Vet. Med. J., Giza. Vol. 44, No. 3. (1996):557-562.

CHICKEN ANAEMIA VIRUS OUTBREAK IN CHICKENS IN SAUDI ARABIA

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SUMMARY

In a poultry farm located in western province of Saudi Arabia, there was a complain of downgraded broiler carcasses at slaughter due to the presence of haemorrhages in the skeletal muscles of the birds. Accordingly, an investigation was conducated concering the outbreak. It revealed clinical and pathological findings consistent with chicken anaemia virus (CAV) infection. The virus was demonstrated in the bone marrow of affected chickens. Further studies such as reproduction of the disease experimentally in chickens confirmed that the outbreak was due to CAV.

INTRODUCTION

Chicken anaemia agent, which was recently designated as chicken anaemia virus (CAV), has a unique genetic structure that identifies it as a member of newly recognized class of viral agents (Gelderblom et al., 1989).

The disease produced by CAV in fully susceptile young chickens is characterized by severe anaemia, haemorrhages in the skin and musculature, aplasia of the bone marrow and generalized lymphoid atrophy (Goryo and others, 1989).

CAV was first described in commercially produced broiler chickens in 1979 by Japanese researchers (Yuasa and others, 1979). Subsequently, it was isolated in Australia, Brazil, Denmark, France, Germany, Great Britain, Malaysia, The Netherlands , New Zealand, Sweden, The USA (Yuasa, 1993) and Argentina (Buscaqlia and others, 1994).

MATERIAL AND METHODS

Specimens were obtained from an outbreak with clinical picture of CAV occurred in broiler chickens in Saudi Arabia during the first six months of the year 1994 in a multiple-age farm of about 200,000 birds allocated in nine flocks. The flocks were purchased from local hatcheries when they were one day old and reared on deep litter in a closed system housing. These originated from parent flock in Saudi Arabia and vaccinations against Newcastle disease (ND),

infectious bronchitis (IB) and infectious bursal disease (IBD) were carried out. The specimens were subjected to clinical, pathological, and laboratory investigations. Pathogenicity testing of the isolated CAV was conducted on one day old chicks proved to be free from CAV antibodies.

RESULTS

Clinical history:

The main clinical features consistently associated with the outbreak were increased mortalities that were estimated about 7% among young chicks. When they reached two weeks of age, the mortality declined to normal and about 25% of the chickens failed to reach the expected body weight by the 7th week of age. The affected groups appeared uneven in size and more than 20% of carcasses were rejected after slaughter due to the presence of muscular haemorrhages in breasets and thighh muscles.

Pathological findings:

Post-mortem findings revealed atrophy of the thymus and bursa of Fabricius, with discolouration and enlargement of the liver. Muscular haemorrhages of breast, legs and thighs were present (Fig. 1). The facia of the muscles showed serosanguineous fluid. Bone marrow appeared light pink in colour with easy detachment of the proximal femoral, proximal tibiotarsal and distal humeral epiphyses. These lesions were prominent, particularly in birds aged among 16 and 34 days.

Laboratory investigations:

Serological examinations demonstrated the absence of antibodies to Reo virus, However, sera samples collected from the broiler chickens that showed clinical signs and aged 28 days and older were found positive to CAV when tested by indirect immunofluorescence technique (IFA) (McNutly et al., 1988).

Isolation of CAV was carried out by Dr. D. Mekkes and his colleagues at the Poultry Health Center - Doorn, Netherlands according to the method described by McNulty et al. (1990). Two isolates were obtained from the bone marrow after 6th tissue culture passage using MDCC-MSBI cell line derived from Marek's disease lymphoma. Both isolates were, resistant to chloroform, and survived exposure to 70°C for 15 minutes in water bath. The virus was identified based on specific inhibition of the isolates with antiserum to the Cux-I strain of CAV (McNulty et al., 1990) and by IFA (McNulty et al., 1988). Both isolates were registered in Poultry Health Center - Doorn system under the following codes; CAV - D94, 452G0100GMSB and CAV-D94.452G0200GMSB.

Experimental infection:

Intramuscular inoculation of CAV positive liver extract (from naturally infected birds) into 32 commercial Ross broiler chicks aged 1-day-old, known to be free of CAV antibodies, revealed pathological changes in all infected chicks having

Fig .1 : Petecheal haemorrhages in the thigh muscles (M. Pubischiofemorales) (arrow) of chicks naturally infected with CAV.

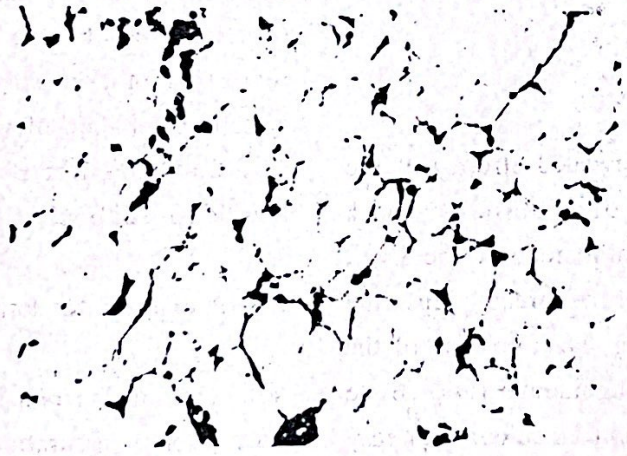

Fig .2 : Marked aplasia in the femoral bone marrow of chicks killed 14-days after inoculation with CAV at 1-day-old. Erythroid and myeloid tissues were replaced by adipose tissues (H & E stain, 136 X).

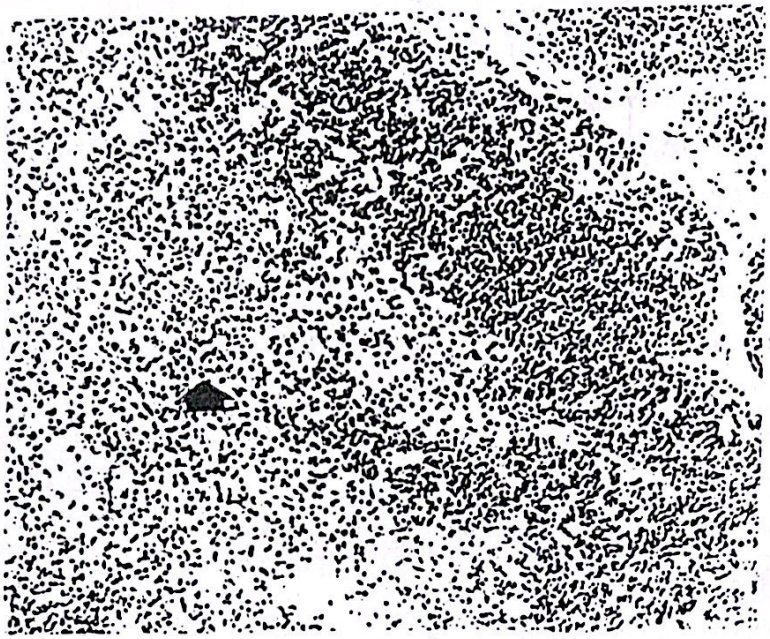

Fig . 3 : Haemorrhages and depletion of lymphocytes in the cortex and medulla of thymus of chicks killed 14-days after inoculation with CAV at 1-day-old. The medulla is almost occupied by reticular cells and erythrocytes (arrow) (H & E stain, 50X).

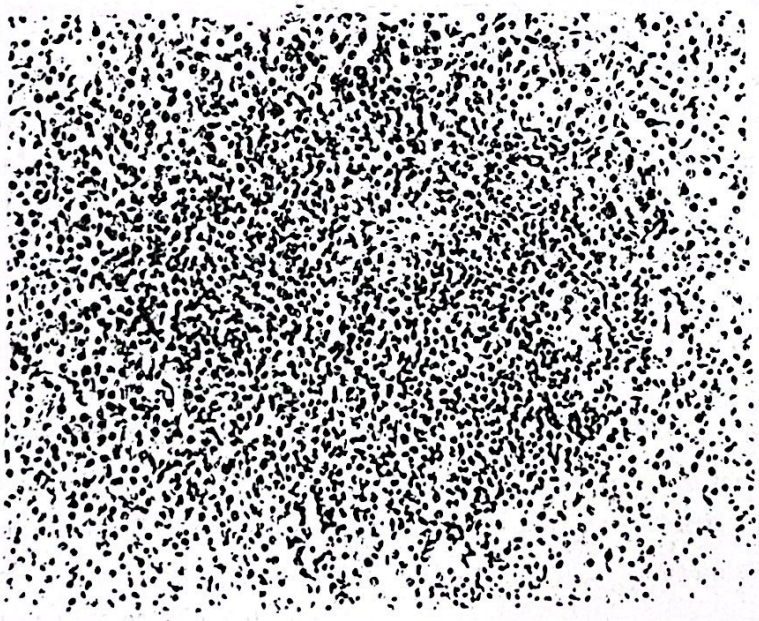

Fig .4 : Focal area of necrosis with mononuclear cell infiltration of lymphocytes and macrophages in the liver of chicks killed 14-days after inoculation with CAV at 1-day-old (H & E stain, 50 X).

the characteristics of CAV infection: retarded growth, severe anaemia, with a mean (± SD) haematocrit value below 27%, (18.4 ± 4.78), a red blood cell count below 5,000 cells/mm3 (3200 ± 480) and caused significant ($P < 0.001$) increase of mortality compared to the un-inoculated birds. IN this group 37.5% of the birds died by the second week of inoculation. None of the un-inoculated controls developed lesions and all had haematocrit values ranging from 29 to 38% (31.88 ± 2.45). All such parameters were measured 14 days post-infection. At autopsy, lesions were found in almost all organs, but were prominent in bone marrow and thymus. Other lymphoid organs showed marked atrophy and there were petechial haemorrhages throughout the body including proventriculus and skeletal muscles. The bone marrow of the femur became light pink and the haematopoietic tissues were almost completely replaced by adipose tissues (Fig. 2). Thymus showed extensive haemorrhages and marked depletion of lymphocytes primarily in the medulla, in some cases the medulla was occupied with reticular cells and erythrocytes (Fig. 3). In liver, swelling of hepatocytes and focal areas of hepatocytes and focal areas of hepatocyte necrosis associated with lymphocytic and macrophage cell infiltration was observed (Fig. 4). Serum samples collected 14 days post-inoculation from the experimentally infected chicks reacted negatively with the CAV antigen. The CAV was successfully re-isolated from liver of experimentally inoculated birds.

DISCUSSION

The evidence presented indicated that the isolated virus has strong similarities with CAV virus. The gross and microscopic lesions were consistent with previous reports of CAV. The identification of the virus strain as CAV was based on, (a) clinical signs and gross pathological findings in affected birds, (b) inducing of the disease in broiler chickens experimentally, (c) reproducing of the lesions in the chickens inoculated with materials treated either with chloroform, or with a temperature of 70°C for 15 minutes and (d) demonstration of the virus in the bone marrow of broiler chickens from the natural outbreak and in the liver from experimentally inoculated chicks.

To the best of the authors knowledge, this probably is the first record of the isolation and identification of CAV from natural disease outbreak in broiler chickens in Saudi Arabia.

It is difficult to trace back the introduction of the CAV infection in Saudi Arabia. However, it is thought that the disease has been introduce in recent years from abroad through the imported ancestries.

These observations provide a strong possibility of the occurrence of vertical transmission of the infection in the above mentioned farm. Some investigators reported that breeder flocks come into production without having been exposed to CAV during the growing period. Following infection of such breeders with CAV would result in vertical transmission to the progeny despite the absence of clinical signs in the breeders and also

the absence of the apparent effects on egg production, hatchability or fertility (Engstrom and Luthman; 1984, Vieltz and Landgraft; 1988). Furthermore, the mechanical spread of the virus through contact with vertically infected chicks and/or contaminated fomites can not be ruled out.

The economic losses due to CAV infection in Saudi Arabia have not been estimated yet. It is highly probable that such high mortality in young chicks, impaired growth and rejected carcasses at slaughter due to such type of viruses were to be associated with big losses in poultry industry in Saudi Arabia. Meanwhile, McNully et al. (1991) showed that subclinical infection of CAV has statistical significant effects on the performance of commercial broilers. It would be profitable to investigate the prevalence of CAV antibodies in chickens in Saudi Arabia.

ACKNOWLEDGMENT

The authors are grateful to Dr. D. Mekkes and his colleagues at the Poultry Health Center - Doorn, Netherlands, for processing the virological materials in their laboratory, and Dr. Omar Al-Jabr for reading the manuscript.

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