

RENAL SYSTEM - 1



DR. MUHAMMAD TARIQ JAVED

Professor,
Department of Pathology,
Faculty of Veterinary Science,
University of Agriculture, Faisalabad, Pakistan.
Email: mtjaved@uaf.edu.pk



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ANOMALIES OF DEVELOPMENT

RENAL AGENESIS

- Renal agenesis is always accompanied by **ureteral aplasia** and may be associated with **aplastic reproductive tissues** on the same side.
- The condition is typically an incidental finding so long as the other kidney is functioning normally.
- **Bilateral agenesis** results in early perinatal death.

RENAL DYSPLASIA AND HYPOPLASIA

- These defects are most common in **dogs** and have been reported in many breeds,
- Renal dysplasia may be unilateral or bilateral.
- Affected animals usually have **polydipsia** and **polyuria** that precede signs of **uremia**.



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ANOMALIES OF DEVELOPMENT

- **Dwarfing** may be noticed if the onset of renal failure occurs within the first few months of life.
- **Uremia** is usually identified between 6 mo to 2 yr of age.

Grossly

Hypoplastic Kidneys

- The hypoplastic kidney is uniformly smaller than normal
- In lobulated kidneys, it may be more severe in certain lobules
- The **capsule** is usually **thickened** and is often adhered to the cortex.
- The **parenchyma** is usually **grayish** in colour and tends to be **firm**.



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ANOMALIES OF DEVELOPMENT

- On cut a reduction in parenchymal volume occur

Dysplastic Kidneys

- Kidneys are usually **misshapen** and **fibrosed**
- They often contain thick-walled **cysts**.
- **Ureters** are usually **tortuous** and **dilated**.



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ANOMALIES OF DEVELOPMENT

Microscopically

Hypoplastic kidneys

- There are less number of **nephrons**.
 - Evident from less **glomeruli** than normal.
- The **tubules in medulla** are often **dilated** and epithelial cells are flattened.
- Usually considerable **interstitial connective tissue** which causes separation of the tubules.

Dysplastic Kidneys

- **immature glomeruli**
- collecting **tubules** ending **blindly**, or
 - primitive ducts surrounded by **concentric layers** of **mesenchyme**



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ANOMALIES OF DEVELOPMENT

MALPOSITION OF THE KIDNEY

- is **rare** in animals
- may be unilateral or bilateral.
- Mal-positioned kidneys are mostly displaced caudally, with the kidneys assuming a **pelvic** or **inguinal** disposition.
 - In such cases the renal **arteries** usually arise from the **terminal aorta** near its bifurcation and the **ureters** are correspondingly **shortened**.
 - Ureters often have **kinks** which predispose affected kidneys to develop hydronephrosis.
 - Mal-positioned kidneys are usually otherwise normal.
 - Malposition of the kidneys in swine may be associated with **vitamin A** deficiency in sows.



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ANOMALIES OF DEVELOPMENT

PERSISTENCE OF FOETAL LOBULATION

- Persistence of foetal lobulation occurs due to a failure of fusion of individual **renal segments**.
- It is normal in cattle and some other animals
- This occurs abnormally in **swine** and has been associated with **vitamin A** deficiency.

HORSESHOE KIDNEY

- Condition is seen in all species
- It results from a fusion of the cranial or caudal poles of the kidneys
- Such kidneys tend to function normally.



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ANOMALIES OF DEVELOPMENT



Figure 4.8 Horseshoe kidney in a calf. Ureters have been severed (arrows).



Textbook: Pathology of Domestic Animals by Jubb, Kennedy and Palmer

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ANOMALIES OF DEVELOPMENT

RENAL CYSTS

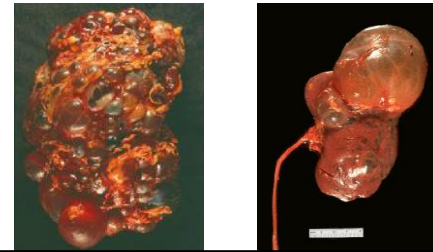
- Are thin-walled fluid filled cavities present in the renal parenchyma.
- They vary in size from hardly visible to very large, often exceeding the size of the affected kidney.
- They may be **solitary** or **multiple**.
- Mostly **congenital** but may develop post-natally.
- They have been reported in all species but occur most commonly in **swine**.
- Renal cysts can arise in any part of the nephron or in collecting tubules
- Most congenital cysts are not caused by **obstructive lesion**.



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ANOMALIES OF DEVELOPMENT

- There seems to be some association between renal **dysplasia** and the renal cysts
- In some cases, polycystic kidney disease is also associated with cystic bile ducts, bile duct proliferation, and cystic pancreatic ducts.
- In severe cases of congenital polycystic kidneys, there is usually **stillbirth** or **early neonatal death** due to renal failure.



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RENAL FAILURE

- Is the condition resulting from suboptimal renal function.
- **uremia is characteristic feature of renal failure**
- The symptoms include
 - tiredness,
 - nausea,
 - vomiting,
 - pruritis and
 - twitching.



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- Renal failure may develop **acutely (develops in minutes, hours, or a few days)**,
- **Chronic** renal failure develops over a longer period of time and usually progresses to terminal stages
- Renal failure may result from decreased perfusion of the kidneys, as in **shock**, but usually it is due to decreased functional parenchyma of the kidneys.



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ACUTE RENAL FAILURE

- Kidney stops working.
- Oliguria to anuria.
- Reversible
- Common cause- acute perfusion failure
- Lab Data:
 - increased BUN and Serum Creatinine
 - Hyperkalaemia (K),
 - metabolic acidosis,
 - urinary sodium loss,
 - same plasma and urine osmolality.

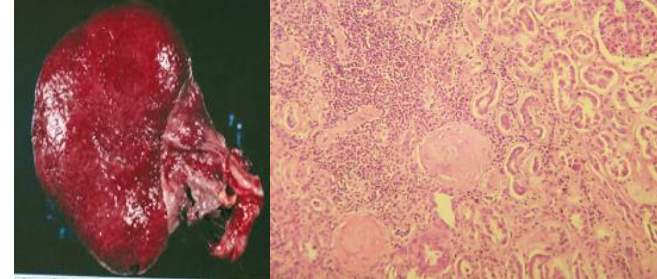


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CHRONIC RENAL FAILURE

- Irreversible
- Slow progression
- Uraemia, polyuria, bone disease, anaemia.
- Common causes: hypertension and diabetes.
- Leads to end-stage renal disease.



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End-Stage Kidney

- Small, shrunken and granular
 - chronic glomerulonephritis
- D/D
- Chronic pyelonephritis
 - hyalinized glomeruli, tubular atrophy and interstitial fibrosis.



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CIRCULATORY DISTURBANCES

RENAL HYPERAEMIA

- Hyperaemia of the kidney may be active or passive
- Active hyperaemia occurs in
 - acute nephritis
 - acute septicemias and
 - bacterial intoxications.
 - In either case, the kidney will be swollen and uniformly dark grossly.
 - Microscopically, all vessels, especially capillaries, are dilated and engorged with blood.



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RENAL HAEMORRHAGES

- It is not uncommon and occurs frequently with a variety of bacteraemias and viraemias.
- Pinpoint haemorrhages occur beneath the capsule in hog cholera and in porcine salmonellosis.

INFARCTION OF THE KIDNEY

- Kidneys are easily infarcted.
- The sequelae to infarction depend on the nature of the infarction material.
- If the obstructing material is non-septic, typical infarcts are formed.



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- Occlusion of a **renal artery** will cause infarction of the entire kidney; but, occlusion of a **branch** of the renal artery will cause infarction of the corresponding region of the kidney.
- Most renal infarcts involve the **interlobular arteries** and result in a **pyramid-shaped** area of necrosis in the cortex of the kidney.



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RENAL CORTICAL NECROSIS & ACUTE TUBULAR NECROSIS

- The two conditions are quite similar, differing primarily in the size of the affected areas.
- In acute **tubular necrosis**, the individual lesions tend to be **small and patchy**, affecting only a few tubules.
- In renal **cortical necrosis**, the necrotic foci tend to involve **larger cortical areas** and result in necrosis of tubules and **glomeruli**.
- These lesions occur infrequently in animals but either may be severe enough to cause renal failure and death.
- Various mechanisms are incriminated in the different species; however, it is probable that these lesions result from **ischaemia**.



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- The ischaemia may be due to decreased perfusion of the cortex (as in hypotension) **or due to disseminated intravascular coagulation (DIC)**.

Grossly

- the lesions tend to be variable, probably depending on the
 - severity,
 - the distribution,
 - the duration, and
 - the quality of the reflow to the area.
- In acute **tubular necrosis**, the cortices are finely mottled by either **reddish or yellowish foci**.



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- In renal cortical necrosis, the entire cortex may be affected or the lesion may be patchy.
 - When the lesions are patchy, they tend to **resemble infarcts**.
 - Affected areas are
 - pale,
 - slightly swollen, and
 - sharply demarcated from the medulla.

Histologically

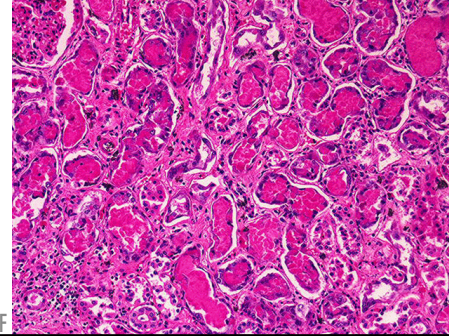
- There is irregular necrosis of the cortex.
- In acute tubular necrosis, the necrosis is limited to small foci involving only a few tubules.



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- The necrosis results in disruption of the basement membranes of the tubules and usually **tubular casts** develop in collecting tubules.
- In renal cortical necrosis, various patterns of infarction may be seen, causing necrosis of **tubules and glomeruli** in affected areas in the cortex.



Acute Tubular
Necrosis,
Toxic



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RENAL MEDULLARY NECROSIS

- Can occur as the primary manifestation of renal disease.
- The mechanism is poorly understood, but it seems to involve a **vascular change**.
- In cases where venous return from the kidneys is **obstructed**, the medulla tends to be more devastated.
 - This is because the elevated intra renal pressure maintains the patency of the cortical vessels but not the medullary vessels causing medullary infarction.
- Also, **prostaglandin synthetase** is found, in the kidney, primarily in the medulla, and inhibition of this enzyme results in **decreased production of PGE2 and loss of its vasodilatory effects on juxtamedullary arterioles**.



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- Sequelae to medullary necrosis are due primarily to **loss of ability to concentrate urine** and include
 - renal failure and
 - uraemia.
- **Acute infarction** may be an **agonal change** in cases of severe dehydration and electrolyte imbalances due to neonatal diarrhea.
- Massive medullary necrosis would undoubtedly lead to renal failure and possibly death.



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Grossly

- If the medullary necrosis is not severe enough to cause death, **scarring** of the medulla occurs.
- The necrotic portion of the medulla may **slough** and lodge in the **renal pelvis** eventually becoming **mineralized**.

Microscopically

- The lesions range from areas of coagulative necrosis with haemorrhage in early lesions to areas of connective tissue scarring in healed lesions.

