TOXIC HEPATITIS IN NAJDI SHEEP SPONTANEOUSLY ASSOCIATED WITH ABNORMAL INGESTION OF MOULDY POULTRY WASTE

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ABSTRACT

A five-year old Najdi sheep suffering from generalized jaundice and encephalopathy was clinically investigated. Case history revealed long time adaptation of the animal for abnormal ingestion of poultry waste. Blood chemistry and haematology indicated hepatobiliary disorder, anaemia and coagulopathy. Pathological studies showed liver degeneration, chronic cholangiohepatitis, fatty infiltration of hepatocytes and proliferation of bile ductules. Application of toxicological techniques, on reticulo-ruminal juice of the diseased sheep and on poultry manure collected from animal yard, could extracted two mycotoxins in both samples. Identified aflatoxic agents, may be the cause of toxic hepatitis in Najdi sheep, possibly because of the relative high susceptibility of such breed for mycotoxicosis.

INTRODUCTION

Sheep do not appear to be very susceptible for mycotoxins ingested in mouldy food (Buxton and Fraser, 1977). However, hepatic disease because of mycotoxicosis is a well recognized consequence of the ingestion of certain mycotoxins particularly in feedlot cattle, pig and poultry (Pier, 1973). Further, generalized icterus (jaundice) is less commonly seen in ovines unless hepatobiliary blockage occurs (Fraser and Arieff, 1985). The present report described a case of toxic hepatitis in a Saudi sheep breed (Najdi) spontaneously associated with abnormal ingestion of mouldy poultry waste.

Clinical History

A five-year old female Najdi sheep was referred to Veterinary Teaching Hospital, College of Veterinary Medicine and Animal Resources, King Faisal University. The animal was off-food, generalized icterus and weight loss, as well as abnormal adaptation of the animal for regular ingestion of poultry waste from birds reared in the same farm. The abnormal condition (coprophagic pica) initiated at the late pregnancy and continued intensively post-parturition lasting about three months.

Clinical Findings

Clinical examination of the diseased ewe revealed a well demarcated generalized icterus tainting the entire skin and mucosae as well as the eye sclera. Bleeding tendency from gums rectum and vagina (Fig. 1) was also recorded. Additionally, impairment of cerebral function and CNS manifestations were other significant clinical findings.

Laboratory Findings

Haematological profile (Table 1) revealed decreased total erythrocytic and lymphocytic counts whereas increased neutrophilic counts. Prolonged bleeding time and failed coagulability were also observed. Biochemical profile of serum (Table 2) showed increased bilirubin, urea nitrogen and alanine transaminase and decreased glucose, inorganic phosphate, albumin and aspartate transaminase. However, serum albumin recorded within normal ranges. Delayed time retention of bromo-sulphathalein dye and prothrombin time were observed (Table 2). Urine of the diseased sheep showed positive bilirubinuria and negative urobilinogen.

Pathological Findings

Necropsy findings shown wide spread icterus and extensive haemorrahges of the sacrificed carcass (Fig.2). Liver was yellow brown, gall bladder markedly distended and bile duct was extensively fibrosed (Fig.3). Cytopathology of autopsied liver specimens revealed fatty degeneration infiltrating the hepatocytes (Fig.4) and chronic proliferation of bile ductules (Fig. 5).

Toxicological Findings

Toxicological techniques for extraction of mycotoxins from samples obtained from reticuloruminal juice of diseased sheep as well as poultry manure

collected from animal yard were used. Two aflatoxin metabolites (B and G) could be identified on chromatographic plates and Table uantitatively calculated (0.5 mg/kg) according to the method of Trucksess *et al.* (1984).

DISCUSSION

An earlier opinion that animals expressed anomalous behaviour syndrome involving oral ingestive responses only when they had nutritional deficiencies in vain attempts to relive the disorder. However, Blood and Radostits (1989) and Fraser (1991) found that hypophosphataemia in sheep commonly leads to ingestion of abnormal materials like faeces (coprophagic pica).

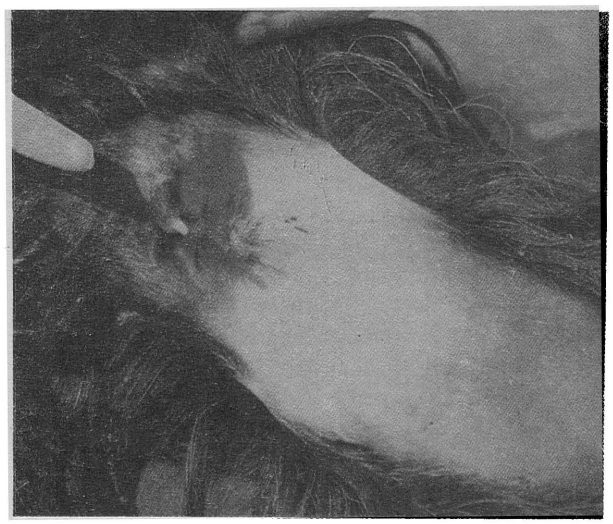

Fig. 1: Bleeding from rectum and vagina of Najdi sheep suffering from toxic hepatitis.

Fig. 2: Wide spread icterus and extensive haemorrhages of sacrificed Najdi sheep suffering from toxic hepatitis.

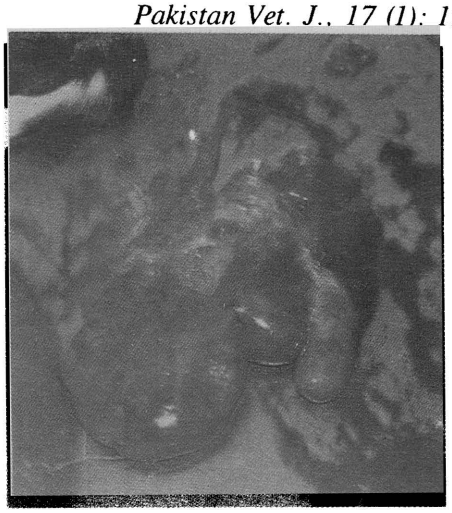

Fig. 3: Yellow coloured liver and marked distended gall bladder in sacrificed Najdi sheep suffering from toxic hepatitis.

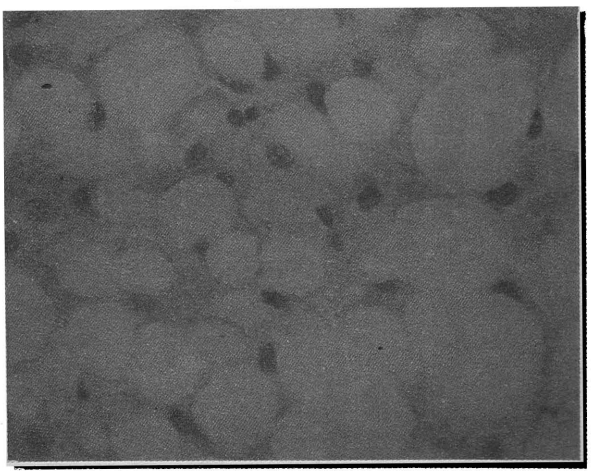

Fig. 4: Fatty infiltration of hepatocytes of autopsied liver obtained from sacrified Najdi sheep suffering from toxic hepatitis (Stain H and E X 400).

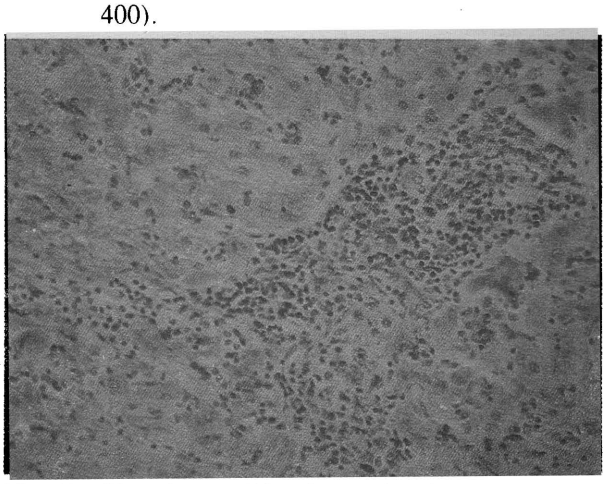

Fig. 5: Chronic proliferation of bile ductules of autopsied liver obtained from sacrificed Najdi sheep suffering from toxic hepatitis (H and E, X 400).

Table 1: Haematological profile of Najdi sheep affected with toxic hepatitis

Analyte	Measured level	Reference level (Jain, 1986)
Haematocrit (%)	18.40	24-45
Haemoglobin (g/dl)	6.80	8-16
Red blood count (count $X^6/\mu l$)	3.80	8-15
White blood count (counts $X^3/\mu l$)	21.20	4-21
Neutrophils (%)	80.00	10-50
Lymphocytes (%)	16.00	40-75
Monocytes (%)	4.00	1-5
Eosinophils (%)	0.00	
Basophils (%)	0.00	
Bleeding time (minutes)	Prolonged	1-5
Clotting time (minutes)	Failed	1-6
		Coagulability

Table 2: Biochemistry profile of Najdi sheep affected with toxic hepatitis

Analyte	Measured level	Reference level (Medway *et al.*, 1969)
Total bilirubin (mg/dL)	5.20	0.52-1.90
Aspartate transaminase (I.U./litre)	75.00	307 ± 43.00
Alanine transaminase (I.U./litre)	60.00	38 ± 4.00
Glucose (mg/dL)	38.00	50-80
Inorganic phosphate (mg/dL)	3.10	5.0-7.30
Urea nitrogen (mg/dL)	85	8-20
Albumin (gram/dL)	5.50	6-7.90
BSP dye retention test (heparinized plasma)	Exceed 48 hours	6 ± 2 hours
One stage prothrombin time (citated plasma)	>15 seconds	< 10 seconds

On the other hand, spontaneous mycotoxicoses have rarely been reported in sheep and no explanation given for such relative resistance particularly for aflatoxicoses (Feuell, 1969). Results of clinical findings indicated kernicterus (bilirubin toxicity) (Kaneko, 1980). This especially because, sheep did not tolerate complete biliary occlusion well and died as early as 3 week in some instance (Fraser and Arieff, 1985). Although, nervous signs associated hepatic disease are multifactorial in origin, the observe coma and impairment of cerebral function in the diseased ewe were considered a neuropsychiatric syndrome of hepatic encephalopathy possibly because of the reported hypoglycaemia (Smith, 1990), or elevated blood urea nitrogen (Coles, 1980). Results of haematological profile confirmed that hepatic function of the diseased sheep was compromised. Also, it can be interpreted as a stressed-leucogram associated anaemia. However, the biochemical findings appeared to indicate hepatocellular damage and cholestasis. Lillehoj *et al.* (1970) and Suttle (1990) reported hepatotoxicoses and changes in bone marrow, erythrocytes and phosphorous along with problem of hepatic necrosis, hypoalbuminaemia and ascites in sheep lambs fed large amounts of poultry waste from hen batteries. As regard for patho-anatomic findings, necropsy indicated fatty degeneration of liver and gall bladder cholestasis because of the extensive hyperplasia of the common bile duct which were confirmed histopathologically.

Both of the extracted mycotoxins (B and G aflatoxin metabolites) are known to act as primarily hepatotoxin (Jones and Hunt, 1983) and possibly were responsible for the reported hepatobiliary disorder (toxic chlangohepatitis) in the diseased sheep. A wide variety of mycotoxins known not all are hepatotoxic (Richard *et al.*, 1979). At a dose rate of 4 mg/kg death occurs to weathers within 15-18 hours due to acute toxic hepatic insufficiency. At dose rate of 2 mg/kg, there is increased respiratory rate, fever and bloody diarrhoea (Armbrecht, 1970). In conclusion, we

believe that the present case is of toxic hepatitis and the possible causality is aflatoxicosis which may aroused as a consequence of abnormal ingestion of mouldy poultry waste because of aphosphorosis (hypophosphataemic coprophagic pica) of the afflicted animal . However, it is noteworthy to mention that mycotoxicosis, may be important in such native Saudi breed of sheep (Najdi) than has been apparent to date in other ovines.

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