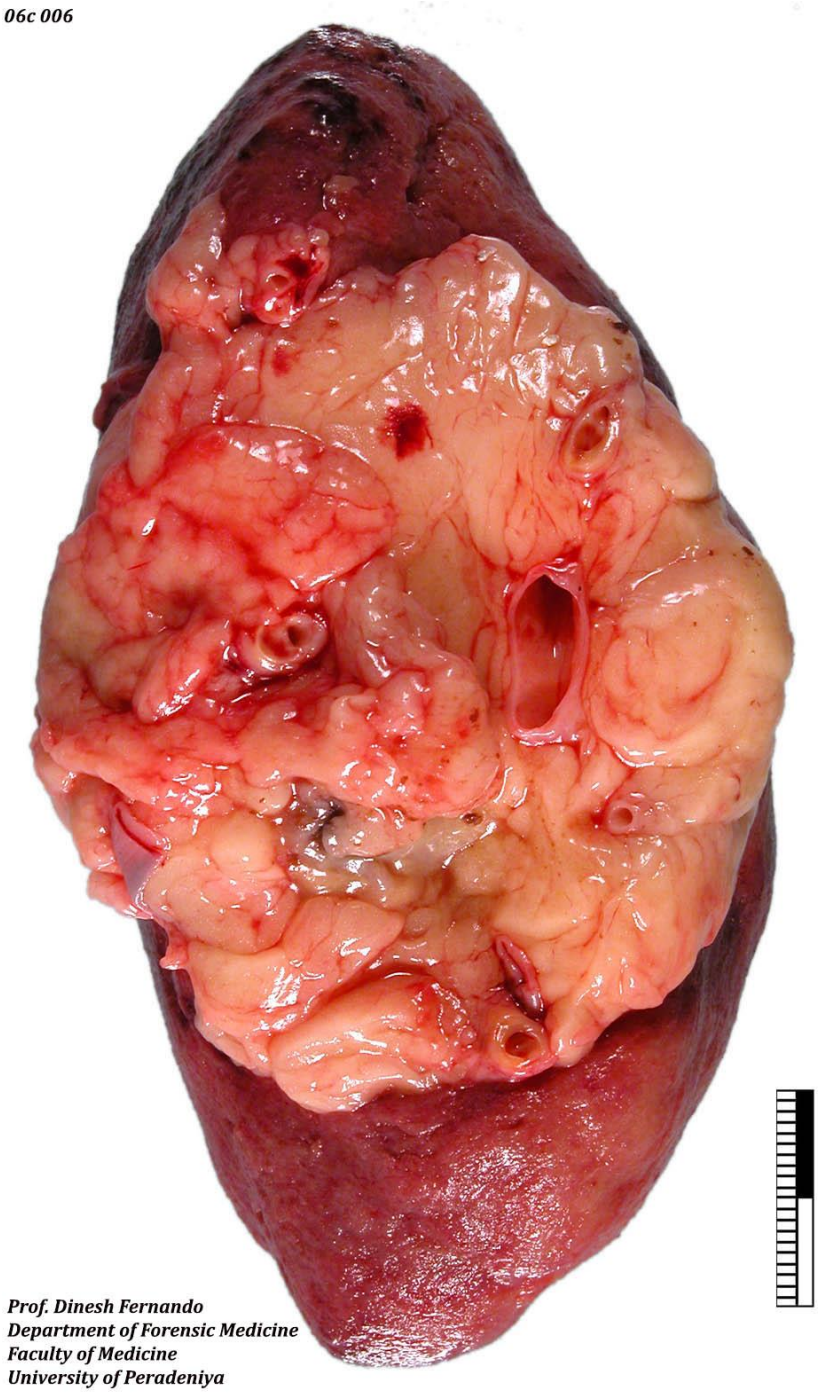


Figure 14: Significant renal artery stenosis

The right renal artery had significant stenosis at its origin while the left renal artery had significant stenosis distally. The distal renal artery divided into multiple branches and supplied the kidney not only at the hilum but at multiple sites

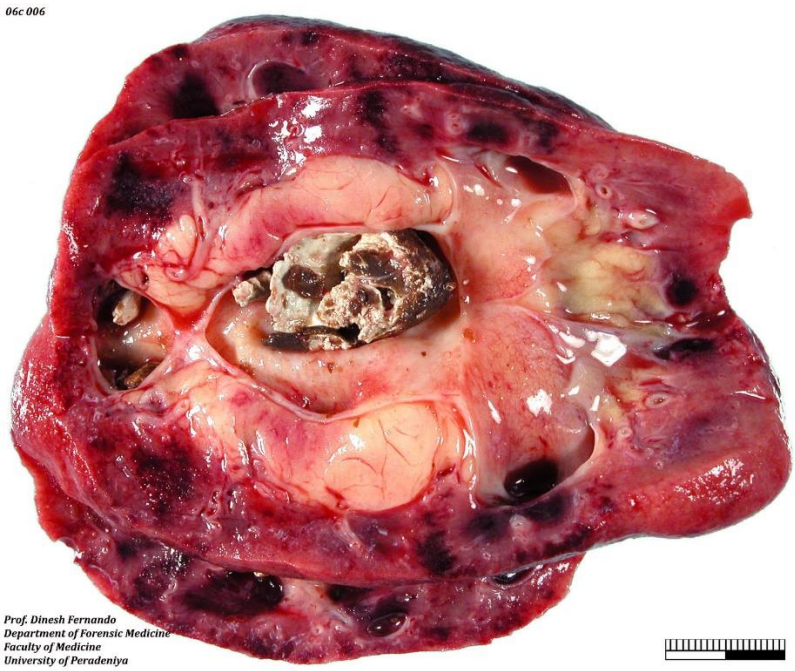


Figure 15: Cut section of the left kidney; note the calculi in the pelvicalyceal system.

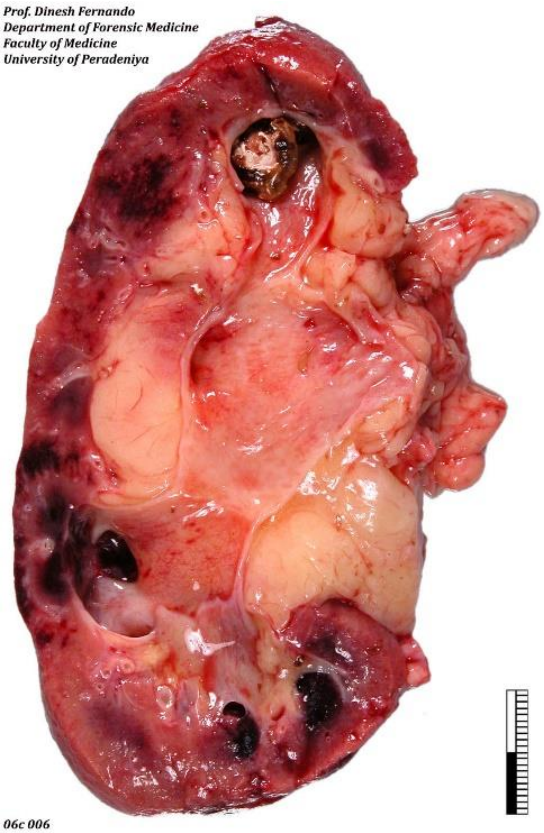


Figure 16: Very thin cortex and markedly dilated pelvicalyceal system of the left kidney

Microscopic Examination

Sections of the kidney showed severe cortical sclerosis with interstitial haemorrhage in medulla and cortex. Arterioles showed medial hypertrophy with thickened intima associated with fibrosis. No pyelonephritis was observed.

Cause of death

Multi organ failure caused by septicaemia.

History

A 59-year old female, with a history of hypertension, hypothyroidism and cholecystectomy presented with shortness of breath, orthopnoea and paroxysmal nocturnal dyspnoea of four weeks duration. Her condition continued to deteriorate, leading to death.

Internal Examination

The right and left kidneys weighed 220 grams and 22 grams respectively. The renal capsules stripped easily. A clear fluid-filled cyst measuring approximately 4 x 5 cm was present on the cortex of the left kidney. Scarring was present in the lower pole of the right kidney. A square-shaped calculus which had a rough surface measuring approximately 1.5 x 1.5 cm was present in the pelvis of the right kidney.

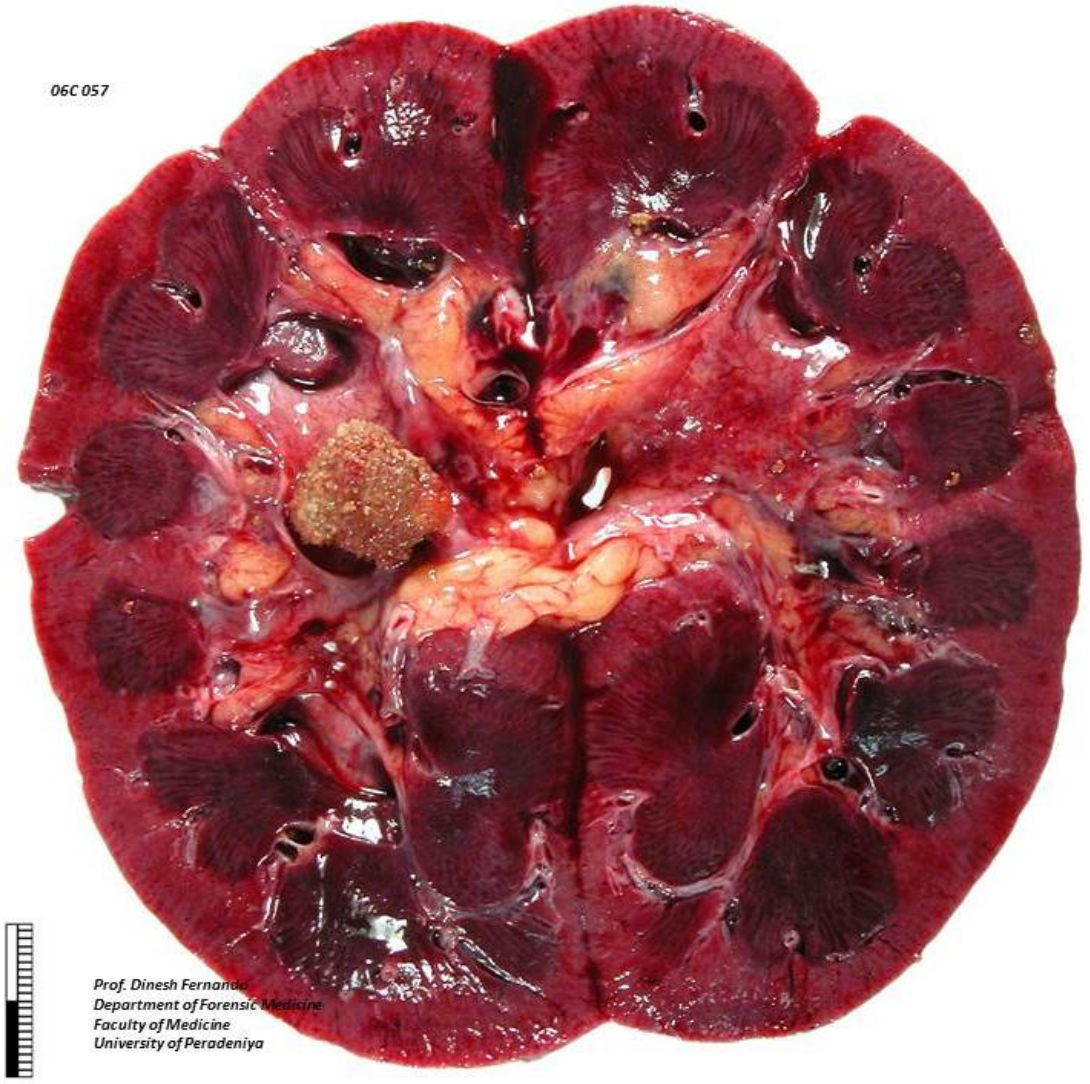


Figure 17: Cut surface of kidney. Note the calculus in the pelvis of the right kidney and thinning of the cortex and congestion of the medulla.

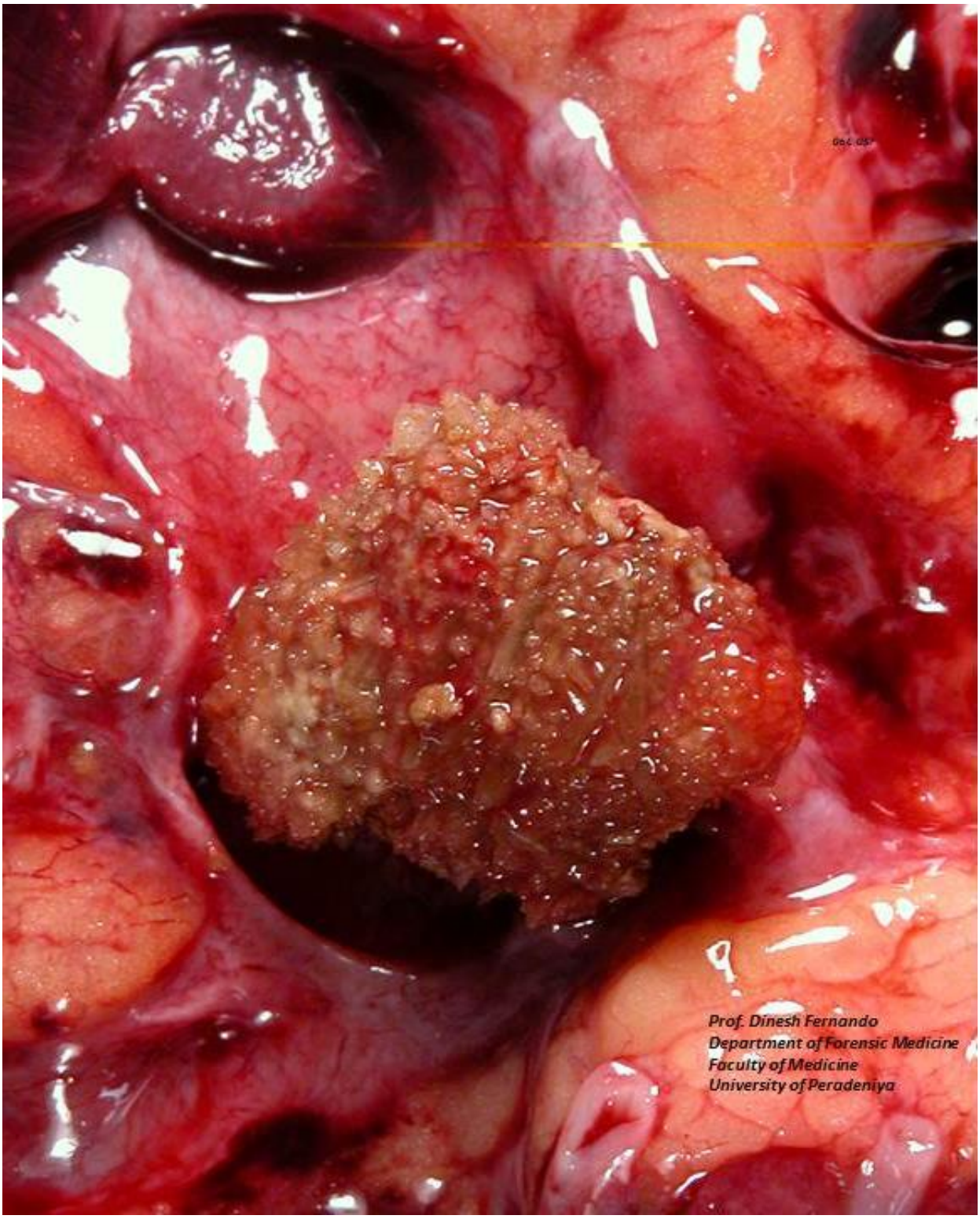


Figure 18: The calculus with a rough surface in the pelvis of the right kidney.

Tumours of kidneys

Tumours of kidneys may be benign or malignant. Benign tumours of kidneys include adenoma, angioma and angiolipoma and the malignant tumours include renal cell carcinoma, urothelial carcinoma and nephroblastoma.

The carcinomas of transitional cell epithelium, which lines the renal calyces, renal pelvis, ureter, bladder and urethra, accounts for 3% of deaths from all forms of malignancy. These are rarely seen in people below 40 years of age. They are commonly seen in males with a male to female ratio of 4:1. Bladder tumours are more common than tumours of ureters and renal pelvis. Transitional cell carcinomas of the renal pelvis or ureters accounts for 5-10% of those of the urinary tract.

Smoking, exposure to industrial carcinogens such as beta-naphthylamine and benzidine, exposure to drugs like phenacetin and cyclophosphamide and chronic inflammation can causes for urothelial tumours.

Patients with bladder malignancy commonly present with painless haematuria. Presenting symptoms may also be due to local metastases. If ureteric obstruction occurs due to ureteric lesions, the patient may present with flank pain.

Urine cytology for malignant cells, urinary tumour markers and cystoscopy are the investigations used in the diagnosis of urothelial tumours. The standard treatment of upper tract transitional cell carcinoma is nephroureterectomy.

Renal cell carcinoma occurs mostly in older individuals. It is associated with risk factors like cigarette smoking, obesity, exposure to asbestos, heavy metals and acquired cystic renal disease. The classic clinical features of renal cell carcinoma are costovertebral pain, palpable mass and haematuria. The haematuria is usually microscopic and intermittent. Once the tumour becomes large, the patient may present with fever, malaise, weakness and weight loss. Renal cell carcinomas can produce a variety of systemic symptoms that are not directly related to the carcinoma, including polycythemia, hypercalcaemia, hypertension, hepatic dysfunction, Cushing syndrome and amyloidosis. It has higher tendency to metastasize widely before giving rise to any symptoms. Radical nephrectomy had been the treatment of choice, but partial nephrectomy to preserve the renal function is also recommended for less advanced tumours. Although these can arise in any part of the kidney, it is commonly seen in the upper poles of the kidneys.

Renal cell carcinomas can be classified according to the cytogenic, genetic and histological basis, into clear cell carcinoma, papillary renal cell carcinoma and chromophobe renal carcinoma. Clear cell carcinoma arises from the proximal tubular epithelium and may contain interstitial foam cells and psammoma bodies. The stroma of the tumour is highly vascularized. Chromophobe renal cell carcinoma tends usually have clear, flocculent cytoplasm with very prominent, distinct cell membranes. The nuclei are surrounded by halos of clear cytoplasm.

History

A 75-year-old female with a history of ischaemic heart disease, renal disease, rheumatoid arthritis, was found dead. She had been a smoker and had been investigated for a carcinoma of an unknown origin.

Internal Examination

Urinary Tract: The right and left kidneys weighed 250 grams and 252 grams respectively. The renal capsule of the right kidney was thickened and adherent. The cortical surface was pale. Upon cross section a tumour was present in the medulla of the kidney which was yellowish/white in colour. Marked dilatation of the pelvicalyceal system was present with thinning of the cortex and obliteration of the cortical medullary junction. The left kidney capsule stripped easily to reveal a pale cortical surface. The renal parenchyma had pale cortex and medulla with distortion of the cortical medullary demarcation. The pelvicalyceal system appeared to be within normal limits. The bladder was empty and no tumours were present in the mucosa.

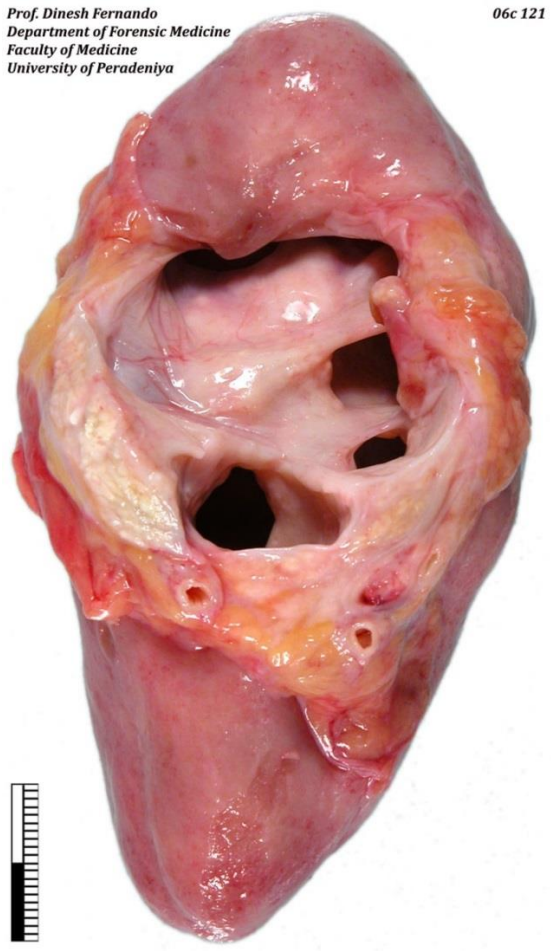


Figure 19: Pale cortex of the kidney and the dilated pelvicalyceal system

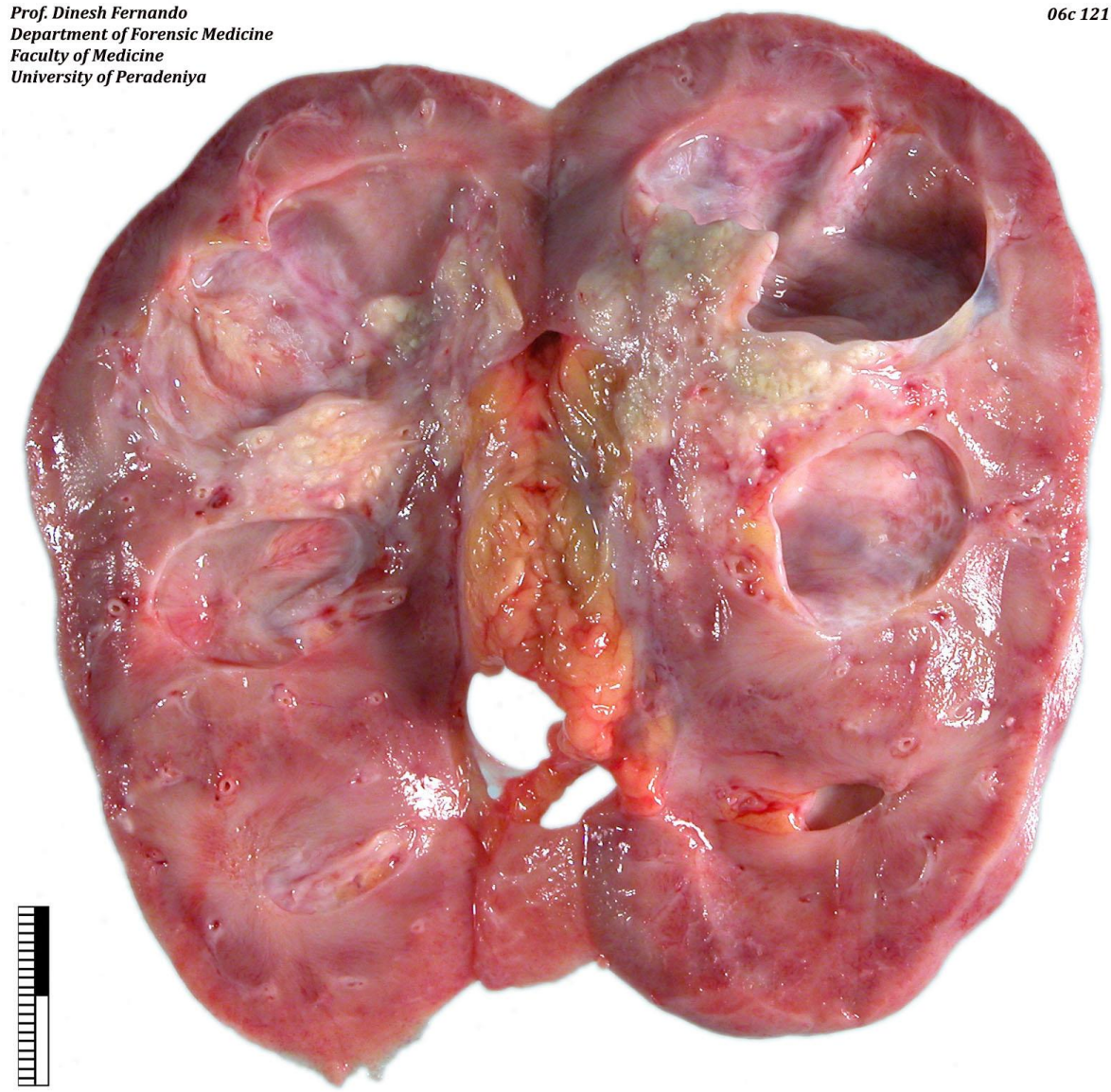


Figure 20: Cross section of the right kidney: note the yellowish white colour tumour in the medulla

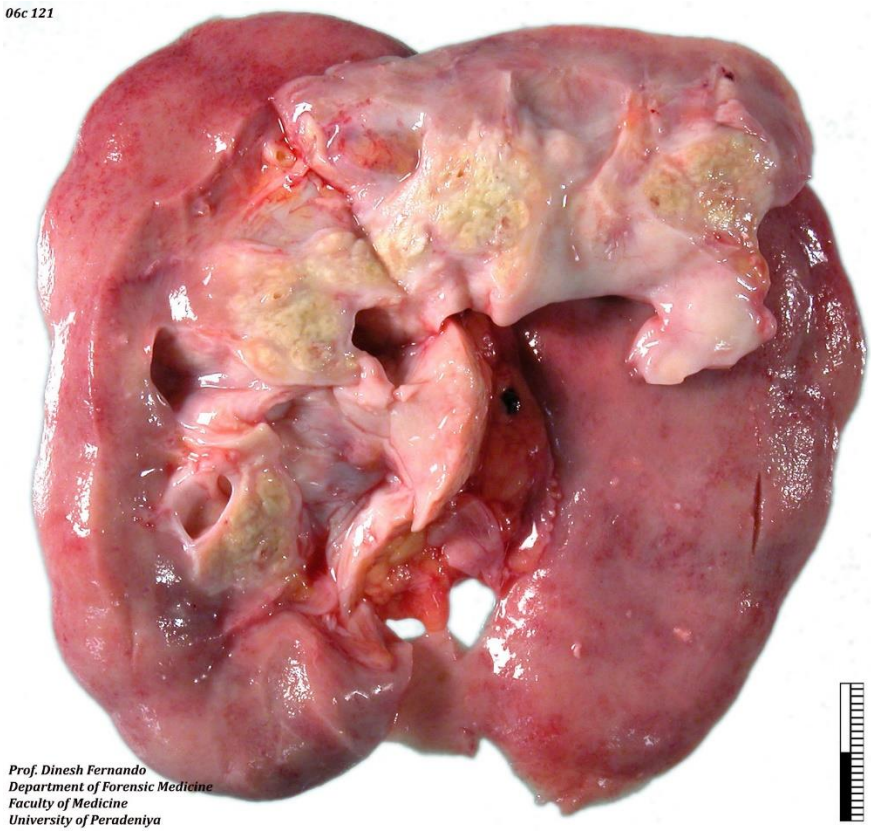


Figure 21: Tumour in the right kidney

Liver and Biliary Tract: The 2,154 gram liver had multiple metastases which ranged from 0.5 cm to 3 cm in diameter and were multiple discrete white hard lesions. The rest of the parenchyma had a nutmeg appearance. The hepatic margins were rounded.

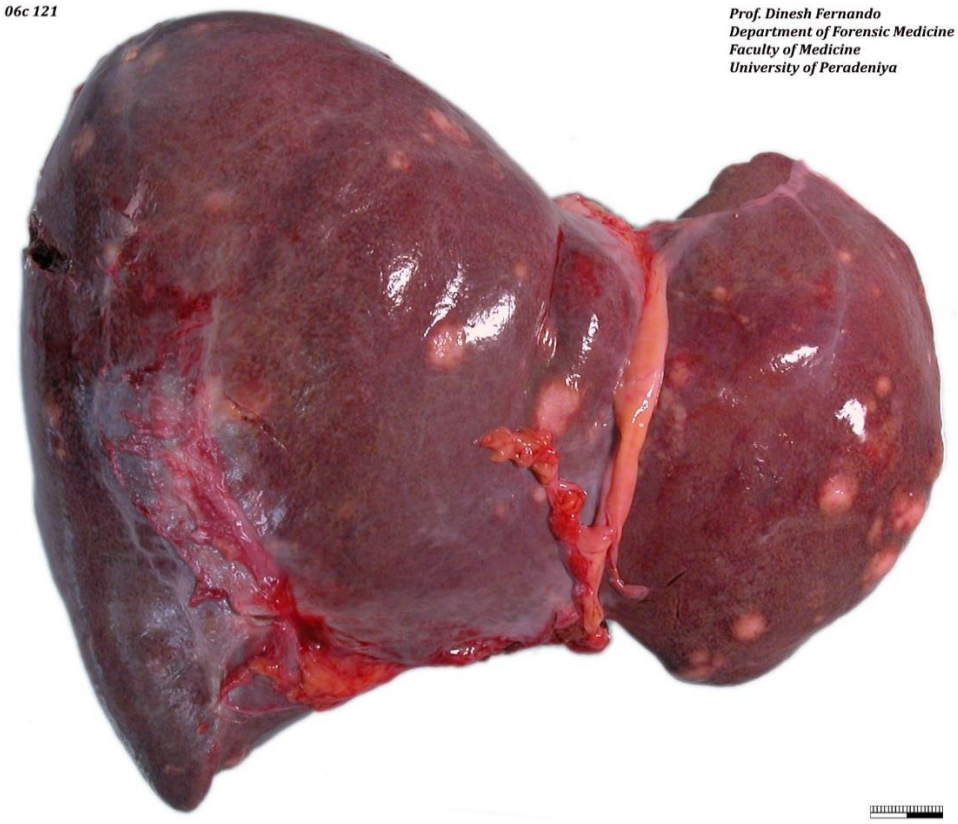


Figure 22: Anterior surface of the liver; note the metastatic tumour

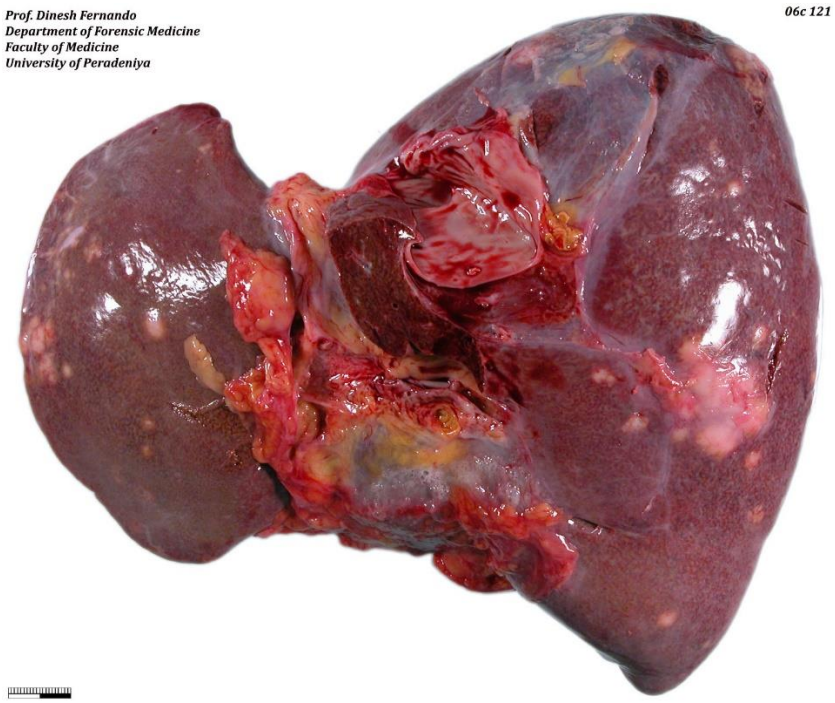


Figure 23: Posterior surface of the liver: note the metastatic tumour

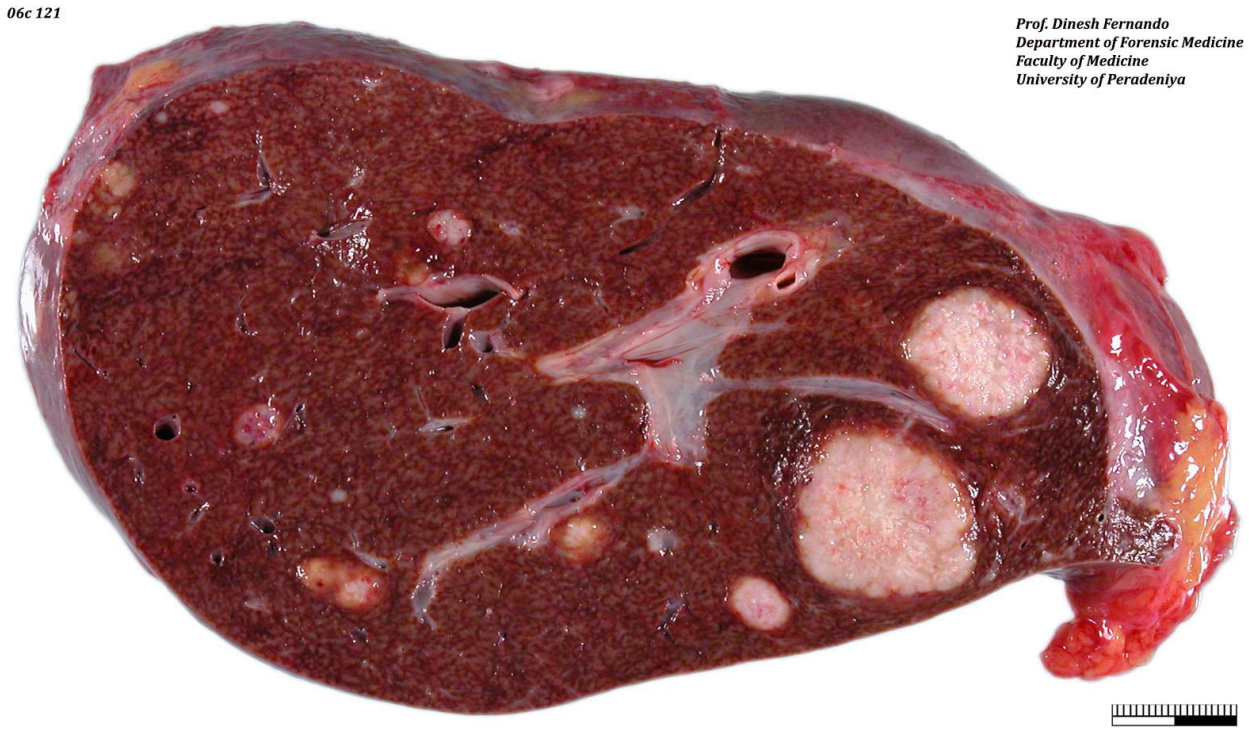


Figure 24: Cross section of the liver; note the multiple discrete hard lesions and nutmeg appearance

Microscopic Examination

Sections of the right kidney showed evidence of an urothelial carcinoma of the renal pelvis. No malignant cells were seen in the left kidney. Both kidneys showed evidence of early acute tubular necrosis.

The bladder showed evidence of urothelial (transitional) carcinoma.

The para-aortic lymph nodes showed evidence of malignancy.

The liver showed central venous congestion and infiltration by malignant cells.

Cause of death

Acute myocardial infarction

Urothelial carcinoma of kidney and bladder with metastases to regional lymph nodes and liver was identified as other significant conditions contributing to the death, but not relating to the disease or condition causing it.

Congenital anomalies

The urinary tract is formed from the cloaca and the intermediate mesoderm. The kidneys are formed by early differentiation of the metanephric blastema. The ureteric bud arises from the distal end of the mesonephric duct and invades the adjacent metanephric mesenchyme, forming a branching collecting system within the primitive kidney. There are several congenital anomalies that are seen in the kidneys, namely, unilateral renal agenesis, bilateral renal agenesis, multi-cystic disease, ectopic kidney, horseshoe kidney, duplex renal pelvis and ureters and ectopic ureters.

In horseshoe kidneys, the two lower poles of the kidneys have fused to form an isthmus and the two upper poles lying on each side of the midline. It can be found at any location along the path of the normal ascent of kidneys. The ascent of the kidneys is prevented by the inferior mesenteric artery. Horseshoe kidneys are the most common form of renal fusion anomaly and are prone to reflux, obstruction and stone formation. The vascular supply could be from the aorta, iliac arteries, or the inferior mesenteric artery.

The horseshoe kidney itself does not cause any symptoms and most frequently is detected as an incidental finding during radiological examination. Abnormal blood supply, abnormal course of the ureters and high insertion points of the ureters may cause problems in this condition. High insertion of ureter into the renal pelvis causes ureteropelvic junction obstruction, which is the most commonly associated finding in horseshoe kidney. Some cancers are commonly associated with this condition, especially, renal cell carcinoma, Wilms tumour and carcinoid tumours. They are more susceptible to blunt abdominal trauma, since it is unprotected by the rib cage. The management depends on the medical and surgical complications of the horseshoe kidney.

Duplex renal pelvis and duplex ureter is a common anomaly of the urinary system. If the ureteric bud bifurcates after its initial development from the mesonephric duct, incomplete duplex ureters with Y ureter arises. If there are two separate ureteric buds, complete ureteric duplication can occur giving rise to one ureter in the normal position and the other one in a lower position. The non-refluxing normal ureteric bud ends at the trigone of the bladder and the lower bud ends in the bladder with a laterally placed ureteric orifice with a short submucosal tunnel and drains the lower pole of the kidney. Duplex ureters are associated with vesico-ureteric reflux.

History

A 79-year old female, with an adenocarcinoma of the sigmoid colon, was found dead in her home.

Internal Examination

Urinary Tract: A single horseshoe kidney was present as an incidental finding, weighing 350 grams. The renal capsule stripped easily to reveal cortical surfaces that were red-brown, smooth and glistening. The renal parenchyma revealed a good definition of the cortical and medullary areas with a normal cortical width. Each side of the horseshoe kidney had a pelvicalyceal system and ureter. The ureters entered into a single bladder normally.

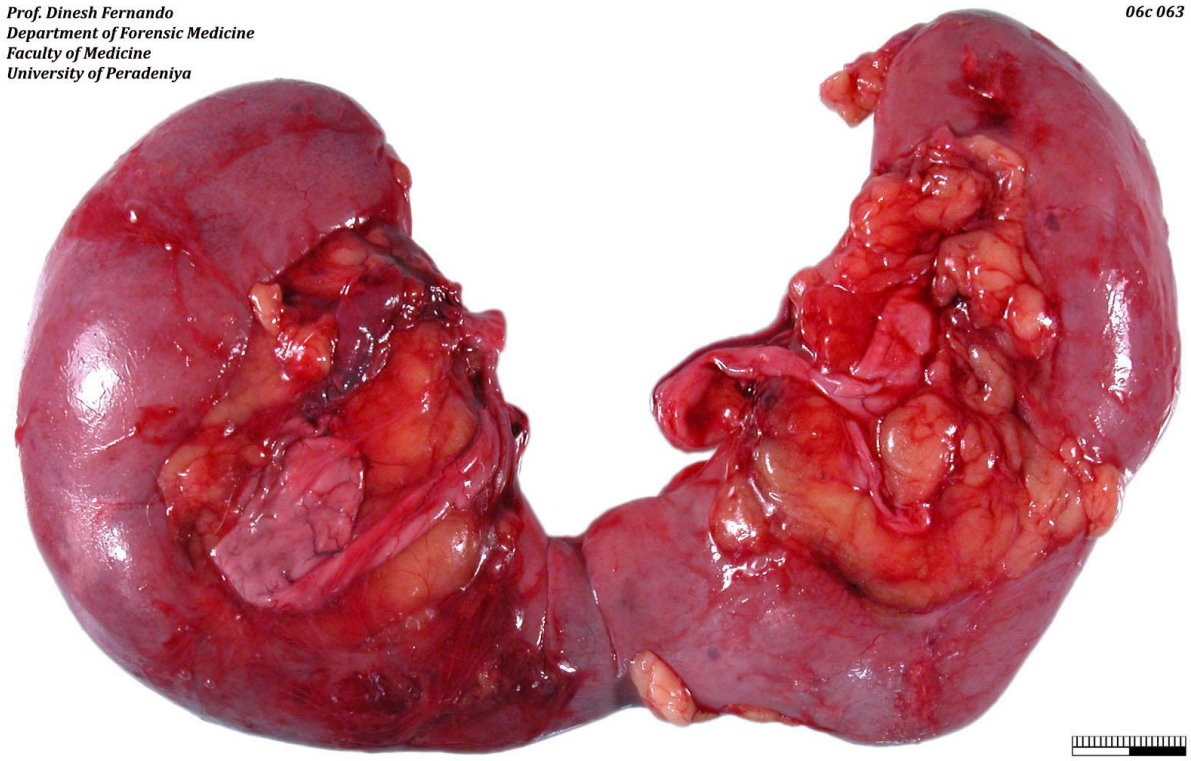


Figure 25: Horseshoe kidney

History

A 41-year-old female with a history of ischaemic heart disease, was found dead in her sleep. One day previously she had complained of chest pain and dizziness.

Internal Examination

Urinary Tract: A single horseshoe kidney weighing 238 grams was found as an incidental finding. The renal capsules stripped easily to reveal cortical surfaces that were red-brown, smooth and glistening. The renal parenchyma revealed a good definition of the cortical and medullary areas with a normal cortical width. Two ureters were present. The papillae, calyces, pelvis, ureters and bladder were unremarkable. The bladder contained 200mls of urine.

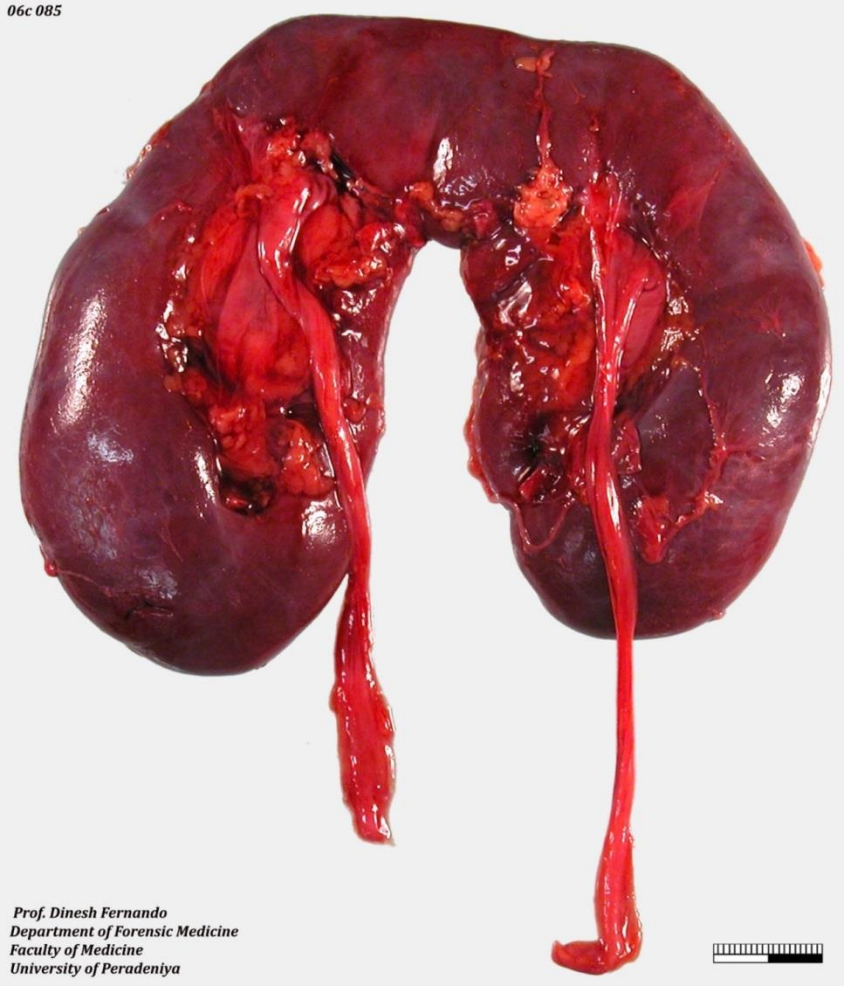


Figure 26: Horseshoe kidney

History

A 72-year-old male was found dead in his home.

Internal Examination

Urinary Tract: The right and left kidneys weighed 172 grams and 186 grams respectively. The renal capsules stripped easily to reveal cortical surfaces that were red-brown, smooth and glistening. The left kidney had a double ureter.

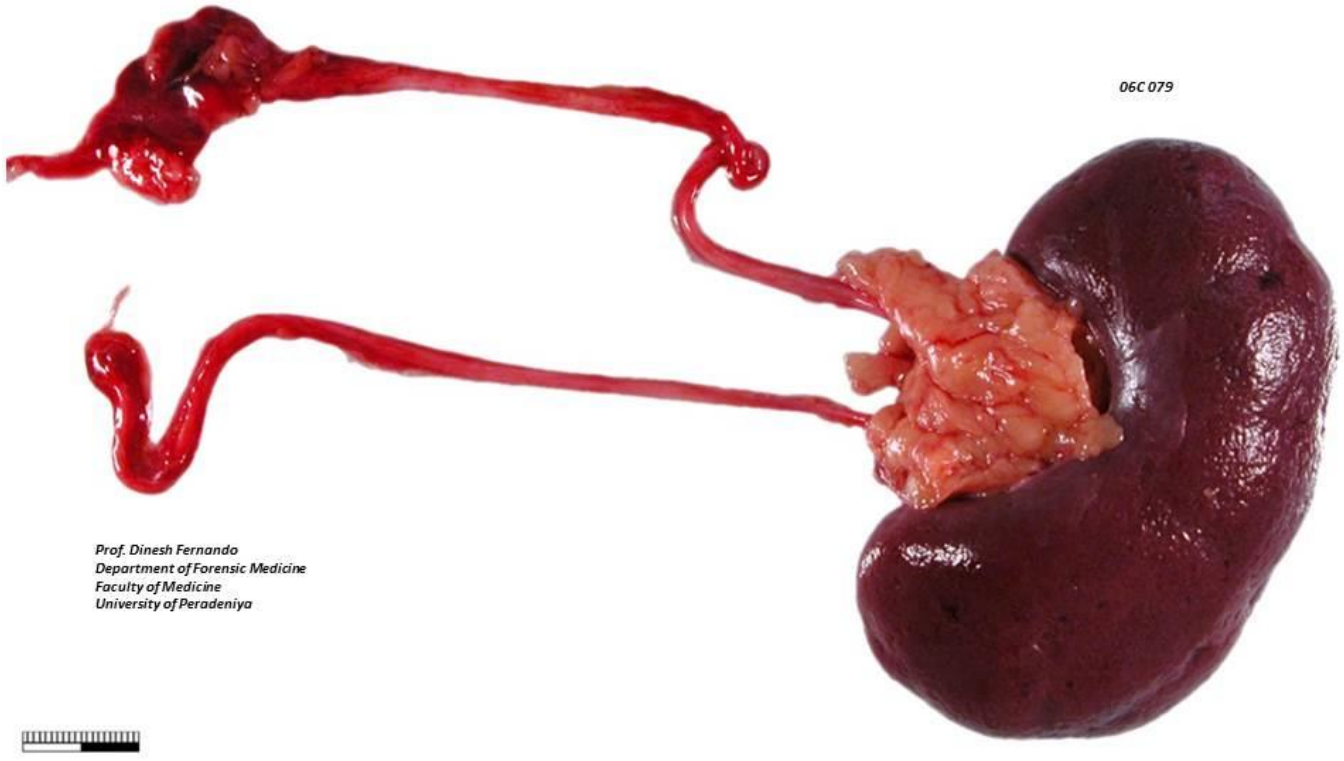


Figure 27: Left kidney with double ureter

Renal arterial stenosis

Renal artery stenosis is the narrowing of the renal artery. It can lead to systemic hypertension. The other complications of renal artery stenosis include chronic kidney disease and end stage renal disease. The stenosis of the artery can occur bilaterally or unilaterally and the two major causes for unilateral renal artery stenosis are atherosclerosis and fibromuscular dysplasia. Atherosclerosis of the renal artery mainly involves the proximal 2 cm of the renal artery. Risk factors for atherosclerosis include, dyslipidaemia, smoking, viral infection, immune injury and increased homocysteine level. Fibromuscular dysplasia commonly occurs in women younger than 50 years and it usually involves the middle and distal main renal artery and the intrarenal branches. When a patient presents with hypertension, the likelihood of renovascular disease being the underlying cause should be suspected. Clinically, this may be suspected by treatment resistant severe hypertension, young hypertension with a negative family history, unexplained hypokalaemia, acute and sustained rise in serum creatinine and recurrent episodes of flash pulmonary oedema. The gold standard for diagnosis of renal artery stenosis is renal arteriography.

Initial treatment for renal artery stenosis is observation of the progression of the disease with accurate correction of dyslipidaemia, use of drugs that block platelet aggregation and control of hypertension.

History

A 42-year-old female with type 2 diabetes mellitus, end stage renal failure which was managed with dialysis, hypercholesterolaemia and obesity was presented collapsed to the hospital. She had previously complained of coughing up of blood and melena. She died after admission to the hospital.

Internal Examination

Cardiovascular System: The abdominal aorta had significant atheroma which had caused partial occlusion of the superior and inferior mesenteric arteries. The left kidney was supplied by three renal arteries which arose from the aorta. These were small caliber vessels which were calcified. The right kidney was supplied by one renal artery which had stenosis at its origin. The renal ostia were not identifiable due to atheroma.

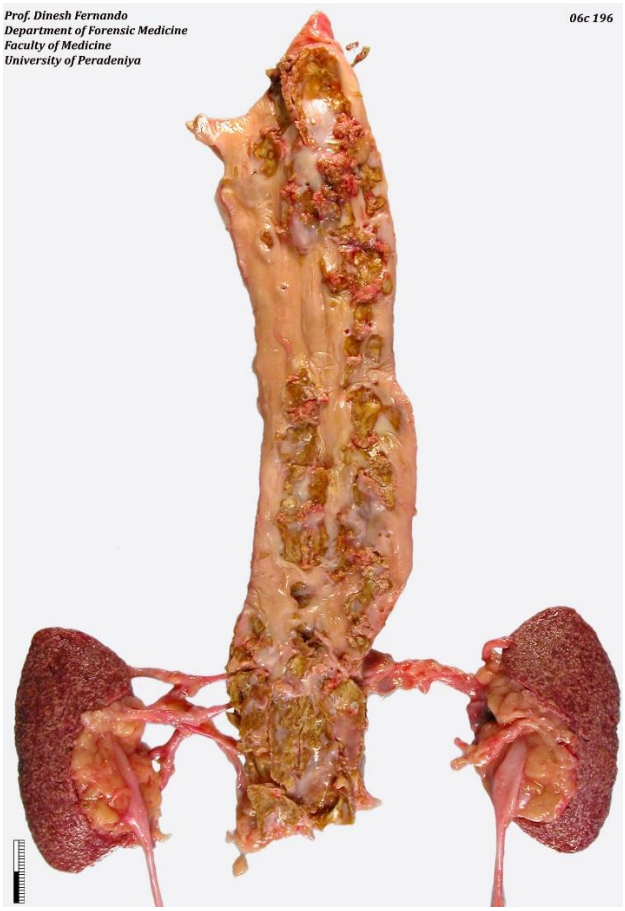


Figure 28: Significant atherosclerosis obstructing the origin of the renal arteries.

Genitourinary System: The right and left kidneys were very small and weighed 46 grams and 42 grams respectively. The renal capsules were adherent to granular cortical surfaces. The renal parenchyma revealed a very thin cortex with no distinct medulla. The pelvicalyceal system and ureters were unremarkable. The bladder was empty and the mucosa was unremarkable.

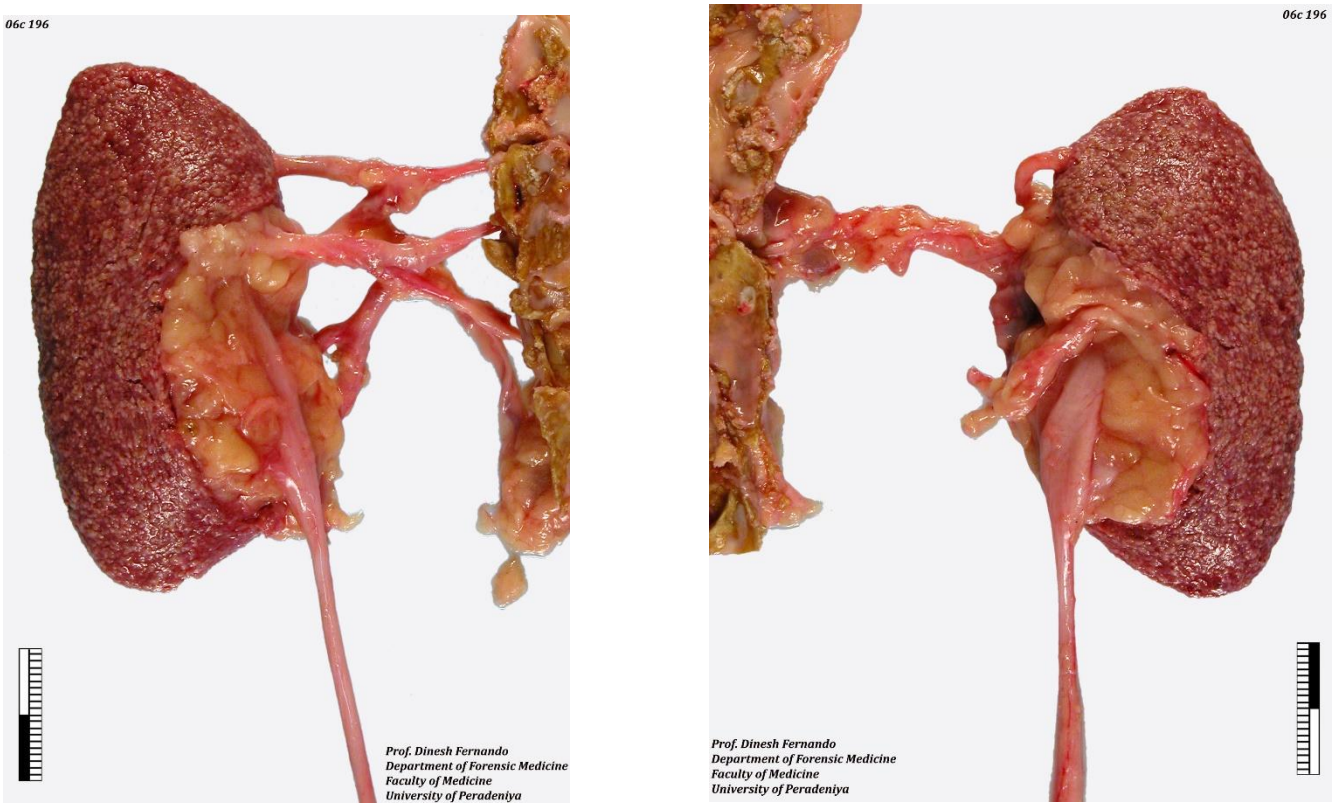


Figure 29: Left kidney supplied by multiple renal arteries. Right kidney supplied by one renal artery. Note the granular contracted kidneys

Cause of death

Gastrointestinal haemorrhage from multiple subacute gastric ulcers

Chronic renal failure secondary to diabetes mellitus and hypertension was determined as other significant conditions contributing to the death, but not related to the disease or condition causing it.

Bibliography

1. Kumar V, Abbas AK, Aster JC. *Robbins basic pathology*. 9th ed. Philadelphia: An Imprint of Elsevier; 2013.

2. Kumar P, Clark ML. *Kumar & Clark's Clinical Medicine*. 9th ed. Edinburgh: Elsevier Health Sciences; 2017.

3. Chirag ND, Nephrolithisis. [Updated 2020 Jan 13], Medscape, Available at: https://emedicine.medscape.com/article/437096-overview.

4. Fulop T, Acute Pyeloneprhitis. [Updated 2021 Jul 01], Medscape, Available at: https://emedicine.medscape.com/article/245559-overview.

5. James WL, Chronic Pyelonephritis. [Updated 2019 Sep 17], Medscape, Available at: https://emedicine.medscape.com/article/245464-overview.

6. Frye TP, Cystic disease of the kidney, [Updated Sep 10], Medscape, Available at: https://emedicine.medscape.com/article/453831-overview.