

## Scientific Report

# Congenital hepatic fibrosis in an aborted calf

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## Summary

An aborted female Holstein foetus with marked generalized anasarca was referred to the Excellence Centre for Ruminant Abortion and Neonatal Mortality, Ferdowsi University of Mashhad. On postmortem examination, red-tinged ascites, pale and firm liver with extreme irregularity and numerous round to oval slightly raised foci on the capsular surface were seen. Histological examination revealed widespread fibrosis, linkage of periportal areas to the central vein region and proliferation of bile ductules forming a branching network within the fibrous tissue. The lesion was diagnosed as congenital hepatic fibrosis.

**Key words:** Aborted calf, Liver, Congenital fibrosis

## Introduction

Congenital hepatic fibrosis is a disorder of biliary system development histologically characterized by diffuse periportal to bridging fibrosis with numerous small often-irregular bile ducts and reduction in the number of portal vein branches, first described by Kerr *et al.* (1961). This condition has been reported, rarely, in some veterinary species such as horses (Haechler *et al.*, 2000), rat (Bettini *et al.*, 2003), cat (Brown *et al.*, 2010), an equine foetus (Brown *et al.*, 2007) and dogs (Brown *et al.*, 2010). There are few reports of occurrence of congenital hepatic fibrosis in aborted calves in literature (Yoshikawa *et al.*, 2002; Testoni *et al.*, 2009).

## Case presentation

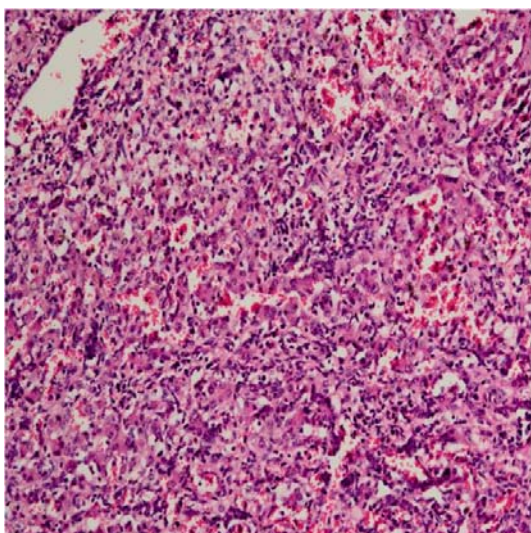
An aborted female Holstein foetus (at 8 month of gestation) was referred to our referral centre for ruminant abortion and neonatal mortality. The aborted calf showed marked generalized anasarca with almost 300 ml red-tinged ascites. The liver was pale

and firm and showed extreme irregularity with numerous round to oval, approximately 1 cm in diameter slightly raised foci on the capsular surface (Fig. 1). The cut surface revealed a lobular structure partitioned by connective tissue and dilatation of the intrahepatic veins. No gross lesions were observed in other organs. Tissue samples from the liver were fixed in 10% neutral buffered formalin and processed routinely and sections were stained with haematoxylin and eosin and Masson's trichrome. Microscopical examination revealed widespread fibrosis and linkage of periportal areas to the central vein region (Fig. 2), connection of portal spaces via connective tissue and frequent formation of cholangiole structures similar to the canal of Herring. Also the proliferation of bile ducts formed a branching network. The sinusoids were slightly enlarged because of hyperplastic proliferation of the fine fibers with numerous fusiform cells which covered the inner wall of the sinusoids. No notable inflammatory reaction in the liver samples was seen. On the basis of the gross and microscopical findings, the lesion was

diagnosed as congenital hepatic fibrosis.



**Fig. 1: Bovine foetus. Congenital hepatic fibrosis. Note the round to oval slightly raised foci on the capsular surface of the liver**



**Fig. 2: Liver, bovine fetus. Congenital hepatic fibrosis. Isolated islands of hepatocytes embedded in abundant fibrous connective tissue, (H&E, ×10)**

## Discussion

Ductal plate malformation arising from incomplete ductal plate formation or reconstruction has been implicated in the pathology of congenital hepatic fibrosis. Congenital hepatic fibrosis in humans is often associated with autosomal recessive polycystic kidney disease (Desmet, 1992; Shorbagi and Bayraktar, 2010), but in the present study, no renal lesion was observed. Bile duct hyperplasia has been attributed to biliary dysgenesis arising from arrested biliary development. Thus, the increased number of bile ducts observed in congenital

hepatic fibrosis may represent primitive liver precursor cells rather than a reparative response to liver injury. Portal hypertension has been reported to be a sequela of congenital hepatic fibrosis in adult humans and even less frequently in children (De Vos *et al.*, 1998). Portal hypertension may be the cause of the ascites found in aborted calf in our case. The generalized anasarca may be due to altered hydrostatic and osmotic blood pressure caused by severe liver disease. When hepatic fibrosis develops, the portal vein system cannot filter effectively through the fibrotic and nodular liver, which results in increased pressure of the blood flowing from the digestive system. This increased pressure forces fluid out of the blood vessels which collects in the abdominal cavity and causes ascites. The levels of albumin decrease in the blood following hepatic failure causes a change in the pressure necessary to prevent fluid exchange (osmotic pressure). This change in pressure allows fluid to seep out of the blood vessels.

Congenital hepatic fibrosis is a rare and sporadic cause of perinatal mortality in cattle and may have a genetic basis that might justify developing cattle as an animal model of human disease. The clinical manifestations of this disorder are non-specific (Zeitoun *et al.*, 2004) and have a lot in common with other hepatic disorders, making the diagnosis difficult. To the authors' knowledge, this is the first reported case of congenital hepatic fibrosis in aborted calf in Iran.

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