Studies on The Incidence of Pathological Conditions In Caprine Kidney

Thesis
Submitted to Magadh University in Partial Fulfilment of the Requirements for the Degree of M. Sc. (Vet.) in PATHOLOGY

BY
B.M. DUTTA,
Post-Graduate Department of Pathology
Bihar Veterinary College, Patna,
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November, 1966.
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This is to certify that the entire work presented in the Thesis entitled "Studies on The Incidence of Pathological Conditions In Caprine Kidney" is the bonafide work of Shri B.M. Dutta, a candidate for the degree of M.Sc. (Vet.) with Pathology as his major subject, which was carried out under my supervision and guidance.

( P.B. Kappuswamy )
1/12/66
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( B.M. Dutta )
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INTRODUCTION.

The study is a novel aspect of physiological chemistry. The necessary steps in the efficiency that while the blood in the body passes through the lungs to oxygenate the arteries. It is essential that all of the oxygen passed to each part of the body should be used as efficiently as possible. In addition to this, the food is digested by the stomach and further passed through the small and large bowel. The bowels are eliminated every few hours. The body is maintained through the nourishment of food. Thus, each moment of existence is a survival process.
INTRODUCTION

The kidney is a paired organ of great anatomical complexity. Its vascular supply is so efficient that whole of the blood in the body passes through the kidney in every few minutes. It is estimated that 20% of the cardiac output at rest goes to the kidneys despite the fact that these organs comprise less than 0.3% of the total body weight (Herbut - 1959).

Substances which are excess or foreign are carried to the kidney by the circulation of blood and efficiently excreted by the filtering beds — glomeruli. In addition to this — tubular parts of the nephrons performing selective reabsorption of water, glucose, chloride, sodium and other substances, escaping through the glomeruli which were useful to the body and unwanted substances and waste metabolic products escape from the body in the form of urine. Thus vital responsibility of maintaining the body's internal environment falls mainly on kidneys and they perform it successfully by regulating normal content of water, protein and salts in the blood, cells and
intra-cellular fluid which must be maintained constant for the normal activity of the body systems.

Any toxic agent in the circulation whether vegetables, minerals or microbial origin while passing through the kidneys produces considerable damage to renal structures. In specific diseases like anthrax, stangles, tetanus or tuberculosis, the organisms lodge themselves either in glomeruli or interlobular blood vessels producing inflammatory condition — the nephritis. While toxic substances as turpentine, mercury, lead, arsenic or sulphanamides show particular affinity for renal tubules resulting in destruction of tubular epithelium. The functional interdependence plays an important role in kidney diseases. If a portion of a nephron is destroyed, the remaining portions usually undergoes atrophy. So primary glomerular disease may ultimately effect both the tubules and interstitial tissue, and tubular disease in reverse may affect the glomeruli, interstitial tissues and vascular alterations resulting in renal insufficiency exhibiting clinically the alterations in the composition of urine, blood and severe symptoms of ill health leading to death.
Literature on pathological condition of various diseases of kidney among the domestic animals particularly in goats is quite scanty.

The goat has been appropriately termed "The poor man's cow" and certainly no better designation could be found to express the position of this useful creature amongst the domestic animals. It is very valuable for its manure, milk, fleece, guts, hide and meat. Among the Indian goats some breeds like Jamunapari, Beetal and Barbari are well known for their milk character. The annual production of milk in India is 560 million maunds of which goat milk comprises about 15 million maunds (Randhawa - 1962). On national level there are over 80 million goats in India yielding about 1,60,000 tons of meat annually (Randhawa - 1962). Goat meat is relished more than mutton. The value of the meat produced annually in this country has been estimated at 30.5 crores at the average rate of Rs 70.00 a maund (Lall - 1953).

Goat is very prolific producing twins or even triplets twice in 14 months. The rapid growth, quick maturity and high prolificity brings the goat to the list of the economical enterprises earning a good devident to the farmers and the goat breeders.
In this present moment India faces great problems primarily due to population explosion resulting in increased scarcity for foods and secondly constant threatening by our War-mad neighbours "JAI JAMAN JAI KISHAN" the bold and timely slogan of Late Prime Minister Lal Bahadur Sashtri shows us right path to meet these challenges. We are therefore now more concerned with greater production in all sphere of Agriculture and Livestock industry. The problem of scarcity can be overcome through increased stress on scientific farming and livestock development.

Among all livestock, goat though a much neglected animal, occupies a remarkable place as it is one of the source of best quality milk required by young generation and its meat is one of our principal source of protein diet which is essentially required for the maintenance of our nations' health, to check the enemy in front and campaign for freedom fight against hunger.

The present problem, therefore, was undertaken with a view to study the pathological conditions of the kidneys in goats so as to enable evolving of suitable control measures against the same for the proper development of goat husbandry.

*****
REVIEW OF LITERATURE.
ANOMALIES IN DEVELOPMENT

Jubb and Kennedy (1939) described complete aplasia of one or both kidneys rarely observed and occurs usually as a part of more generalised malformation associated with the anomalies of the female genitalia. Unilateral aplasia was found usually affecting the left kidney than the right. The corresponding ureter showed either complete absence or hypoplastic in a small mass of connective tissue instead of kidney. Vymetal (1965) reported the presence of only one kidney weighing 1.5-2 times more than normal in 8 Beagles out of 770 autopsy cases. Robbins (1965) found same condition in 2 Beagles. The single kidney present was approximately double the normal size. In a Bitch uterus of the affected side was abnormal.

Krock (1957) reported development anomaly and renal cortical hypoplasia in 40 autopsy cases of cocker spaniel dogs. He suggested the heredity factor as etiological cause and renal changes are exhibited by cortical hypoplasia including secondary hyper-parathyroid and increased renal insufficiency. Pun (1961) observed hereditary kidney hypoplasia in Brownleghorn caused by a simple recessive gene of variable penetration and expressiveness. The lesions were found restricted to anteroposterior surface due to improper development of the metanephric diverticulum. Cordes and
Dodd (1965) studied bilateral renal hypoplasia in 19 of 72 piglets sired by a large white boar. 12 affected piglets died at or soon after birth and 7 died between the ages of 11 and 69 days. Inheritance by a simple autosomal recessive factor was suggested responsible by the found evidences. Hypoplasia and faetal lobulation were present and microscopically poor development of the collecting tubules were observed and concluded the basis defect was the failure of development of mesonephric mesenchyme.

Jubb and Kennedy (1963) found malpositions of the kidney was a caudal displacement in the pelvic or inguinal position, were more frequently encountered in pigs than in other species. The renal arteries are originated close to the bifurcation of aorta of from iliacs. The ureter was short thereby predisposed to hydronephrosis and pyelonephritis.

Story (1943) reported an incidence of horse-shoe kidney in a domestic cat.

**Cysts of the Kidney**

Gaus (1937) reported cystic condition in the kidney and liver of a calf. Cysts were found sterile on bacteriological examination. Maqsood (1944) found a sterile echinoceous cyst in the cortex of the right kidney in a cow and the cyst was of the pigeon's egg size. Marathe (1952) described dystokia in a buffalo due to congenital polycytic kidney of the foetus. Groth and Rosdail (1935) observed the occurrence of multiple cysts in the liver and kidneys of three piglets died when 10 days old. The
condition was congenital and apparently hereditary. Blanc and Ascione (1961) studied cystic kidneys in three groups of guineapigs and reported the incidence as 19 in 1182, 28 in 500 and 7 in 91 respectively. Bartar (1961) examined a congenital cystic kidney in a still born (36 weeks gestation) and concluded that though minor changes were observed in renal corpuscles, no cyst could be demonstrated and attributed that they arise by dilatation of the various parts of the formed tubules of the nephron. Fox (1964) studied five cases characterised by absence of one and cystic degeneration of the remaining kidney in Beagles. Breeding record suggested that the condition was hereditary resulting in early death from renal failure. Kardevan (1964) observed the presence of numerous minute cysts in the perirenal area of the renal cortex and kidneys were slightly enlarged and pale brown yellow in colour. He found Bowman's capsules were enlarged, containing eosinophilic material and atrophied glomeruli. The deeper layer of the cortex contained slightly hypertrophic intact glomeruli. The absence of efferent canals in the affected layer of cortex was suggested a congenital abnormality brought about by inadequate unification of Malphigian bodies and canaliculi in younger peripheral area of the cortex during embryonic development.

**Pigments in Kidney**: Bos (1936) reported abnormal pigmentation in the kidneys and adrenal glands of two bovines. Brownish
pigments were detected in epithelial cells of the convoluted tubules. But narrow loops of Henle and glomeruli showed little or complete absence of pigment granules. A thin layer of mucous containing pigments was found covering the renal pelvis. Micro-chemical reactions showed the pigment was melanin and present in connective tissue of the cortical substance and in the cells of medulla. He suggested that some relation might be existing between the activity of the adrenal glands and the formation of skin pigments. Corsico (1953, 1955) described the deposition of xanthine - a brownish red pigment in both the kidneys of an apparently healthy calf. He further reported the presence of the accumulating crystals of a purine body - xanthine was in the collecting and papillary tubules of two slaughtered calves. Orlop (1956) studied 2.3% bovine kidneys had dark brown discolouration of the cortex. Pigments were present in the form of granules of varying sizes in the convoluted tubules. The pigment was proved to be lipofuscin and also found in renal and mesentric lymph nodes. Al Zahawi (1957) reported the incidence of renal cortical siderosis in goats grazing in areas rich in copper and iron, but deficient in cobalt. Spontaneous bilateral siderosis of the renal cortex was observed but not in other organs. The deposition of ferric compounds in the proximal convoluted tubules did not manifest any significant changes except the thickening of basement membrane. Light (1960) reported a condition "Cloisoune" kidney frequently encountered in white Angora goats. It resembled a condition
previously described in Iraq, probably associated with cobalt deficiency. It was present in 36 of 412 castrated male goats. In 9 cases lesions were severe, in 14 moderate and in 73 slight characterised by darkbrown pigmentation, thickening of the basement membrane of the proximal convoluted tubules and haemosiderosis deposition of renal cortex. Thompson (1961) further studied the chemical composition of brown pigment associated with "Cloiseune" kidney and found the presence of inorganic mineral substances not of iron or calcium salt but of significant amount of vicinal glycolic groups and demonstrable quantities of cysteine, cystine, tyrosine and amino groups. Jubb and Kennedy (1963) cited congenital porphyria- a brownish pigmentation affecting the renal cortex of cattle and swine. Pigments were present in the tubular epithelium and interstitial tissue and detected in the urine. The urine and tissue were fluoresced blue green in the presence of ultra violet light.

INFILTRATIVE AND DEGENERATIVE CHANGES :-

Dible and Hay (1940) produced fatty changes in rabbit kidney by starvation. The fat deposition appeared in the wide parts of Henle's loop specially in the descending limbs. They considered the condition as infiltration not fat phenorosis. Lobpen (1955) studied the occurrence and distribution of intracellular lipid in the kidneys of 55 adult cats and 32 kittens. A marked increase in the deposition of intracellular kidney lipoids was found to be associated with loss of sexual function.
in males and with intercal phase of oestrous cycle in females. Baumann (1956) reported the occurrence of fatty calcareous infarcts in kidneys of dogs and cats. It was found in Henle's loop as a result of faulty secretion during interstitial nephritis. Pellegrini and Pellegrini (1960) observed a progressive fatty changes in the epithelium of nephrons particularly in the principal segments resulting fatty infiltration of renal lobes during advance pregnancy.

Hjarce (1933) recorded 64 cases of amyloidosis in 8 horses, 32 cattle, 11 dogs, 8 fowls, 2 ducks and 1 turkey. Anderson (1936) reported large white kidney in cattle was due to amyloid degeneration. The amyloid deposition very often masked by other alterations. Histopathologically amyloids were found in the glomeruli, vasa afferentiae and vasa rectae in the medullary substance. According to him these changes were primary stages and termed them as pre-amyloid or archamyloid substances. He claimed experimental generalised amyloidosis could be produced by injecting fowl albumin in a cow. Dunn (1944) studied the frequent occurrence of spontaneous amyloidosis and renal disorder in certain inbred mice. Amyloid deposits in the intertubular interstitial tissue of the papilla resulting in occlusion of collecting tubules and urinary obstruction was recorded in 4 mice.

Sheehan and Davis (1960) observed transient ischaemia of the kidney causing sufficient damage of the tubules resulting in hyaline droplets or colloid degeneration.
in the proximal tubules and colloid casts in the distal
segments.

Pasqua (1938) found an ossified plate of connective
tissue in the medullary boundary of renal pelvis in a cow
showed enlarged kidneys and ureters. Lowenhaupt and
Greenberg (1945) stated chloride deficient diet causing
precipitation of calcium in the lumen of the collecting
tubules which were ultimately blocked and hydronephrosis
developed. Haston et al (1945) observed calcification in
lungs, aorta, arteries, heart, kidney and gonads of cats
which were fed cystine and protein deficient diet. Dick
and Prior (1951) found magnesium deficiency constantly
produced calcification in kidneys without inflammatory
reaction of lower nephrons in experimental rats. They also
observed calcification at the cortico-medullary junction
in chloride deficient rats. Selye and Bois (1956)
experimentally produced calcification in the intermediate
zone of one kidney of adrenalectomised rats injected with
deoxytisone acetate and substituting 1% sodium phosphate
solution instead of drinking water. Kretzschmar (1956)
detected metastatic calcification in pulmonary artery, 
aorta, myocardium, kidneys, lungs, sole pads and joint
of 5 dogs which were suffered from chronic contracted
kidneys.

Demrich (1958) found metastatic calcification in
the kidneys, trachea, epiglottis, endocardium, larynx and
aorta in 16 of 39 dogs. Kidneys were showing calcium
deposition in glomeruli, Bowman's capsule, the capsular
space, basement membranes and interstitial tissues. Guarda (1958) observed metastatic calcification in the left kidney of a cow suffered from pyelonephritis. The renal pelvis was occupied by a spongy bone tissue measuring 11 x 8 cm. Carone et al (1960) found structural alteration in ascending limb of Henle, distal convoluted and collecting tubules of kidney in hypercalcemic dogs which were previously injected parathyroid extract. Anderson et al (1961) reported excess of calcium in urine often produced calcium deposition in renal parenchyma. They conclude the condition of renal calcification and calculus formation was due to excess calcium in blood and oxalate in urine. Renaud (1962) observed 90% of female breeding rats were showed interstitial calcification in kidneys. Basement membrane of the tubules were found the early sites of calcium deposition.

CIRCULATORY DISTURBANCES AND DISEASES OF BLOOD VESSELS

Intyra et al (1943) stated arterial hyperaemia in kidneys was associated with excretion of irritant, toxic agents, bacterial toxins and diseases. Smith and Jones (1957) described active hyperaemia as a feature of acute inflammation and passive congestion as a part of generalised passive congestion. The congestion was prominent in medulla due to the presence of venulae rectae which were running in groups.

Caissar (1943) studied petechial haemorrhages in 82 slaughtered and 28 healthy calves. Tests for bacterial or toxic etiology were found negative. The Petechial
haemorrhages were also observed in calves having traumatic injury to the spleen. According to him petechial haemorrhages often encountered in salmonella infection. Di Domizio (1962) reported 24 cattle fed mainly on sorrel showing petechial haemorrhages in the kidneys. Abramov (1962) examined 17 calves died from toxic dyspepsia showing the condition in renal cortex but could not able to isolate salmonella or coliform bacteria.

Larsen et al (1962) reported a case of perirenal oedema in pig. No general reduction of total serum protein demonstrated though decrease of albumin globulin ratio and slight increase alpha-globulin was recorded.

Gaven and Kaufman (1956) experimentally produced infarction in 36 albino male rats. Left renal artery was exposed and ligated. The kidney was swollen and turned bluish hue within 30 seconds. The right renal artery left intact so right kidney was used as control. The animals were killed in groups of four at intervals of 1,2,4,8,12,18,22,36 and 48 hours after ligation. Histopathologically they observed in case of 1 hour after ligation, hyperemia at the corticomedullary junction, cloudy swelling, slight hydropic degeneration and necrosis in the occasional cells. These degenerative changes rapidly progressed after four hours of ligation resulting in necrosis of tubular epithelium. The tubules which were immediate contact with renal cortex preserved, while the rearing cells necrosed. Mora and Delgındice (1958) reported renal infarcts in slaughtered animals. They stated that infarcts were found common in cattle and their etiology was obscure. Alexander et al (1961)
produced infarction in left kidney in a group of rabbits by injecting a variety of particulate substances into renal artery. The animals were killed at intervals of 1 hour to 150 days. A temporary elevation of blood pressure was found in most of the rabbits whether opposite kidney was removed or not. There was no correlation between the size and the number of infarction and elivation of blood pressure. The rise of blood pressure was considered due to the presence of necrotic materials. Sheehan and Davies (1959) noticed occlusion of main renal artery left open colateral arterial supply only to the kidney and area of healthy tissue varies considerably from case to case. The remaining patches of kidney undergo infarction. Blood gradually oozed into the infarct from surrounding vascular supply resulting in the production of red infarcts.

Simon and Hepler (1945) experimentally produced selective injury to the various parts of the nephron by injecting various chemicals. They concluded potassium dichromate produced necrosis of the first part, mercury bi-chloride and uranyl nitrate caused necrosis of the terminal part of the proximal convoluted tubules. Innes (1950) observed pancreatic sclerosis in sheep due to diabetogenic a action of alloxan which could not prevent even after the ligation of pancreatic duct. The animal showed severe renal cortical necrosis after death. Blainey (1962) reported four cases of patchy diffuse necrosis of renal cortex associated with sudden onset anuria. The lesions were considered as
sequelae of extreme stasis of blood circulation in renal cortex following dilatation of the capillaries of the glomeruli. Thal (1955) experimentally produced renal necrosis in rabbits by injecting staphylococcal toxin. Animals died 9 hours after receiving injection exhibiting dull, greyish and necrotic changes in the medulla. The cells of the glomeruli, convoluted tubules, wall of blood vessels and interstitial tissues showed cytoplasmic coagulation and pyknotic nuclei. Hewitt (1956) reported an outbreak of renal tubular necrosis in male mice resulting from accidental exposure to chloroform. Paronetto (1965) studied the presence of gammaglobulin, complement, fibrinogen and albumin in the necrosed area in 11 of the 14 cases. Lucke and Hunt (1965) reported the incidence of papillary necrosis in 6 cats. The kidneys were nodular in appearance and the lesion varied from small haemorrhages to complete separation. Interstitial collections of lymphocytes were observed in areas of increased intertubular fibrosis with atrophy and dilatation. Tubular loss was present in the cortex particularly just below the cortico-medullary junction.

Bentozon and Mihajlovic (1959) reported an incidence of chronic bovine haematuria. The source of casual agent was considered food or soil and digestive tract as route of entry. Farse (1964) observed massive intravascular haemolysis in complete renal failure. The condition is known as "Crush kidney". Two sheep died of renal failure with chronic copper poisoning and two dogs with renal failure
of unknown cause but suspected for toxic etiology exhibiting haematuric condition.

Rossi and Lamy (1947) reported uraemic condition in calves caused by potassium nitrate poisoning. The animals showed increased excitement followed by torpor, coma and death. On analysis presence of uric acid was detected in blood. Schiefer (1960) described the similar condition in 16 dogs suffering from chronic nephritis. The significant changes were pulmonary oedema, catarrhal purulent bronchitis, fibrinous deposition and pseudohyaline membranes in alveoli.

Artioli (1949) studied an incidence of primary arteriosclerosis in the kidney of a horse. Grossly kidneys were normal but microscopic examination showed the presence of increased chromatrophic substances, hyaline degeneration, elastic hyperplasia in arteries and hyalinization of the external coat of Bowman’s capsule.

GLomerulonephritis

Wester (1933) considered chronic glomerulonephritis in bovines occurring bilaterally caused by bacterial toxin. Langhan and Hallman (1941) studied 236 cases of nephritis in all species of domestic animals. They could classify only 7 cases of glomerulonephritis in different species. The primary lesions in glomeruli were characterised by thickening of the basement membranes, increased amount of connective tissue and hyalinization. Nilsson (1945) experimentally produced kidney diseases in 14 dogs by
injecting nephrotoxin from rabbits. In seven dogs acute glomerulonephritis were developed with or without exhibiting renal disorder. Seegal and Loeb (1946) produced chronic glomerulonephritis in 13 of 32 rats by injecting rabbit anti-rat placenta serum. Margolis et al (1947) established the same in 4 of 9 dogs by repeated injections of Brucella suis. Dog 1 which was received 21 repeated injections died on 198th day showing severe symptoms of ill health. Dog 4 was also received 22 injections and died on 261st day. Active inflammatory lesions were present in glomeruli superimposed on chronic glomerular scarring. Dog 2 on 37th weeks became severely ill after 35 intravenous injections. The animal returned to clinical recovery when injections were discontinued and killed on 437th day. Dog 3 was given 39 intraperitoneal injections within a period of 43 weeks and was killed on 461st day. Both the dogs were exhibiting subacute type of glomerulonephritis.

Hartford (1955) reported focal and diffuse type of glomerular nephritis in choline deficient rats similar to condition found in diabetic men. The conditions arose due to the arrest of multiple fat emboli of hepatic origin in the glomerular capillaries. Rerk (1955) observed acute serofibrinous type of glomerulonephritis in pig associated with specific diseases as swine fever, swine erysipelas and streptococcal infection. Lenone (1957) described a case of haemorrhagic glomerulonephritis in cow. He concluded the etiology was of allergic origin. Siller (1959) reported 145 cases
nephritis of 436 autopsy cases in fowl. Acute glomerulonephritis similar to acute diffuse proliferative glomerulonephritis found in mammalian, while chronic lesions were comparable to the human sub acute form recorded in 31 cases. Marcati and Testoni (1962) studied focal glomerulonephritis associated with chronic tuberculosis in cattle. In the early stages the changes were manifested by degeneration and necrosis of the capillary loops with varying degree of endothelial proliferation in the glomeruli while cellular infiltration of the adjacent periglomerular and peritubular interstices found in the chronic stages.

Wettimuny (1963) studied and classified the incidence of 178 cases of nephritis as follows:

- Glomerulonephritis: 11%
- Interstitial Nephritis: 7%
- Pyelonephritis: 10%
- Embolic or Pyaemic Nephritis: 5%

**NEPHROSIS**

The term nephrosis was first used by Mallar (1904) cited by Vollhard, denoting changes of a purely degenerative nature in the kidneys, or those changes in which inflammation played a doubtful part. But with the recognition of an increasing variety of renal lesions of obscure nature or pathogenesis, the term has come to be applied to a rather heterogeneous collection of changes ranging from cloudy swelling to necrosis. Neiberle (1922) cited by Udall (1937) classified all degenerative changes resulting from metallic poison like mercury under the term nephrosis. The degenerative changes
described were cloudy swelling of the renal epithelium, hyaline degeneration, amyloidosis and necrosis. He suggested all inflammatory changes characterised by exudative, infiltrative and proliferative changes should be treated as nephritis. Bloom (1939) described nephrosis as non-inflammatory condition of the kidney. He classified nephrosis as acute nephrosis (mild, moderate and severe) and chronic or amyloid nephrosis.

Saxton and Kimball (1940) found chronic nephrosis occurring more frequently in rats which are fed on high protein diet. Ottosen (1949) reported a calf of one month old that suffered from diarrhoea and was treated with sulphpathiazole twice daily for 5 days and at 10th day the animal was sacrificed and kidney lesion was characterised by development of calculi composed of acetylsulphathiazole in the tubules responsible for the development of nephrosis. French (1950) observed glomerulonephrosis in toxic oliguria, anuria and lower nephron nephrosis. Welt and Peters (1951) stated the toxic effects of sulphanamides, carbontetrachloride and mercury salts producing massive destruction, perfusion reaction, circulatory collapse and finally the development of lower nephron nephrosis. They suggested factors responsible for tubular injury as follows:

1. Some toxic agent for renal epithelium;
2. Anoxia of renal tissue;
3. Increased reabsorption of water from tubules.
raised the concentration of urine more which finally
destroy the tubular epithelium. Stammle (1956) experiement-
ally produced necrotizing nephrosis in rats by injecting
mercuric chloride in rats. Pattanaik and Kuppuswamy while
studying the effects of experimental mercurial poisoning in
rats observed that in low dose there was varying degree of
degeneration in the cells of the proximal convoluted tubules.
In high dose group the degenerations were more pronounced in
the initial and middle third of proximal convoluted tubules
of the outer cortex instead of inner cortex as the time
interval increased.

Stammle and Harboff (1956) established
experimentally acute nephrosis by injecting massive doses of
neomycin and viomycin in rats. They conclude calcification
was less in comparison to mercuric chloride and epithelium
of the damaged renal tubules showed regenerative tendency.
Dalham and Friberg (1967) stated tubular damage was recorded
in rats caused by cadmium sulphate.

Bond and Murrey (1962) reported that the condition
of kidneys were dark, shrunken and nodulated in chronic
flourosis and degenerative changes more pronounced in tubular
epithelium. Cater and Peters (1961) recorded regenerative
changes in the mitochondria in the cell of the proximal
convoluted tubules and distension of Bowman’s capsule with
reported salicylamide is well tolerated in almost all species
of animal except in cat where the symptom was characterised
by acute oliguric nephritis.
INTERSTITIAL NEPHRITIS

Smith (1925) claimed to be the first to record focal interstitial nephritis of bovines in 1917. Sakauchi (1917) quoted by Smith (1925) described interstitial nephritis in 68 bovine kidneys. Pfenninger and Krupski (1923) cited by Smith (1925) produced renal changes which were identical with white spotted kidneys of bovines by repeated injections of Bacillus Cali and they described the lesion as an embolic interstitial focal nephritis. Lubke (1934) infected two calves with Brucella abortus. First calf which was injected intravenously just after birth exhibited foci of cellular infiltration of the interstitial tissue with endothelial cells, monocytes and lymphocytes around the small arteries. Second calf which was injected intramuscularly when seven days old recovered but when it was killed at the age of two and a half months lesions identical with that of the previous one except they appeared more circumscribed were noticed. Magnusson (1934) quoted the statistics worked out by Bergman. According to reports incidence of white spotted kidneys in new born calves and fat calves was 0.02% and 2.03% respectively. As attempts for isolation
of *Brucella abortus* from the failed, lesion he did not consider the organism responsible for white spotted kidney. Wester (1935) stated in primary diffuse interstitial nephritis both kidneys were very small and hard due to increased interstitial connective tissues.

Van Vloten (1936) described large white kidney in 44 cattle and 4 calves. The kidneys were exhibiting hyaline-amyloid degeneration of the glomeruli associated with primary lesion interstitial nephritis, which started developing before the degenerative changes.

Moore and Hallman (1936) experimentally produced white spotted kidney in calves fed on Vitamin A deficient diet.

Kurtze (1936) failed to isolate *Brucella abortus* from 14 pairs of white spotted kidneys. Netzger et al (1937) described the incidence of pyelonephritis, sub-acute and chronic interstitial nephritis in thirteen cattle exhibiting albuminurea from calf-hood development. They also isolated *Bacillus Cali*, non-haemolytic streptococci and
Stanhylococci from these cases. Wiidik (1937) examined 90 cases of white spotted kidney in three-six weeks old calves. Bacteriological examination was found negative. He concluded the etiology not due to bacterial infection but of toxic origin. Hoppe (1938) reported Brucella infected foetus was exhibited non-purulent type of interstitial nephritis. There was increased proliferation of capillary endothelial cells and infiltration of lymphocytes and histiocytes in cortical region. Heinan (1958) tried for isolation of Brucella abortus from nine calves with white spotted kidneys, but failed. Hutyra et al (1946) reported white spotted kidneys found commonly in calves which were in the form of small grey patches and scars. They quoted Dornis according to him incidence of white kidneys was 2.5%.

Unger and Bernkopf (1947) studied the pathology of bovine leptospirosis in experimentally infected calves of ten-fourteen days old. Six calves developed fatal jaundice and died in between seventh and eleventh day. The kidneys of these animals exhibited degenerative changes of tubular epithelium
and interstitial nephritis with lymphocytic infiltration. They suggested it should not be confused with that of white spotted kidney as experimental animals never showed whitish patch of discolouration. Artioli and Zanacca (1949) studied ten cases of white spotted kidney in young calves. Histopathological examination exhibiting myeloid metaplasia with a tendency of erythroblastosis and megakaryocytosis. McIntyre Montgomery (1952) studied interstitial nephritis in *Leptospira Conicola* infection of dogs. Increased cellular infiltration localised in cortico-medullary junction was characteristic in acute stage while scarring and fibrosis observed in chronic stages. They concluded that for the development of granular contracted kidney the period required was about two years. Nielsen and Mosherry (1954) described an account of severe interstitial nephritis in a dog causing uraemia, acidosis and hyperphosphataemia.

Larsen and Tondering (1954) reported the incident of leucocytic interstitial nephritis after examining 101, 668 slaughtered pigs and concluded 0.13% animals suffering from nephritis resulted from faecal contamination of the vulva.
Haga et al. (1955) gave an account of 28 slaughtered pigs with interstitial nephritis due to haematogenous bacterial infection. Dhame (1955) reported shrunken kidneys of chronic interstitial nephritis in dogs.

The condition commencing as interstitial lymphohistiocytic nephritis which in turn became fibrosed. Further changes in the kidney were non-inflammatory degeneration, with tissue contraction, and restriction of blood supply caused by increased fibrous tissue.

Hofmann (1955) investigated focal interstitial nephritis in twenty-nine calves of from six weeks old. Acute form was exhibited by small whitish foci with increase lymphocytic infiltration. Subacute stage was characterised by larger foci with increase fibroblastosis but cellular infiltration was regressing. Few but large lesions with concave surface due to increased fibrous connective tissue proliferation were found in chronic stages. Stevens et al. (1956) experimentally produced interstitial nephritis in dog by repeated injections of lithium carmine and staphylococcus toxin.

Tsiroyannis (1957) observed white spotted kidneys in 1376 out of 45,000 slaughtered calves. He
examined 250 pairs and concluded that the lesions were produced by an allergen or allergens of unknown origin. Fubbrimann (1957) reported that the incidence of white spotted kidneys was 8% and more common in fat animals. According to him bacteria gain entrance in the kidney through the blood circulation from other loaded digestive tract.

Corsico (1960) noticed eosinophilic interstitial nephritis in pig and suggested that the lesion was produced by migrating helminth larvae.

Sastry (1961) experimentally produced interstitial nephritis by injecting Escherichia coli, proteus and Pseudomonas into the renal artery. Proteus in broth culture containing 0.1% agar considered to be most successful.

Burdin (1963) described the account of leptospirosis in 12 acute (4 bovine, 6 swine, and 2 ca prine) and 34 chronic (32 bovine, 1 ovine and 1 procine) cases. In acute cases spirochaetes were localised in ascending limb of Henle and distal convoluted tubules whereas in chronic cases leptospiroae were found along with syncytical giant cells in lower nephron. The authors found the presence of hyaline
cast in dilated descending Henle's loops only in leptospirosis. Iyer and Nanda (1965) reported the incidence of white spotted kidneys in goat. Among 21 animals (5.2%) showed lesions having considerable degree of variations. In some cases lesions were very few and showing focal type in distribution while in others entire cortex underneath the capsule was studded with spots. Characteristic changes in early lesions revealed light coloured radial striations in the cortex but in chronic stage lesions became reduced in size, having distinct margin and extended in the cortex as wedge shaped. Kidneys were usually affected indicating the source of infection through the blood stream. Microscopically lesions exhibiting acute congestion of the glomerular tufts, thickening of the basement membrane, degenerative changes of the Bowman's capsule and dilatation of the convoluted tubules. The tubular epithelium showing changes were varies from degeneration to necrosis moreover sometimes they overdilstended by the presence of granular debris, protein casts and blood pigments. Lymphocytes, plasma cells, few macrophages and occasionally mutrophiles were observed
in focal or diffuse type of infiltration throughout the interstitial tissue of the renal cortex and medulla. In chronic cases lymphoid cells were gathered into discrete foci in the interstitial tissue of the cortex and occasionally in the medulla resulting atrophy and even disappearance of the surrounding parenchymatous structures. Occasionally dilatation of the tubules, presence of hyaline casts and increased interstitial fibrosis were also observed. The authors stated that spirochaetes were detected on histo-pathological examination in 2 cases exhibiting acute and subacute types of interstitial nephritis. It is however to be noted that the organisms had not been isolated.

**PYELONEPHRITIS**

Hess (1888) cited by Udal (1947) described the lesions and symptoms of pyelonephritis and mentioned it occurs independently. Enderlein (1890) cited by Udal (1947) isolated an organism from the pus of a pyelonephritic cow and named the organism as *Bacillus renalis bovis* after artificial cultivation. Ernst (1905) cited by Feenstra and Throp (1946) described the condition in kidneys as small elongated
abscess around the vessels. He noted the organisms are scattered throughout the abscess irregularly. The organisms were found surrounding the Malphigian corpuscles, and also present in convoluted tubules with leucocytes and tissue debris in the disintegrated collecting tubules. Jones and Little (1925) reported specific infections cystitis and pyelonephritis in cows.

The infection was originated in pelvis and then extend into the adjacent structures. Wester (1933) described the condition in a cow as cystouratero—pyelonephritis which was ascending type of infection started as cystitis. The causal organism was corynebacterium renale. Boyd and Bishop (1937) stated pyelonephritis is always urogenous in origin and rarely haematogenous, and the condition in cattle and horse caused by primary organism bacterium while streptococcus was considered as secondary invader. Thorp et al. (1943) isolated corynebacterium renale and another deptheroid from six cases of pyelonephritis in cattle. The lesions were characterised by leucocytic infiltration, hyalinization and increased interstitial fibrosis.

Feenstra and Thorp (1946) who reported
the localization of organisms in pyelonephritis stated that it varies considerably from case to case. In milder form they were present in cellular debris of the kidney calyces, ureter and mucosa of urinary bladder whereas in severe cases they were found scattered throughout the kidney tubules and necrotic tubular epithelium. Morse and Robert (1949) isolated Corynebacterium renale and Bacterium Coli from the urine of pyelonephritic cow. On postmortem examination they recovered C. renale from left kidney and left urater while Bact. Coli were predominating in the bladder. They considered C. renale as primary and Bact. Coli as secondary organism. Feenstra et al (1949) experimentally produced pyelonephritis in rabbits by injecting Corynebacterium , renale,. Animals were died after the development of disease showing papillitis and pyelitis. Organisms were found in necrotic debris and pelvis. The lesions were similar to those found in necrotic debris and pelvis. The lesions were similar to those found in bovines and the condition was described as haematogenous origin. Navasquez 1961,1963) reported intravenous injections of coagulase positive staphylococci into the rabbit constantly produced pyelonephritis. The infection was descending type and
Main factors for localization of organisms in the kidney are coagulase production which subsequently producing coagulative necrosis and suppuration of renal parenchyma. These suppurative changes undergoes organization and replaced by fibrosis. Presence of perinephric abscesses and amyloid degeneration were considered as sequelae of pyelonephritis. He further described that pyelonephritis can be experimentally produced by inoculating *Bacterium coli* and *Pseudomonas procyanes* in those rabbits having pre-existing scar due to *staphylococcal* infection.

Biswal et al. (1953) isolated ten strains of *Diptheroids* which were differ from Corynebacterium renale in the biochemical properties. Dhanda and Das (1954) isolated *corynebacterium renale* from a cow exhibiting haematuric condition. They describe pyelonephritis for the first time in India.

Erlandeon et al. (1959) reported *Strep-tococcus faecalis* from a man urine when injected in mice produced pyelonephritis. The organisms present in the kidneys upto 152 days while from all other organs they were eliminated within 57 days. Corril and Navasques (1960) (1964) showed *staphylococci* under suitable controlled conditions can
produce predictable renal lesion in mice. The pathogenesis and evolution of the lesions were comparable with human pyelonephritis. They also found ability for the production of pyelonephris highest in Pseudomonas aeruginosa, in comparison to Escherichia coli and Proteus mirabilis, which gradually leadings to papillary necrosis, hydropnephrosis, pyonephrosis and cystitis. Soltys (1961) isolated Corynebacterium suis from the case of pyelonephritis usually associated with pregnancy or pururition in sows. The organisms were associated in pathological conditions only in sows but they were present in urine, penile sheath and semen of even apparently normal boars. Experimentally they found that in intra-renal inoculation organisms rapidly spread to the other uninoculated kidney and adjacent structures as well. Sanford et al (1962) reported usually aquired immunity plays an important role in many gram negative organism but in case of pyelonephritis conflicting results were obtained. They however found considerable resistance developed against haematogenous pyelonephritis in rats caused by Escherichia coli, klebsiella pneumonia or Proteus mirabilis.

Siller (1964) recorded 331 cases of pyelonephritis from 1631 autopsy cases of fowl of unknown etiology. The kidneys were greatly swollen ,
exhibiting an interstitial and intertubular accumulation of inflammatory cells, and dilated distal convoluted tubules in acute conditions. In chronic stages the lesions were interstitial mononuclear infiltration, hyperplasia of the tubular epithelium, interstitial fibrosis ending in a scar.
OTHER INFECTIONS

Jowett (1922) isolated Corynebacterium pyogenes from pyaemic nephritis in a cow responsible for the development multiple pidd-heed or millet seed size abscesses on the kidney surface. Iland (1951) described degenerative and necrotic changes in kidneys guineapigs when experimentally inoculated with human or bovine type of tubercule bacilli. Christiansen (1952) isolated a new species Eimeria Somateriae causing renal coccidiosis from 24 goslings, 4 swans and 20 common ducks. Casarosa (1953) encountered hepatic and renal lesions in 16 calves which were distinctly different from the tuberculus lesions, caused by the larvae of Necascaris vitulorum. Hartman (1961) demonstrated Kloaciella equi in the kidneys of 8 out of 14 Mexican donkeys. The stages of sporogony were quite identical to those found in equine species. Dekker and Schaaf (1962) isolated bovine type of tubercule bacilli from the urine of an emaciated circus camel which was exhibiting 6 mm. positive reaction to bovine tuberculin test. On autopsy, lesions were observed in kidneys and other organs. Chang Sue Kheng and Lim Sin Xin (1962) found Corynebacterium renale
associated with the development of pyelonephritis in buffaloes.

Neoplasms:

Hytyra et al (1948) mentioned adenoma; adeno-carcinoma; sarcoma and papillary tumours were frequently encountered in the kidneys of dog. Di Domizio (1948) observed the incidence of renal tumours in a dog, three in horses and twelve in cattle of which fourteen were malignant epitheliomas and remaining two were sarcoma. McClure and Ross (1951) described two cases of renal carcinoma where majority of viable cells contain one or more cytoplasmic inclusions which were exhibiting marked differences with known virus inclusions. After studying 81 intra-dominal tumours Flir (1952) recorded an adenoma of the left kidney in a ten year old dog and an adenocarcinoma of the left kidney in a five year old dog exhibiting metastasis in the renal nodes, right kidney, liver and lungs. A sarcoma in mare and a mixed renal tumour in a one year old rabbit also was recorded by him. Thompson et al (1959) observed acidophilic epithelial cells of liver and kidneys in 13 of 45 apparently healthy dogs. These inclusions exhibiting many characteristics differs from viral inclusions bodies and Thompson et al (1959) confirmed that the
development of inclusion bodies were due to altered protein metabolism within affected nuclei. Kasbohm and Lettow (1961) reported the incidence of twenty-nine tumours in liver, seven in spleen, three in kidneys, two in adrenals, fifteen in ovaries, six in uterus, two in bladder, one in intestine and one in stomach of dogs. Rosen et al. (1961) observed high frequency of renal tumours which were mainly cortical adenoma, cortical carcinoma and transitional cell carcinoma in irradiated rats. Lülliger (1962) experimentally produced atypical kidney lesions in poultry consisting of cystadenoma, cavernous haemangioendotheliomata, epithelial metaplasia and hyperplasia of tubules by transmission of an avian leucosis virus. Lombard (1962) stated an account of a pinkish white tumour on each ovary in a sow and metastatic changes were found in the kidneys and all lymph nodes in the abdominal cavity. Kilham (1962) found nuclear inclusions in the kidneys of apparently healthy wild rats. But he failed to isolate virous and concluded that these inclusion bodies were responsible for the development of a varieties renal tumours ranging from circumscribe tumours to invasive, metastasising carcinomas. Tolle and Schreier (1962) reported the incidence of kidney tumours in mice when injected with bovine leucocytic tissue. The tumours
which developed after 5–6 months at the site of inoculation were haemangioepitheliomas and raticular-cell neoplasia but there was no evidence of leucosis. Lawrence, J.A. (1965) recorded incidence of hygroma in pigs. The apparently normal kidney was lying within the cyst which was attached to the hilus but not associated with the obstruction of flow of urine.
MATERIALS AND METHODS.

(Additional text not visible in the image.)
In the present study the specimens of kidneys having gross lesions were collected from goats from different slaughter houses at Patna and suburbs, from Livestock Research Station, Patna where the goats were sacrificed for manufacture of Rinderpest Vaccine and from the autopsy cases performed in the Department of Pathology, Bihar Veterinary College, Patna. All these animals generally came from the plain districts of Bihar, belonging to different breeds and were of age groups varying from 1-3 years. The period of collection was from 15th March to 30th September, 1966.

Altogether 946 goats were examined and kidneys showing gross lesions were collected from 150 animals. These goats were maintained under different condition of feeding and management. The specimens were mostly collected from male goats.

Among the specimens collected gross lesions were encountered in different degrees. As such it was thought worthwhile to catagorise the specimens into different groups, so that a selective incidence of each pathological lesion could be accurately dealt with.
The different lesions thus encountered are tabulated as under:

<table>
<thead>
<tr>
<th>Gross pathology</th>
<th>Groups</th>
<th>No. of animals from which specimen collected</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>i. Pale and anaemic kidneys ... PA</td>
<td>23</td>
<td>15.33</td>
<td></td>
</tr>
<tr>
<td>ii. Petechial haemorrhagic kidneys ... PH</td>
<td>3</td>
<td>5.34</td>
<td></td>
</tr>
<tr>
<td>iii. Congested kidneys ... C</td>
<td>26</td>
<td>17.34</td>
<td></td>
</tr>
<tr>
<td>iv. White spotted kidneys ... WSK</td>
<td>43</td>
<td>22.66</td>
<td></td>
</tr>
<tr>
<td>v. Scarr and contracted kidneys ... KS</td>
<td>39</td>
<td>26.00</td>
<td></td>
</tr>
<tr>
<td>vi. Miscellaneous ... RM</td>
<td>11</td>
<td>7.33</td>
<td></td>
</tr>
<tr>
<td><strong>TOTAL :=</strong></td>
<td>150</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

After each collection, gross pathology of individual specimen was noted, marked with identifying number, gross photographs where necessary were taken and kidneys preserved in 10% formal saline solution before any autolytic or putrefactive change took place. Formal saline which is an ideal preservative as well as good fixative was used throughout the present study. It penetrates, kills, fixes and hardens the tissues quickly.
so that they can resist dehydration, clearing, staining and mounting during the process.

As a well fixed tissue is always essential for obtaining perfect tissue sections and satisfactory staining reactions, the preserved specimens were taken out and cut into small pieces measuring few millimeters according to the nature of the lesions and preservation was done for another 24 hours to ensure perfect fixation.

Fixed tissues were washed thoroughly in running tap-water for 24 hours, dehydrated in ascending grades of alcohol, cleared in several changes in xylol and then transferred to paraffin having 57°C, congealing point in paraffin bath which was maintained for constant temperature at 60°C, and three changes in melting paraffin were done for infiltration of paraffin into the tissues. Finally blocks were made by tissues embedding in paraffin.

Paraffin sections were cut 5-6 micron in thickness, shifted to paraffin bath and collected on previously cleaned slides. For sticking tissue section to the slides Mayer’s Egg albumin was used.

Tissue sections were finally stained with
Harris's alum haematoxylin and counter stained by 1% alcoholic eosin. For the identification and demonstration of special structures abnormally present or developed due to altered tissue reaction, the sections were stained with special stains viz.:

i) Van Gieson's stain (Mallory - 1942) for collagen fibres;

ii) MacCallum - Good pasture stain (Mallory - 1942) for bacteria in tissues;

iii) Ziehl Neelsen's stain (Mallory - 1942) for acid fast bacteria;

iv) Lievaditi's method (Mallory - 1942) for staining spirochaetes in blocks;

v) Gridley's stain (Gridly - 1953) for fungus;

vi) Gallego's ison Fuchsin stain (Lillie - 1948) for calcium and other structures;

and vii) Bernbold's Congo red method (Carleton and Drury - 1957) for staining of amyloid.

Finally all stained sections were mounted permanently in Diapex (Gurr's).
PRESENT OBSERVATIONS
**PRESENT OBSERVATIONS**

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**CYSTS IN THE KIDNEYS**

**Gross pathology**—A caenurus cyst measuring 7.6 cm. in diameter belonging to the family Taeniidae, containing fluid and developing scolices—about 21 in number, was found situated in between the renal cortical surface and capsule of left kidney (Fig. - 1). In another kidney KM - 7, one small cyst measuring 5 cm. in diameter containing clear fluid was found on renal surface. Its sagittal section showed, the accumulation of the fluid in the cortical structures. In another left kidney KM - 10 three small cysts of varying sizes from 3 - 7 mm. in diameter were encountered in the renal cortex.

**Histopathology**—In caenurus kidney no significant changes were observed as cystic development was restricted in between the renal cortex and its capsule, but, the presence of haemorrhages in the cortical structures and slight increase of leucocytic infiltration was observed. In KM - 7 renal corpuscular spaces and tubular lumens were overstretched by the pressure of the cystic fluid resulting in atrophy and degeneration of the glomeruli, Bowman’s capsular endothelium and tubular epithelium (Fig. - 2). In KM - 10 an area of coagulative necrosis and
increased lymphocytic infiltration adjacent to the atrophied and degenerated area, due to cystic development were observed.

**PIGMENTS IN THE KIDNEYS:**

_Gross pathology:_ Small elongated scarred areas were found in the kidneys K3 = 18 and K3 = 28. On sagittal section corticomedullary portion showed marked contraction; the renal pyramids were reaching almost at the level of the cortical surface and greyish black colour of the medulla was noticed.

_Histopathology:_ Chronic interstitial nephritis with increased fibroblastic development resulting in degeneration, atrophy and complete replacement of normal structures. Iron pigment hemosiderin varying from greyish to black-brown in colour was found around the distal convoluted tubules just below the capsular interstitiae. They were also present in the vascular lumen and surrounding area (Fig. - 3).

**INFILTERATION AND DEGENERATIONS:**

_Hyaline degeneration:_ The kidneys from 23 animals exhibited the presence of hyaline degeneration which was observed only on microscopic examination. The hyalinization was associated with 4 out of 6 groups of kidneys. The incidence of lesions were 2 in PA,
1 in C, 2 in WSK and 16 in K3 group respectively.

**Histopathology:** Small spherical pinkish homogenized bodies appeared in the convoluted and collecting tubules (Fig. - 4). The tubular epithelium was degenerated and atrophied due to increased fibrous connective tissue in pale anaemic kidneys - PA 4 and PA - 19. In congested kidney C - S hyalinization was observed in the descending and ascending parts of the loops of Henle and the collecting tubules in the medulla. The walls of the collecting tubules were found extremely haemorrhagic (Fig. - 5). In K3 - 2, K3 - 18 and K3 - 26 kidneys the lesions were chronic type of interstitial nephritis exhibiting increased fibroplasia producing atrophy, degeneration and disintegration of renal structures. The hyaline degeneration was showing considerable variation in size and distribution. They were present in the tubular lumen as small spherical bodies (Fig. - 6) but in places they completely occupied the whole space of the tubular lumens (Fig. - 7) and in more chronic lesions renal structures were found completely replaced by hyaline degeneration.

**Calcereous Infiltration:**

**Grosspathology:** Calcium deposition in the renal tissue was not detected on gross examination of
the kidneys K3 = 28 and K3 = 32 where the lesions were characterised by the formation of scarring areas in the renal cortex.

**Histopathology:** Calcium deposition appeared in the form of blue amorphus irregular bodies which was found in the spaces of the malpighian bodies, lumen of ascending tubules (Fig. - 9 and 10).

**CIRCULATORY DISTURBANCES AND DISEASES OF THE BLOOD VESSELS:**

**Pale and anaemic kidneys:**

**Grosspathology:** The kidneys from 23 animals showed abnormal paleness almost dull white in colour. Sagittal sections represented uniform paleness throughout the cortex but medullary portion varied considerably from dull grey to faint pink colour and extreme whiteness of the renal calyces was also observed.

**Histopathology:** Paranchymatus degenerations of the tubular epithelium, haemorrhages in the tubular lumen and interstitial tissue, increased leucocytic infiltration, and fibroblastic proliferation were the most common types of structural changes. In addition to these changes hyaline degeneration in the tubular lumen, coagulative necrosis and the renal cortical
structures, increased and thickening of the intima of the arterioles were encountered and are described separately under their respective headings.

**Peteochial haemorrhagic kidneys:**

*Gross pathology:* The presence of petechiae observed in some kidneys was less while in other cases they were numerous and uniformly distributed throughout the renal cortex. Cut surfaces exhibiting the haemorrhages were restricted to the renal cortex only and the medullary appeared almost normal.

*Histopathology:* Tubular lumens narrowed considerably due to swollen epithelium, atrophy of the tubules, haemorrhages, albuminous casts in the tubular spaces (Fig. - 11), interstitial tissue and increased lymphocytic infiltration were observed.

**Congested kidneys:**

*Gross pathology:* The colour of the congested kidneys varied considerably from light red to dark purplish (Fig. - 12). They were enlarged and cut surfaces bulged out from the capsules. The renal surfaces were found smooth and did not adhere to the capsules. The medullary portions were extremely dark
red and in places appeared dark brown in colour. Renal pyramids were exhibiting slight inflammation.

Histopathology: Clumps of red blood corpuscles in the spaces of the Malpighian bodies, tubular lumen and interstitiae of the medulla (Fig. - 13), inflammation of the glomeruli, increased neutrophilic infiltrations and the fibroblastic thickening of the Bowman's capsules were found. In C -10 (Fig. - 14 and 15) increased proliferation of fibrous connective tissues which were collagen showing positive for Van Gieson's stain started from the cortex and travelled irregularly into the medulla resulting in atrophy, degeneration and disappearance of glomeruli and tubular epithelium.

Infarction:

Grosspathology: Left kidney KM - 3 (Fig.-16) exhibiting an irregular but spherical dark greyish area which was slightly shrunken and found below the level of the renal surface. Cut surface presented a wedge shaped area of dead tissue, found originated from medulla gradually increasing in size as it reached the cortex. In another left kidney KM - 4 an irregular dark brown area of dead tissue was
found situated below the level of the renal surface.

**Histopathology:** - Stagnation of blood observed in the cortico-medullary portion of the dead tissue resulting in development of red infarction which appeared as homogenised mass (Fig. 17, 18). In many of the convoluted and collecting tubules adjacent to the area of infarction were found containing hyaline and albuminous casts (Fig. 19). The iron pigment hemosiderin was found distributed uniformly throughout the area of infarction originated from the haemolysis and disintegration of the stagnant red blood cells. Around the area of infarction coagulative necrosis was found starting developing.

**Macroscopy:** - Gross pathological observations in the pale and anaemic kidneys (PA - 9 and 15 small irregular areas which were found dull in comparison to the surrounding tissues. The corresponding medullary portions were exhibiting dull yellow to greyish in colour, swollen renal pyramids and gelatinized substance was present in the renal pelvis. In congested kidneys C - 22 a small circular dull yellowish area was found restricted to the cortex and medulla was found
engorged with blood. The scared kidneys were characterized by the presence of irregular areas. The kidney KM - 1 was attached firmly with the perirenal fat and exhibiting an area of depression which was dull white in colour. In KM - 2 capsule was found firmly attached to the renal cortex. In miscellaneous group another kidney KM - 5 showing a small circular area measuring about 8 mm. in diameter with an uneven surface which was dull greyish in colour and was visible even before decapsulation (Fig. - 20).

**Histopathology:** Most of the proximal and distal convoluted tubules were exhibiting coagulative necrosis which was patchy in distribution. Most of the tubules were retaining their structural outlines but individual cell could not be differentiated from each other as the whole structure appeared as strongly acidophilic homogenised mass. All the nuclei were showing pyknotic and karyolytic changes (Fig. - 21, 22 and 23). In many places corresponding glomeruli were exhibiting haemorrhages, increased endothelial proliferation, leucocytic infiltration, atrophy and even disintegration. In chronic lesion proliferation
of young fibroblast around the necrosed area was also observed.

**Arteriolar sclerosis:**

**Gross pathology:** The kidney PA - 10 which was very pale and exhibited slight irregular surface and in KS - 30 both the kidneys were showing chronic contraction.

**Histopathology:** Increased thickening of the intima and proliferation of the media was found in small arterioles in the cortex as well as medulla(Fig.-24).

**Glomerulonephritis:**

**Gross pathology:** The kidney C - 18 was marked swollen and dark reddish blue in colour not adherent to the capsule, which was removed easily. The medulla was exhibiting hyperaemic condition and pelvis was containing gelatinized substances.

**Histopathology:** The sub - capsular space was completely occupied by increased endothelial proliferation and leucocytic infiltration in the glomerular tufts (Fig. - 25 and 26). The corresponding proximal and distal convoluted tubules were showing nephrotic changes. Extensive haemorrhages were found in the tubular lumen and medullary interstitialae.
Crosspathology: The presence of a small circular whitish area showing considerable variation both in size - ranging from 2-5 mm. in diameter and position was found in one and often both the kidneys. The white spots did not rise above the level of the renal surface and on examination of sagittal cross sections, they appeared as dull greyish areas restricted only to cortex. In some other kidneys increased number of white spots were found present though size and nature of the lesions were similar. Numerous white spots were found uniformly distributed throughout the renal cortex visible through the capsule and affecting both the kidneys (Fig. 27 and 28) simultaneously. Sagittal section showed the lesions to be restricted to the cortex only and in medulla adjacent to the some of the cortical lesions, was exhibiting a dull yellowish area.

Medium sized white spots measuring 1 cm. in diameter similar to those of small white spots in other respect were found present in the kidneys from five animals (Fig. 29). Dull pale yellow
areas were found in the medulla in continuation of the cortical white spots. The renal pyramids appeared dull grey and slightly inflamed.

Large white spots measuring several centimeters near the posterior pole of the left kidney were recorded (Fig. - 30). The right kidney was also exhibiting the presence of two medium sized spots. The lesions were restricted to cortical portion only.

**Histopathology - Cloudy swelling and degenerative changes of the renal epithelia**: some of the tubules exhibiting dilatation and were lined by flattened cells, swelling of the tubular epithelium resulting in narrowing of the lumen was very common (Fig. - 31). The proximal convoluted tubules were mostly affected while distal convoluted and the loops of Henle showed only slight changes while collecting tubules exhibiting little or no change. Albuminuous and hyaline casts were present in some of the tubular lumens.

Though some glomeruli appeared normal many of them exhibited atrophy, degenerative changes, increased leucocytic infiltration, endothelial proliferation of the glomerular tufts. Many of the
glomeruli were haemorrhagic and showed the presence of foamy substances.

In most of the advanced lesions coagulative necrosis of the proximal and distal convoluted tubules were noticed frequently (Fig. - 32) Picketotic and karyolytic changes of the nuclei and the whole necrotic mass appeared as acidophilic homogenized mass.

**INTERSTITIAL NEPHRITIS**

Almost all the kidneys showed interesting and important lesion — grossly scar and microscopically interstitial nephritis. It has been endeavoured to describe these kidneys individually, to present a clear picture of various changes encountered in this group which covered a major part of the study.

**3.1**

**Grosspathology:** Right kidney showed an irregular scar on the dorsal surface which was smooth and did not adhere to the capsule. Cut surface exhibited a wedge shaped area of depression and did not extend beyond the cortical portion (Fig. - 42).

**Histopathology:** The lesion started from the outermost zone of the cortex and was found restricted there itself (Fig. - 35). Increased interstitial fibroplasia mainly of collagen fibres were detected on
on Van Gieson's staining. The glomeruli were exhibiting endothelial proliferation and atrophy. Bowman's capsules were thickened considerably. The tubular epithelium exhibiting degenerative and necrotic changes and hyaline casts were found present in tubular lumen. The small arterioles were showing increased vascular thickening and the infiltrative cells were lymphocytes.

**Gross pathology:** An irregular area of scarring was found in the lateral border of the right kidney.

**Histopathology:** Lesions were exhibiting several starting points from the outer most zone of the cortex and having patchy distribution. The capsule was found thickened considerably. Increased interstitial proliferation mainly of young fibroblast was observed. The Glomeruli and tubules were found considerably damaged. The infiltrative cells were lymphocytes (Fig. 40).

**Gross pathology:** The scarring area exhibiting irregular in outline but smooth surface and depth was more in comparison to others (Fig. 45). Cut surface exhibiting triangular areas in the cortical portion.
Medulla was slightly dark grey in colour and renal pyramids reached near the external border of the cortex.

**Histopathology:** Increased interstitial fibroplasia was found destroying the normal structure and lymphocytic infiltration in places was found scanty while in other places it was plenty forming a spherical mass.

**KS - 4**

**Grosspathology:** The scarring area somewhat uniform oval in shape and was situated on the lateral border.

**Histopathology:** Areas of scarring originating from several places and travelled irregularly in the cortex. Extreme lymphocytic infiltration with hyperchromatic nuclei was found (Fig. - 39).

**KS - 5**

**Grosspathology:** Right kidney showed the presence of two scarring areas. One almost oval but slightly irregular in outline while oblique linear appearance of the other was found. Sagittal section showed areas of scarred tissue distinctly dull in comparison to the surrounding healthy tissues (Fig. - 50).
Histopathology: The area of the lesion was very extensive. There was increased connective tissue deficient in collagen resulting in atrophy and degenerative changes of the glomeruli as well as tubular epithelium. Bowman's capsules were found much thickened. Hyaline degeneration was found in the space of Malpighian bodies and in the tubular lumen. Lymphocytes in places were diffusely sprinkled and resembling like that of lymphoma. Presence of plasma cells were also observed (Fig. - 36 and 57).

Gross pathology: An irregular area of scar was visible even before decapsulation present in the lateral border near the anterior pole. Typical wedge shaped scarred area was showing marked contraction.

Histopathology: An extensive area of interstitial fibroplasia mainly consisting of young fibroblasts originated from the margin of cortical region and extend into the medullary portion. Normal renal structures found completely damaged and infiltrating cells were found bizarre appearance. Glomerular and tubular destruction was very prominent. In places hyaline degeneration was found completely
replacing the glomeruli (Fig. - 47).

**Grosspathology:** Right kidney showed the presence of slightly elongated scar in the lateral border.

**Histopathology:** Triangular pyramid shaped lesions were found starting from several points. The renal capsule was showing increased thickening. Dense cellular infiltration mainly of lymphocyte was found in the tubular lumen and around the glomeruli (Fig -44).

**Grosspathology:** A small scarring area was found in the lateral border of the right kidney having circular outline with increased depth.

**Histopathology:** Triangular lesion was observed travelling up to the boundary zone. Most of the glomeruli were atrophied or showing complete absence. Infiltrating cells were lymphocytes and few plasma cells and they entered into the tubular lumen in such a manner that individual tubules could not be differentiated from the other (Fig. - 43).

**Grosspathology:** Right kidney was showing the presence of an irregular scar on the dorsal surface.
Histopathology: An extensive damage where proliferating cells showing bizarre appearance and hyper chromatic nuclei were found. Haemorrhages were present in glomeruli and tubular spaces. Around the scarring area the tubular structures were found necrotized.

Grosspathology: An area of scar which was of primitive type found on the dorsal surface.

Histopathology: Bowman's capsules thickened due to increased fibrous proliferation. The wall of the small arteries were showing increased thickening.

Grosspathology: Scarred area was found situated near the lateral border of the right kidney.

Histopathology: The area of the lesion was extensive damaging most of the glomeruli and convoluted tubules. The tubular epithelium showing hyperplastic changes. Two mild lesions started independently and the degree of damage by them was observed less extensive.

Grosspathology: Right kidney exhibiting a large irregular scarring area which was found on the lateral border.
Histopathology: The lesion was found extending upto medulla and tubular lumens containing hyaline and albuminous casts. Intratubular haemorrhages was present (Fig. - 63).

Histopathology: Circular scarring area with smooth surface was found in the lateral border.

Histopathology: Lymphocytic infiltration was very extensive in places as they appeared in diffused masses and arranged in an irregular manner (Fig. - 56).

Histopathology: Right kidney was showing an irregularly outlined but slight depressed lesion on the ventral surface (Fig. - 37).

Histopathology: Damage of the renal structures was found moderately less. The glomeruli and tubules were exhibiting normal and interstitial fibroplasia was less developed. Hyaline degeneration was present in the tubular lumen and mild lymphocytic infiltration was observed in vacant places (Fig. - 52 and 62).

Histopathology: Scarring area was present on
the dorsal surface.

**Histopathology:** Renal capsule was found thickened considerably and damage started from the capsular interstitia.

**Grosspathology:** Very faint irregular lesion was found on the dorsal surface of the right kidney (Fig. - 33).

**Histopathology:** The glomeruli and convoluted tubules were retaining their normal structure but some of the collecting tubules containing hyaline casts. Lymphocytic infiltration was moderate.

**Grosspathology:** Right kidney showing an irregular scarring area slightly shrunken on the dorsal surface while a circular depression measuring 5 mm. in diameter was present in the ventral surface (Fig. - 34).

**Histopathology:** Lesion was focal in distribution and less extensive.

**Grosspathology:** Dorsal surface of the right kidney was found representing an elongated scar.

**Histopathology:** Extensive proliferation of interstitial resulting in complete damage of renal structures and lymphocytic infiltration was not.
structures and lymphocytic infiltration was observed very predominating. Iron pigment hemosiderin were deposited in places and in some places coagulative necrosis was found.

**Grosspathology:** Small scarred area was found on the anterior pole of the ventral surface (Fig. - 41).

**Histopathology:** The lesion was spreading type and destructions were similar to the other.

**Grosspathology:** Two scars were present on the right kidney.

**Histopathology:** Interstitial fibrous proliferation was similar but in tubular portion mainly proximal and distal convoluted tubules were showing the presence of coagulative necrosis.

**Grosspathology:** Irregular wedge shaped scarring area was found on the lateral border.

**Histopathology:** Except mild focal interstitial
nephritis most of the renal structure were found normal.

**Grosspathology** - Irregular area of scarring was present on the ventral surface.

**Histopathology** - Fibrous proliferation and infiltration were found extensive. In the cortical region most of the malphigian corpuscles were exhibiting complete absence of glomeruli and containing albuminous casts (Fig. - 64). Haemorrhages were found in tubular lumen and interstitiae.

**Grosspathology** - Small less defined scarring area was found on the posterior pole of the dorsal surface.

**Histopathology** - Very aggressive type of lesions as cellular proliferation and infiltration was markedly present in irregular fashion which was destroying the normal structures in that area (Fig. - 55).

**Grosspathology** - Irregular depressed area of scar was found on the dorsal surface of the right kidney (Fig. - 50).
Histopathology:- Nature of the lesion and degree of damage was found similar to those in the group some of the glomeruli were exhibiting atrophy, degeneration and disappearance. Bowman's capsules thickened considerably due to increased fibrous connective tissue (Fig. - 61).

Grosspathology:- Two scars were situated one on the dorsal surface and the other ventrally near the pelvis of the right kidney.

Histopathology:- Glomerular endothelial proliferation with increased lymphocytosis and few plasma cells were found (Fig. - 59). Most of the tubular epithelium were exhibiting coagulative necrosis.

Grosspathology:- Right kidney was showing the presence of elongated and sharply oblique scarring area laterally.

Histopathology:- Extensive cortical damage and increased lymphocytosis which were present innumerably in the spaces of the malpighian bodies and the tubular lumen. Haemorrhages were found in the medullary interstitia.
Gross pathology: Left kidney characterised by the presence of a long oblique scar on the ruminal surface (Figure - 53).

Histopathology: Glomeruli were showing increased endothelial proliferation and cellular infiltration. Most of the tubules mainly proximal and distal convoluted tubules were necroosed.

Gross pathology: Left kidney exhibiting the presence of small scarring area on the ruminal surface. The cut surface showed the dark brown appearance of the wedge shaped area of scarred tissue.

Histopathology: Increased presence of collagen fibres positive for van Gieson's stain replacing the renal tubular epithelium which were showing atrophy and degeneration. Iron pigment hemosiderin was found distributed in the cortical portion and in or around the blood vessels (Fig. - 51 and 52).

Gross pathology: Right kidney was exhibiting irregular area of scar laterally.
Histopathology: The lesion was similar but infiltration of leucocytes and fibrous proliferation was found less extensive (Fig. = 43).

Grosspathology: Chronic contracted kidneys as both left and right kidneys were affected simultaneously. The surfaces of the kidneys were found uneven as a result of numerous whitish constrictions. The capsule was found adhering to the organ firmly and could be separated with difficulty. Cut surface presented the cortical as well as medullary portion showing extreme damage. The medulla was dark black in colour and degeneration of the renal pyramids, atrophy of the calyces and wall of the blood vessels were found thickened (Fig. = 54).

Histopathology: Renal capsule was showing extensive fibrous thickening. Increased fibrous connective tissue mainly of young fibroblast but in places matured collagen fibres also found present resulting in congestion, atrophy, degeneration and disappearance of glomeruli. Bowman’s capsules were thickened considerably, narrowing the sub capsular spaces and in many places containing hyaline casts. Tubular epithelium also exhibited degenerative changes, atrophy and disappearance. In places increased hyperplasia of the tubular epithelium
with hyperchromatid nuclei were developed as a result of compensatory activity. In some places coagulative necrosis of the tubules mainly convoluted and collecting tubules were seen. The cellular infiltration mainly of lymphocytes and few plasma cells were present in or around the Malpighian bodies, tubular lumens in such extensive manner that individual structures could not be differentiated from the others. The blood vessels and the small arterioles were characterised by the increased vascular thickening (Fig.-65).

**Grosspathology:** Small scarred area present on the dorsal surface of the right kidney.

**Histopathology:** Structural changes were found similar.

**Grosspathology:** Small area of scar was found on the lower part of the lateral surface of the right kidney.

**Histopathology:** The lesion started from the boundary zone of the cortex and damage was not extensive but definitely spreading type as it proceeded irregularly near the boundary zone.
**Grosspathology:** Small scar with irregular in outline was found on the ventral surface anteriorly.

**Histopathology:** Glomeruli were hyperplastic and covering the whole subcapsular spaces and respective tubules were exhibiting coagulative necrosis.

**Grosspathology:** The scarring area was present near the hilus on the dorsal surface.

**Histopathology:** Changes were found similar.

**Grosspathology:** The scarring depression was found near the pelvis.

**Histopathology:** Changes were found similar.

**Grosspathology:** Two scars were found one on dorsal side while other one was present ventrally.

**Histopathology:** The glomeruli and the tubules showing atrophy most of them were found retaining their normal structures.
Grosspathology: An oval shallow scar was found in left kidney (Fig. - 38).

Histopathology: Increased interstitial fibrous tissue resulting in atrophy, degeneration of the glomeruli, thickening of the Bowman’s capsule (Fig. - 60) and marked lymphocytic infiltration and hyperplastic changes of the tubular epithelium were found.

Grosspathology: Small irregular scar was found on the dorsal surface of the right kidneys.

Histopathology: Area of lesion was extensive. The renal capsule was found thickened considerably. Most of the renal structure in the scarring area damaged and hyaline casts were found in the spaces of the Mulpighian bodies.

Grosspathology: An oval scar with smooth surface and slightly irregular outline was present on the dorsal surface.

Histopathology: Increased lymphocytic infiltration was found gained their entrance into the
tubular lumen forming a homogenized mass. The tubular epitheliums were showing hyperplastic changes and atrophied as a result of increased interstitial fibrous connective tissue.

The kidneys KM - 8 and KM - 9 from miscellaneous group showing minute elevated area where capsule was found firmly attached. Cut surface showing granular area distinct from the surrounding tissue was present in the cortical portion. On histopathological examination focal and in places diffuse infiltration of leucocytes mainly of lymphocytes and few plasma cells were found in or around the Bowman's capsule and glomeruli. The tubular epithelium showing atrophy, degenerations and disappearance and their places were found filled by infiltrating lymphocytes. The proliferation of interstitial connective tissue mainly young fibroblasts were found in or around the damaged area.
DISCUSSION AND CONCLUSION

The present investigations were made with a view to study the possible etiology of various psychological conditions encountered in inpatient illness. It was to enable us to appraise the role of adaptive coping techniques against the role for the maintenance of psychosocial or other neurotic and psychotic reactions (clinical) factors.

Emphasis should be given to the study of different conditions in the context of the overall scope of the present study, however, an attempt to correlate the available literature and the findings with the above-mentioned conditions.

In the present study, a number of ecological factors were taken into account while investigating the effects of the different factors on the mental health and well-being of the patients. The exact correlation of the findings could not be determined due to the nature of the patient population and the variables involved. However, the findings revealed an association between the level of education and the psychological well-being of the patients. In the context of the present study and the results of the correlation of the variables of independence, it was observed that the level of education and the psychological well-being of the patients were significantly correlated. The findings of the present study are consistent with the findings of previous studies in the field of psychology.
DISCUSSION AND CONCLUSIONS.

The present investigations were made with a view to study the possible etiology of various pathological conditions encountered in caprine kidneys so as to enable evolving of suitable control measures against the same for the maintenance of good health of goat husbandry and thus ensure maximum financial return.

Experiments could not be conducted to find out the etiology of different conditions as the same were beyond the scope of the present study. However, an attempt is being made to discuss the possible etiological factors from the available literatures and correlating them with histopathological conditions.

In the present study, a coenurus cyst measuring 7.6 cm in diameter containing 21 visible scolecis was found on the ruminal surface of the left kidney. The exact species to which it belongs could not determined as in the genus Taenia intermediate stages of the different species are very similar morphologically. However, the incidence similar to that of Maqsood's (1944) finding who reported a sterile Echinococcus cyst of region's egg size in the cortex of the right kidney and thus confirmed the possibilities of the development of intermediate stages of the Taeniidae family in the kidney of domestic animals.

The deposition of hemosiderin in the form of brownish granular pigment in or around the proximal and
distal convoluted tubules of the cortical portion and in
the wall of the blood vessels during the present
observation is similar to the findings of Al Zahawi (1957)
Light (1960) and others.

The presence of hyaline cast in the form of
acidophilic homogenized spherical body in the spaces of
Malpighian bodies and tubular lumen was one of the
commonest finding associated with subacute or chronic
type of interstitial nephritis during the present
observation. This hyaline deposition seems to be either
products of degenerative changes within the cells or
proteins which passed through injured and abnormally
permeable glomeruli and was reabsorbed by the tubular cells.

Lowenhaupt and Greenberg (1945), Heston
et al. (1945), Dick and Prior (1961), Selye and Bois (1956),
Kreteschmar (1956) and others reported kidney is one
of the frequent sites of calcium deposition. In the
present observation calcium deposition in the form of
blue amorphous bodies was detected in the spaces of renal
corpuscles and the tubular lumen of 3 animals in H.E.
stained sections. The calcium deposition here seems to
be of dystrophic type as the deposition was influenced by
the alkalinity of dead and damaged tissue due to increased
interstitial fibrous connective tissue.

According to several authors (Smith and Jone, 1958, Runnells et al. 1960, and Jubb and Kennedy 1963) petechial haemorrhages and congestion due to active hypoaemia in kidneys were noticed very frequently in a variety of bacteraemias viraemias, poisoning and sometimes even in healthy slaughtered animals. In the present study the specimens collected from different sources were showing presence of petechia and congestion in kidneys and their respective histopathological findings compared favourably with the descriptions of the above authors but in the kidneys collected from the rinderpest goats there was a slight increase in lymphocytic infiltration.

In the present investigation coagulative necrosis was found in 16 out of 150 animals (10%). The most sensitive structures of the renal cortex-proximal and distal convoluted tubules were the main sites of affection. The necrosed area was having typical patchy distribution. The general architecture was retained but cellular details were lost and pycnotic and karyolytic changes of the nuclei, seen. Most of the collecting tubules were found normal. These findings are in agreement with Innes (1950), Thal (1955), Hewitt (1956), Baronetto (1961) and Pattanaik and Kuppuswamy (1963), Hunt (1965). The
etiological factors for necrosis varies considerably and it was not confirmed here that whether presence of toxic substances in the circulation or the arrest of blood supply are responsible for the development of necrotic condition.

Incidence of white spotted kidney in the present observation was found in 43 cases out of 946 animals, i.e. 4.54% which is higher than dorsis cited by Mutayra et al (1946) who found it as 2.3% but less than 8% reported by Pubrimann (1957) in cattle.

Etiology of white spotted kidney is very conflicting as most of the workers differ in opinion, Lubke (1934) reported Brucella abortus responsible for the development of white spotted kidney in experimental calves. But his opinion was contradicted by Magnusen (1934), Kurtsae (1936), and Heinr (1938) as they failed to isolate Brucella organism from the white spotted kidneys. Unger and Bernkopf (1947) studied the Leptosiral infection responsible for the development of intestinal nephritis and suggested it should not be confused with white spotted kidney as none of the experimental animals showed the symptoms of white spots in the kidneys. But Iyer and Nanda (1965) were able to demonstrate of leptospira in the convoluted tubules of white spotted kidneys from two goats where the incidence of white spotted kidney was 5.8%. The spirochaete was considered responsible for the development of subacute interstitial
nephritis which needs confirmation by bacteriological examination.

Rieck cited by Hautyra (1926) suggested secretion of toxin which originated either through the influence of unknown micro-organism or ganism or without them and fattening of calves with whole milk and confinement in dark, close and badly ventilated quarters favours the development of white spotted kidney.

Moore and Hallman (1930) suggested vitamin A deficiency can produce white spotted kidney in calves. Whidik (1937) examined 30 cases and his attempt for isolation of bacteria was another failure which led him to the conclusion that toxic substances are responsible for the development of white spotted kidney and his view was further supported by Tsairoyannis (1957) who observed the condition in 1376 out of 45,000 slaughtered calves. He examined 250 pairs of spotted kidney but failed to isolate any organisms and suggested an allergen or allergens of unknown origin as the possible etiological factor for the development of white spotted kidneys.

In the present study white spots were found occurring single or multiple white foci of varying sizes in one or both the kidneys. They were found restricted to cortex only as medulla showed little or no change. Histopathological examination showing nephrosis varying from cloudy swelling to necrosis affecting glomeruli as well as corresponding tubules were observed, but none
of them showed interstitial nephritis. This is in disagreement with the findings of most of the workers who observed interstitial nephritis usually associated with white spotted kidneys.

The incidence of scar and contracted kidneys is one of the very important and significant pathological changes which was encountered in the present investigation. Grossly scar of the renal cortex was found in 4.12% (39 out of 946) animals, having constant microscopic lesion of interstitial nephritis in all the affected kidney were observed. A considerable variation and certain in development and certain peculiarities in distribution of the scarring areas were found, as they were present in the 35 right kidneys, 3 left kidneys and bilateral development affecting both the kidneys simultaneously in only one.

Marshall (1956) suggested the congenital abnormalities i.e., failure of the development of a group of nephrons was responsible for the production of scar in the renal cortex in case of human and the size of the scar always depended basically on the number of affected nephrons. This congenital hypothesis is based on the number of affected propositions i.e., all scars having typical structures and therefore of common origin and due to lack of information regarding the other etiological
factors responsible for the development of scar in the renal cortex. But the present study does not agree with that of congenital abnormalities as an etiological factor, as increased interstitial fibrosis was found responsible for the atrophy and degeneration of the normal structures resulting in the development of scar in the renal cortex.

In diffuse interstitial nephritis both the kidneys became small and hard due to increased interstitial fibrous connective tissue (Wester - 1935). This shrunken condition started as interstitial lymphocytic nephritis which in turn become fibrosed producing non-inflammatory degeneration, tissue contraction and restricted blood supply to the affected area. In the present observation except in one case only single kidney was found exhibiting the lesion instead of in both the kidneys as observed by Dhane.

Jubb and Kennedy (1963) described that in chronic diffuse interstitial nephritis it is characterized by the formation of scar tissue. Fibrous proliferation mainly of young fibroblast were present in the areas of cellular infiltration and in places mature collagens were also observed. Progressive alteration in the nephron i.e., many atrophy, degeneration and eventually replacement of the tubules were observed, by fibrous connective tissue, while others exhibited dilatation and hypertrophy. The
glomeruli were found atrophied and shrunken and Bowman's capsule showing considerable thickening in many places. A large proportion of the renal parenchyma were replaced by fibrous scar tissue containing random collection of inflammatory mononuclear cells. Many of the tubules were containing granular or hyaline materials. In this present study changes were in agreement with those described above, except, the excessive lymphocyte infiltration, and few plasma cells were observed during the study. In several places numerous lymphocytes were present forming a spherical mass and most of the hyperplastic tubular epithelium exhibited bizarre appearance.

Hofmann (1955) considered the appearance of white spots only in acute stage of interstitial nephritis and concavity of the renal surface resulting from increased proliferation of fibrous connective tissue in chronic interstitial nephritis. In the present investigation the high incidence of white spotted kidney (4.54%) and scar kidney (4.12%) is in agreement with Hofmann (1955), where he observed that there may be some common etiological factor responsible for the development of white spots in acute and scar or chronic stage.

But failure of demonstration of spirochaetes in the stained tissue section in the present observation did not correspond with the findings of Iyer and Nanda (1965).
and McIntyre and Montgomery (1952) as they claim to have demonstrated leptospira in white spotted and scarred kidneys respectively associated with interstitial nephritis.

Similarly attempts were made for isolation of bacteria from three white spotted and five scarred kidneys showing pronounced gross lesions but without significant results further attempts for the finding of bacteria and fungi, if any, of etiological significance in the stained tissue sections also proved negative.

As in white spotted kidney, etiology of interstitial nephritis is also confusing. Most of the workers described white spotted kidney exhibiting interstitial nephritis. But in this present observation, the occurrence of interstitial nephritis was found in the kidney showing only one scar. The probabilities of systemic infection by haematogenous route was also doubtful as most of the scars developed unilaterally affecting single kidney. Probably the toxins originating from sources like vegetables, minerals, microbes or auto intoxication due to disturbed metabolism from remote parts of the body might be responsible is also doubtful.

From the present study it may be concluded that the incidence of white spotted and scar kidney of undetermined etiology is quite frequent in the goats, and
require further study particularly on etiological aspects. The association of the lesions with nutritional disorder, specific infections, diseases and climatological factors needs further exploration to elucidate the etiology of the white spotted and scar kidneys to adopt control measure.
SUMMARY

In the present investigation the kidneys were collected from 50 male mongol pathologists. The additional control material used 7 male guinea pigs were selected and examined for the incidence. All the specimens thus obtained were divided into 2 groups according to their age and sex.

A congenital abnormality was not observed, 1 in normal development of a substance that was recorded by the left kidney, showing no significant abnormality.

Incidence of a large number of atherosclerotic plaques was noted in the collected material and in the blood vessels in the kidney.

Atherosclerosis was the common denominator in the asymptomatic changes from correlation with most of the clinical samples of the kidney.

Sudden type of atheroma development was noted in the material by both renal and 1 in the blood vessels in the kidneys.

Atherosclerosis, potential membranes, and aneurysms were rarely seen in males.

1) In the cases discovered of atherosclerotic renal phenomenon finding area of dead tissue, associated with the blood vessel.
In the present investigation the specimen were collected from 150 goats showing pathological conditions encountered when 946 goats were surveyed and examined for the incidence. All the specimens thus collected were divided into 6 groups according to their gross lesions.

1) Congenital abnormality was not observed.

2) An unusual development of a coenurus cyst was recorded in the left kidney showing no significant alterations.

3) Presence of iron pigment hemosiderin was observed in or around the convoluted tubules and in the lumen of the blood vessels in two specimens.

4) Hyaline degeneration was the commonest degenerative change found associated with most of the chronic lesions of the kidney.

5) Dystrophic type of calcium deposition was noticed in the damaged or dead area of the tubules.

6) Anaemic, petechial haemorrhagic and congested kidneys were mostly seen in goats.

7) In two cases incidence of infarctions were recorded showing area of dead tissue engorged with stagnant blood.

8) Coagulative necrosis was found most frequently affecting the convoluted tubules in the cortex.
9) Arteriolarosclerotic condition was recorded in two cases exhibiting increased thickening of intima and proliferation of the media.

10) Calomerulonephritis was found in only one case showing increased endothelial proliferation and leucocytic infiltration.

11) All the white spotted kidneys (4.5%) were exhibited nephrosis which covers the changes from cloudy swelling to necrosis effecting most of the convoluted tubules.

12) Scar kidney showed sub-acute and chronic interstitial nephritis in all cases which were very common lesions encountered during the present observations. Attempts for demonstration of leptospira other bacteria and fungi were unsuccessful.

13) No incidence of pyelonephritis was encountered in the present study.

14) Incidence of renal tumourlymphoma was found in one case only during the present investigation.
REFERENCES


Boyd, W.L. and Bishop, L.M. (1937).


(Vet. Bull. 33, Abs. no. 3849).


Histological Technique ed J. Oxford
University Press, London.


Amer. J. Path. 36: 77-103.

Casarosa, L (1953).


Christiansen, M (1952).


Corsico, G. (1953).


Idem (1960).

Ibid. 83: 353-366.
(Vet. Bull. 31, Abs. no. 2309).


Allatrov Lapok 20: 115.


(Vet. Bull. 33, Abs. no. 4081).


Mh. Tierhelik. 2: 17-35.

Dalham, T. and Friberg, L (1957).

(Vet. Bull. 27, Abs. no. 3394).
Tijdschr. Diergeneesk 87: 1133-1140.
Dibble, J. H. and Hay, J. D. (1940).
Amer. J. Clin. Path. 26: 409-422.
Dunn, T. B. (1944).
93-119.
French, J. (1950).
Arch. Path. 49: 43-54.
Fubrimann, H. (1957)


Idem (1964) Ibid 87: 79.


(Vet.Bull.32, Abs.no. 4323).

Ibid. 6: 95-46.


(Vet. Bull. 25, Abs.no. 1793).

1-2: 93-119.

(Vet.Bull.33, Abs.no.238).

Lowenhaupt, H and Greenberg, D.M. (1945)
Arch.Path. 45: 49-55.


(Vet.Bull.5: 695).

Mallory, F.B. (1942) Pathological Technique, Philadelphia cited
by Gridley. 20: 260-261.


Marzato, P.S. and Testoni, L (1962)
(Vet.Bull.33, Abs.no.2505).


Rosen, V.J., Castanera, T.J., Kimeldorf, D.J. and Jones, D.C. (1961)  
Amer. J. Path. 38: 369.

Rossi, P and Lamy, E (1947).  
(Vet. Bull. 18, Abs. no. 1652).

Principles of Veterinary Pathology.  
The Iowa State University Press, Ames, U.S.A.

(Vet. Bull. 32, Abs. no. 2170).

pp.185. Abs. from Diss. Abstr. 21, 3428.  

Saxton, J.A. Jr. and Kimball, C.C. (1941)  
Arch. Path. 32: 951-965.

73: 384-387.  

Seegal, B.C. and Loeb, E.N. (1946).  
J. Exp. Med. 84: 211-222.  

Seyle, H and Bois, P (1956)  

Idem (1960) 
Ibid 80: 259-270.

Siller, W.G. (1959) 
(Vet. Bull. 30: Abs. no. 524).

Idem (1964) 
(Vet. Bull. 34: Abs. no. 4691).

Simond, J.P., Hepler, O.E. (1945)
Arch. Path. 39: 103-108.

Smith, H.A. and Jones, T.C. (1957)
Veterinary Pathology. Ed. 1st.
Lee Fibiger, Philadelphia.

Smith, T (1925) 
J. Exp. Med. 41: 413-424.

Soitys, M.A. (1961) 
J. Path. Bact. 81: 441-446.
(Vet. Bull. 31: Abs. no. 2427).

Staemmler, M (1956) 
(Vet. Bull. 27: Abs. no. 233).


Stevens, G.E., Clark, J and Seller, A.F. (1936)
(Vet. Bull. 26: Abs. no. 3617).

Story, H.E. (1943) 

Thompson, S.W., Cook, J.E. and Hoey, H. (1959)
Ibid 607-623.

Thompson, S.W., Wiegeud, R.G., Thomas, R.W., Harrison,
M. and Turbyfill, C.L. (1959)
Amer. J. Path. 35: 1105-1115.
(Vet. Bull. 30: Abs. no. 1559).

Thompson, F.W. Jr. (1960) 
Lab. Invest. 9: 228-238.
(Vet. Bull. 30: Abs. no. 3688).

Thorp, F. Jr., Langham, R.F., Clark, C.F. and Doll, F.R. (1943),

Tolk, A., Eger, W and Schreier, C (1962).
Dtsch. tierarztl. Wehr. 69: 609-612
& 680-685.

Udal, D.H. (1947). The Practice of Veterinary Medicine Ed. 5th Ithaca, N.Y.

Arch. Path. 44: 59-70.


(Vet. Bull. 20, Abs. no. 1646).

Wester, J (1935)
Diergeneesk 62: 62-67 & 128-140
(Vet. Bull. 6: 60).

(Vet. Bull. 35, Abs. no. 4346).

Wiidik, R (1937)