There seems to be a change in the poultry disease scenario. The pathological lesions of many diseases are presenting a different picture and besides, new syndromes are emerging in many parts of the world.

There is an increasing involvement of kidney lesions in a number of diseases. The kidneys are an important organ in the chicken’s body, responsible for the excretion of wastes in the form of urine which consists of a semi-fluid product containing 0.75g of nitrogen of which 85% is uric acid and the rest from ammonia, urea and amino acids. Its ash contains sodium, potassium, magnesium, calcium, phosphorus, chloride and sulphur.

Chicken urine is usually slightly acidic and approximately 120ml is passed daily by an adult bird. Kidneys also regulate and maintain the acid balance of extra cellular fluid volume by excretion or reabsorption of water. Selective reabsorption and conservation of glucose and sodium chloride also takes place in the kidneys. The normal rate of flow of chicken urine is so low that only 0.02 ml is passed in one minute by a bird weighing 1kg while the bird is drinking 100ml of water during 24 hours. Under normal conditions 28.8ml of water is retained by the body while 71.20ml is passed out with urine in spite of the fact that considerable water and sodium is reabsorbed in the cloaca, making the urine pasty due to the concentration of insoluble uric acid. During a state of dehydration as much as 15% of water and 50% of sodium is reabsorbed. The excretion of whole water through the kidneys depends upon a number of factors, including environment, feed and water quality, besides disease. Depending upon the amount of feed and its crude protein content, it is estimated that an adult chicken’s urine has a total nitrogen content of 440-450mg/100ml and 84% of uric acid, 7% ammonia, 5.5% urea and smaller amounts of amino acids. Uric acid is a breakdown product of protein and is synthesized in the liver but excreted through tubular filtration and secretion at a rate of 1.5mg/min. Various excreting inorganic ions, sodium, potassium and ammonium are passed in urine along with sulphate, phosphate and chloride. Their contents depend on the feed, bodyweight/growth, egg production, the body physiological status and the water intake. During a number of disease conditions, water intake is effected and so is the excretion and contents of urine. Pathological lesions in the kidneys are marked but often overlooked in routine post-mortem examination in spite of the fact that they can greatly help in reaching a conclusive diagnosis.

Kidney Lesions In Poultry Diseases

The poultry disease scenario appears to be changing with an increasing involvement of kidney lesions. — By Dr A.A. Qureshi

Visceral gout lesions in a young pullet showing precipitate deposition of ureates in kidneys, heart and visceral mesenteries.

Urolithiasis lesions in a broiler chick showing distended ureters and kidneys with uroliths.
Amongst the most common situation is water deprivation of birds from their water requirements so that dehydration sets in due to the depletion of extracellular water as the osmolarity is raised, while the urinary output is decreased but the serum concentration of electrolytes is raised. Feed content of sodium and potassium also effects water intake and help to regulate and maintain isotonicity of the extra-cellular body fluids. During dehydration, the urinary output falls while the sodium contents of urine diminishes which ultimately leads to kidney failure and death. Pathological lesions of visceral gout, quite common in birds reared in cages and characterised by precipitation of urates in the serous membranes of kidneys, liver, mesenteries, air sacs and peritoneum often results from poor water intake or dehydration resulting in failure of the urinary excretion. Similar pathological lesions of visceral gout are seen in cases of Avian Monocytosis often involving the kidneys.

Other conditions involving kidney lesions showing abnormal urates deposition include vitamin A deficiency, excess of calcium in feed and high protein diet as well as mycotoxin poisoning. Enlargement of kidneys and deposition of urates is also seen in some outbreaks of Infectious Bronchitis disease because of a specific virus strain. In cases of visceral gout there is an over-production of uric acid but its precise biochemical cause is now known except that it is induced by an upset of certain enzymes and hormones which lead to hyperuricaemia and hypercalcaemia.

Urolithiasis is another common renal condition seen in broilers, layers and breeders, most commonly due to feed-associated factors. It is characterised by distended urates with a hard core like stone formation and a varied degree of nephrotic atrophy. The histopathology of the affected kidney shows tubular degeneration and dilated urethral branches with fine micro-pleomorphic crystalline urates. The composition of uroliths varies with the underlying disorder responsible for their production but generally they are crystalloids of calcium sodium urate while uroliths in case of gout, consist of uric acid only. These stones are formed due to both physiological and pathological factors but the various mechanisms favouring their formation include dehydration, hard water, excessive mineral contents of feed, vitamin D intoxication and excessive excretion of crystalloid constituents. A physicochemical change in the urine such as extreme urinary pH is also responsible for the formation of uroliths. An alkaline urine leads to the formation of calcium stones while an acid urine leads to uric acid stones. Uric acid is synthesized in the liver and excreted through the kidneys by means of filtration and tubular secretion in
the presence of an enzyme known as xanthine oxidase. In an adult bird with a plasma level of 2.5 mg/100ml of the uric acid, nearly 90% is excreted in urine through secretion but in disease and with some drugs, it is greatly cut down. The incidence of urolithiasis increases with increased uric acid production as it is much less soluble than monosodium urates. As such, it is therefore a predominant compound in the formation of urinary stones. Hyper vitaminosis-A in toxic reaction also induces hypercalcemia and subsequent lithiasis. The main histopathological lesions in urolithiasis are tubular degeneration. At present, due to an unusually increased incidence of nephrosis and Urolithiasis, there is a common practice in this country called 'flushing' the kidneys using a number of products, the commonest being a 2% sugar solution, with a lot of success.

During recent years the problem of mycotoxins in feed has caused heavy losses in broiler and layer flocks due to lesions of Hepato-nephrosis. Although the liver is the main organ involved, kidneys are invariably affected, showing swelling and haemorrhages. Ochratoxins are the most damaging of the mycotoxins for poultry. In some feed samples ochratoxin contents of 570 to 1650 ppb have been determined during the last year by ELISA.

Histopathological studies indicated hyperplasia of the tubular epithelium, heterophylic nephritis and presence of urate casts. Citrinin and oosporein are also nephrotoxic mycotoxins.

Newcastle Disease has an epidemic incidence in Pakistan and Infectious Bronchitis outbreaks are quite common. In fact, the incidence of viral infections has increased manifold due to mycotoxin problems of feed quality and a lack of biosecurity. Respiratory infections including Infectious Bronchitis have a high incidence during the winter season in spite of vaccination. Respiratory symptoms are more prominent in IB outbreaks, but kidney lesions are invariably seen, too, with lesions of swelling and paleness of kidneys and distention of tubules with urates. There is an interstitial nephritis and degeneration of tubular epithelium. Although certain strains of IB virus do cause urolithiasis, such condition has not been seen in this area.

Like ND the incidence of Infectious Bursal Disease is quite high since the last few years. Though variant strain of IBD virus has not been isolated but the whole pathognomosis of the disease has greatly changed. Various outbreaks have been recorded as late as 14 weeks in young pullets and as early as three weeks in broilers. There is usually an enlargement of the bursa of fabricius while the kidneys are swollen with distended tubules, along with haemorrhages in kidney tissue. There seems a mass failure of vaccination and vaccines in controlling the incidence of IBD due to various reasons. Haemorrhagic lesions in kidneys are seen in both vaccinated and non-vaccinated birds but some researchers believe that Hydropericardium autogenous vaccine from the liver filtrate and the presence of adenoviruses is responsible for changing the disease scenario.

Kidney lesions are being so frequently observed that some line of treatment is being suggested by some pathologists like dosing birds with urinary antiseptics and diuretics as a routine to alleviate the blockage and reduce mortality. However, in view of the worsening disease situation in the country, a disease monitoring and control committee of the avian pathologist and poultry experts was recently held in Islamabad and suggested a countrywide computerised epidemiological survey in order to lay down a uniform policy for disease control for which a chain of disease diagnostics and control laboratories has been proposed. — Dr A.A. Qureshi, K&N’s Poultry Diagnostic & Research Institute, Karachi.