

PATHOLOGY OF NON-ABORTED FOETUSES IN BUFFALOES

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INTRODUCTION

During the relatively long prenatal life, embryos and foetuses are sometimes subjected to different deleterious agents, which do not necessarily lead to death but probably to pathological lesions which may result, postnatally, in an adverse effects. These include growth retardation, malformations or lesions predispose to infections. Study of the pathological changes, incidence and pathogenesis of the lesions in our domestic buffaloes during prenatal life helps much to outline the preventive and control measures of many of the diseased conditions occurring postnatally.

MATERIALS AND METHODS

The materials consisted of buffalo-foetuses at various crown-rump measurements collected from the abattoir. Only these in which the placentae were apparently normal were included in this study. After removal, the external features and gross appearance of the internal organs and body cavities were observed; the brain of these cases did not examined. Histopathological study of paraffin embedded materials stained by the conventional methods was also carried out.

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RESULTS AND DISCUSSION

Different pathological conditions were found to occur in the examined foetuses, these are shown in table 1. Of these lesions, haemorrhage in different parts of the body, internal organs and body cavities was one of the remarkable change. Hypoxia and anoxia may be responsible for the occurrence of this haemorrhage in individual cases but it is unlikely to be the solitary cause in all since that both old and recent haemorrhagic areas were observed, and that many of these foetuses were free from these changes. Other factor leading to haemorrhage which must be considered is thrombocytopenia; probably due to destruction of platelets in the circulation. This is known to occur under conditions of septicaemias and acute viral infections in animals (JUBB and KENNEDY, 1970). Many viruses were isolated from foetal tissue or foetal fluids including the viruses of Pseudorabies, IBR, Malignant Catarrhal fever, Foot and mouth disease, PI-3, Cattle Plague, Rift Valley fever, Bovine Viral Diarrhoea, Bovine Enterovirus as well as Unclassified Bovine Syncytial Virus (HUBERT et al., 1973). It is of interest that specific antibodies to some of these viruses, namely bovine viral diarrhoea, pseudorabies, malignant catarrhal fever and IBR, have been detected in precolostrum serum from newly-born calves and serum from apparently normal foetuses, indicating a previous intrauterine infection which could not have caused the death of the foetus (KNIAZEFF et al., 1967; SOLBERG, 1975; MIURA et al., 1974). ANDRIEVSKII and REDKIN (1967) reported, moreover, field observations of newborn calves with aphthous lesions. In the foetus, infection with IBR virus is characterized grossly by haemorrhage in the lung and pleura, and petechiae may also be present on the endocardium and pericardium, and occasionally other organs (JUBB and KENNEDY, 1970). REED et al. (1971) investigating the use of fluorescent antibody technique for the detection of IBR viral antigen in aborted foetuses found that in three

out of 31 cases where IBR viral antigen was detected by the fluorescent antibody technique the histological findings were negative. LUDWIG and STORZ (1973) suggested that IBR virus may be present in the latent form in the bovine foetus. This evidence indicates that on occasion the virus may be present in the bovine foetus and not cause abortion.

In the present study, although the participation of viral infection in this wide-spread haemorrhage is quite probable, however, the exact cause cannot be determined unless a study of the correlation between pathological lesions and the occurrence of infection is carried out.

In Egypt, BAYOUMI (1973) studied the incidence of pathological lesions in calves one to six months age; affection of the respiratory system was found in 43 out of 50 cases. Thirty of the affected cases showed pneumonia, while 13 showed only congestion and oedema of the lung. In the present study, 19 out of 59 abnormal foetuses revealed pulmonary haemorrhage. In man, the latter condition is known to occur in premature infants, infants small-for-date, or stillborns; asphyxia appears to be a common denominator (BABSON and BENSON, 1971). Although hypoxia is to be suspected as the cause of haemorrhage in the present cases, and indeed it is in individual cases, other factors may be responsible also.

Cystic dilatation of the umbilical cord is thought in itself to be of no great significance but the correlation of this lesion with pathological changes in the kidneys of affected foetuses must receive more attention. Renal changes in these cases consisted of destruction of many tubules and glomeruli especially at the corticomedullary junction, and the development of microcysts. These changes are probably due to the backpressure imparted by stagnant urine in the urachus associated with constriction of this part. Electrolyte imbalance due to renal failure may occur postnatally. Moreover,

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growth retardation, partly from pressure of these cysts on the umbilical vessels, and partly due to renal failure is to be expected also. One of the suggested function of the kidney is to convert the large protein molecule of growth hormone to a smaller, biologically active molecule (AUSTIN and SHORT, 1972).

ANON (1964) found that the incidence of deaths in calves at the United Kingdom due to developmental defects was 0.7%. In the present study, two cases of cystic and polycystic kidneys; the latter could be lethal, were found.

Developmental malformations of the reproductive organs, although not lethal, are of great economical importance. Sacular dilatation of the uterus due to uterine hypoplasia, freemartinism, ovarian hypoplasia and the presence of cystic follicles and intrafollicular haemorrhage have been observed in female buffalo-foetuses (GHANNAM and DEEB, 1969; DEEB and GHANNAM, 1976; DEEB and OMAR, in press; DEEB and SALAMA, in preparation). In cattle, the hereditary nature of uterine and ovarian hypoplasia and cystic ovaries have been determined (ERICKSSON, 1938; LAGERLOF and SETTERGREN, 1952, PALSON, 1961; ARTHUR, 1964), and it seems that buffalo does not differ. The mechanism of development of cystic follicles in our buffalo-foetuses is unknown and may be a reflection of high level of maternal gonadotrophin with a predominating follicular stimulating fraction associated with an increase of its transport across the placenta, or it is due to defected control mechanism of the gonads with unbalanced hormone production in the corresponding foetuses accompanied with reduced response of the ovaries to luteinizing hormone which is probably due to the effect of estrogen or estrogen-like substances.

The effect of the occurrence of cystic follicles, the most frequent of malformations observed in foetal ovaries, is worthful to be discussed. Beside degenerative changes of

primary follicles induced by pressure imparted by the large follicles, the question arises whether the estrogen which may be produced by these follicles has an effect on the central nervous system, especially the hypothalamus, comparable to what has been obtained in experimental animals. This effect consists of a hypothalamus incapable of cyclic release of ovulating amount of luteinizing hormone in female rat treated with exogenous steroids.

Table 1. Some Pathological Changes of Buffalo-Foetuses Collected From The Abattoir.

Haemorrhages

(cutaneous - subcutaneous - internal - subepicardial)

Pulmonary haemorrhages.

Torsion of the umbilical cord.

Cystic dilatation of the umbilical cord.

Dilatation of the hepatic vessels.

Hepatic cirrhosis.

Cystic kidneys.

Developmental abnormalities of the female reproductive organs.

(ovarian hypoplasia - cystic follicles - intrafollicular haemorrhage - uterine hypoplasia - freemrtinism).

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