Dystocia Due to Congenital Polycystic Kidney Disease (PKD) of Foetus in a Non-Descript Doe

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Abstract
A non-descript full term primiparous doe was presented with a history of dystocia. The foetus was dead and it was in anterior longitudinal presentation, dorso-sacral position with bilateral shoulder flexion. The foetus was absolutely larger in size and the abdomen was extremely distended. The dead foetus was delivered after the correction of pastural defects. Gross examination of internal organ of foetus revealed that the kidneys were markedly enlarged and were pale, tan, smooth, irregular and contain numerous fluid filled cysts. Hence, the condition was diagnosed as polycystic kidney and this may be a cause for dystocia in primiparaous in non-descript goat.

Key words: Polycystic kidney; foetus; goat; dystocia

Case History and Observations
A non-descript full term pregnant doe of around one year of age was presented with a history of straining and the foetal head slightly protruding out from the vagina for six hours. On clinical examination, the goat was observed to be dull and depressed (Fig. 1) and to have a slightly elevated temperature (40.6°C). The doe was straining for past four hours since rupture of first water bag earlier. Vaginal examination revealed completely relaxed cervix and dead foetus was in anterior longitudinal presentation, dorso-sacral position and with bilateral shoulder flexion. The foetus was absolutely larger in size, the abdomen was extremely distended and the head was present over the vulval passage.

Treatment and Discussion
After correction of postural abnormality, traction was applied to relieve the foetus. However, the attempt to relieve the foetus by traction failed because of the larger size of the foetus. The foetus was therefore relieved by evisceration over the lower abdomen of the foetus using standard surgical procedure by using concealed palm knife. Gross examination revealed that the foetus was absolutely large in size and both the kidneys were markedly enlarged (Fig. 2). The kidneys were pale, tan, smooth, irregular and 10 x 8.2 cm in size and did not retain the normal reniform shape (Fig. 3). The capsules of both the kidneys were thin, tightly adherent and translucent through which numerous fluid filled cysts were observed. On cut section, numerous round to fusiform cysts of 1–5 mm diameter with clear fluid were observed. Clear distinction between cortex and medulla was absent because of cysts that were diffusely distributed throughout the...

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cortex and medulla. The ureters and bladder were grossly normal.

Microscopically, there was severe dilatation of all renal tubules. The cortico-medullary junction was obscured. Dilated tubules were lined by low cuboidal to flattened squamous epithelium. Little normal renal parenchyma was present interspersed among dilated tubules. Most dilated tubules were empty, but some contained a homogenous or flocculent eosinophilic material. Dilated tubules were occasionally separated by variable amounts of expanded, loose interstitial tissue. The number of glomeruli was greatly reduced. Those present were small and located within a dilated Bowman’s capsule (Fig. 4). No inflammatory reaction was observed and other organs from foetus revealed no changes.

In domestic animals, polycystic kidney disease (PKD) is most often consistent with the human autosomal recessive polycystic kidney disease (ARPKD) in that the disease manifests as stillbirths or death within the first few weeks of life. However, manifestations consistent with the adult or autosomal dominant polycystic kidney disease (ADPKD) have also been described (Newman, 2012). In humans, ARPKD is generally seen in the neonate and presents as bilaterally symmetrical nephromegaly that is invariably associated with generalized portal and interlobular hepatic fibrosis and biliary hyperplasia (Ward et al, 2002). Many humans with ARPKD have been found to have mutations in the gene denoted as polycystic kidney and hepatic disease–1. The longest continuous open reading frame of this gene is predicted to
code for a protein that is known by 2 different names, fibrocytatin and polyductin. This protein is expressed on adult and fetal kidney, liver and pancreas and may be a receptor protein that plays a role in collecting duct and bile duct differentiation. The basic defect in ARPKD may, therefore, be a failure of terminal differentiation in collecting and bile ducts. Polycystic kidney and hepatic disease-1 gene products are members of a novel class of proteins that share structural features with hepatocyte growth factor receptor and plexins, members of a class of proteins involved in the regulation of cell proliferation, cellular adhesion and repulsion (Zerres et al., 2003).

In the present case, the presence of numerous cyst in kidney due to polycystic kidney condition resulted in absolutely larger in size of foetus and had distended abdomen which might be the cause for the dystocia. To approach this kind of case, the dead foetus might be eviscerated to reduce the size of the foetal abdomen and traction may be applied to deliver the foetus.

Summary
In the present case, the condition was diagnosed as polycystic kidney after gross and microscopical examination of foetal kidneys. The presence of numerous cyst in the kidney resulted distended abdomen which might be the cause for the dystocia. To approach this kind of case in goats, the dead foetus might be eviscerated to reduce the size of the foetal abdomen and traction may be applied to deliver the foetus through per-vagina.

References