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Original Contribution

CHOLANGIOHEPATITIS IN BROILER CHICKENS IN BULGARIA PATHOMORPHOLOGICAL STUDIES OF FIELD CASES

Iv. Dinev*

Trakia University, Stara Zagora, Bulgaria

ABSTRACT
Gross and histological examinations of spontaneous cholangiohepatitis in broiler chickens were performed. The specimens were collected from different batches (hybrids) originating from 2 poultry farms in different regions of the country. Cholangiohepatitis-specific gross lesions were observed by the end of the finisher period or in carcasses at slaughterhouses. Liver histological lesions were detected at a different stage of development. The described results both gross (enlarged and hard livers with pale yellow colour, acinar surface, necroses) and histopathological (proliferative changes in bile ductules with secondary fibrosis, multiple liver granulomas, cholangitis and cholecystitis) corresponded to signs described in broiler chickens with cholangiohepatitis.

Key words: cholangiohepatitis, broiler chickens, pathomorphological studies

INTRODUCTION

Gross lesions related to enlarged, pale liver of hard consistency associated with distended gallbladder with thickened walls in slaughtered broiler chicken carcasses were first described in Scotland (1). Later, a similar condition was also reported from Scotland (2). In Canada, the state was described as hepatosis and fibrosing cholehepatitis (3). The disease was termed cholangiohepatitis during the last decade (2).

Although the etiological agent was unknown, *Clostridium perfringens* has been isolated from visceral organs (liver, gallbladder, extrahepatic bile ducts) of affected chickens (1, 2, 4, 5, 6). *Escherichia coli, Pasteurella haemolytica, Streptococcus equisimilis, Campylobacter* sp. and adenovirus have also been isolated from impaired livers and bile ducts (1, 2, 4).

The purpose of this study was to present the results from gross, histological investigations of spontaneous cholangiohepatitis in broiler chickens in Bulgaria with regard to the diagnostics and differential diagnosis of this relatively recent avian pathology.

***Correspondence to**: *Ivan Dinev,PhD,DVM, Dept of General & Clinical Pathology, Faculty of Veterinary Medicine, Trakia University, 6000 Stara Zagora, Bulgaria, tel.:+359 42 699 679, E-mail:idinev@uni-sz.bg*

MATERIAL AND METHODS

Specimens from visceral organs of 20 chickens were collected for histological examination after observing cholangiohepatitis-specific lesions on 30 broiler carcasses, 12 of them culled at the slaughterhouse and the other – detected during routine gross anatomy inspection. The materials originated from various broiler batches (different hybrids) of two poultry farms in different regions of the country. Specimens were obtained from the liver, gallbladder, spleen, heart, pancreas, duodenum, jejunum and ileum with the caecal tonsils. They were fixed in 10% neutral formalin, routinely processed and embedded in paraffin. Sections of approximately 5 μm were stained by haematoxylin/eosin (H/E). Liver sections were also Gram-stained.

RESULTS

Gross lesions, specific for cholangiohepatitis, were observed by the end of the finisher period or on slaughtered carcasses. At this stage of broilers' development, no clinical signs or increased daily mortality rates were present, although several chickens exhibited retarded growth and dehydration. Pathoanatomically,

the liver was enlarged and with paler yellow colour. In some instances, its surface exhibited the specific acinar appearance, while in others, it was scattered with multiple grey-whitish or green-coloured foci **(Fig. 1).** These foci penetrated deep into the parenchyma, as could be seen on cut sections. The gallbladder walls were thickened, sometimes up to 506 mm and non-transparent **(Fig. 2 and 3)**. The gallbladder was filled with a thick bile secretion or cream-coloured viscous substance in some instances **(Fig. 4).** The subcutaneous and carcass fat of some chickens were icterically tainted. No gross lesions of the other organs were established.

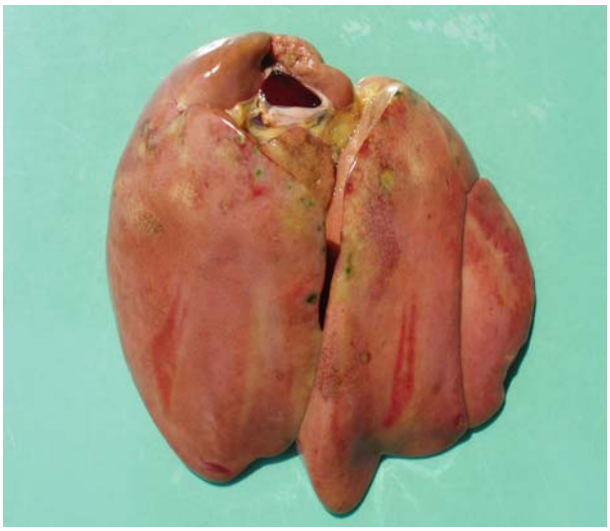

Fig. 1. Pathoanatomically, the liver is enlarged and with paler yellow colour. In some cases, its surface has a characteristic acinous appearance and in others – is mottled with multiple small greyish-white or greenish foci.

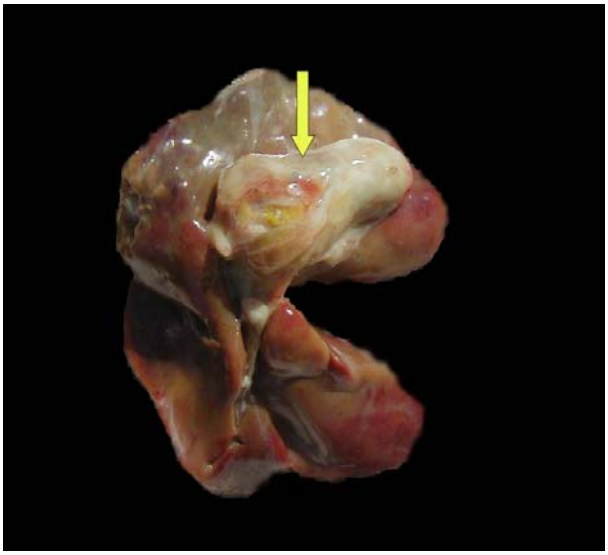

Fig. 2. The walls of the gall bladder are thickened, sometimes up to 5-6 mm, and opaque.

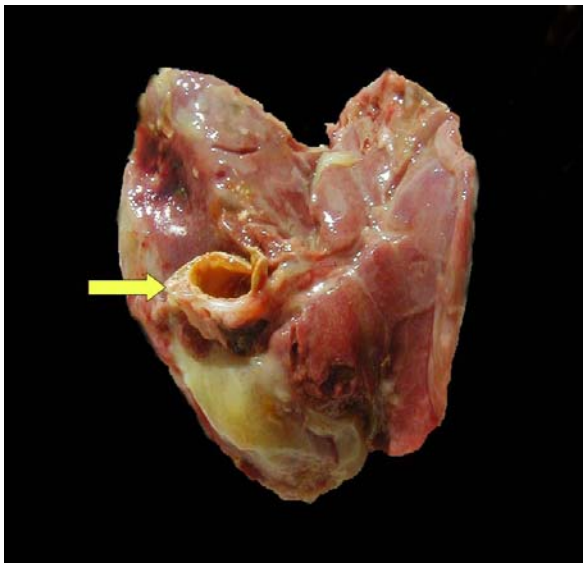

Fig. 3. A transverse cross section through the thickened gall bladder wall (arrow).

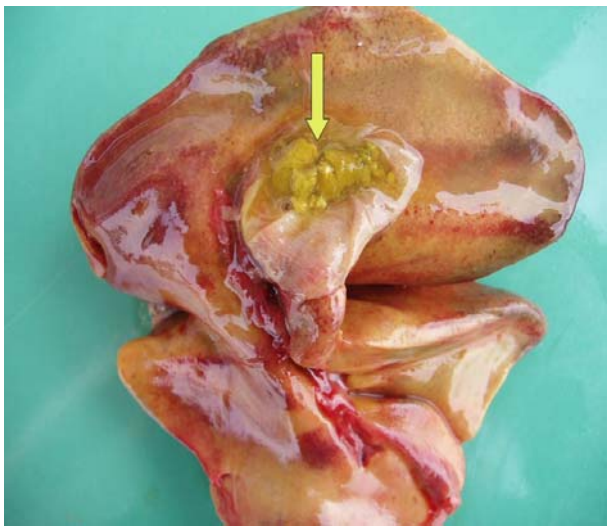

Fig. 4. The gall bladder is filled with a thick bile secretion or a dense matter with a creamy colour.

Histological liver lesions were detected at a various stage of development. In most studied specimens, proliferative alterations of bile ductules were present. Proliferated biliary epithelial cells exhibited most often a columnar or cube-shaped pattern with round nuclei and a pale cytoplasm. Proliferated bile ductules formed granulomatous structures, surrounded by fine reticular fibres **(Fig. 5)**. Centrally, some granulomas showed an initial, while others – advanced necrosis and mild to moderate granulocytic infiltration. Peripherally to granulomas, the liver parenchyma has undergone compression atrophy.

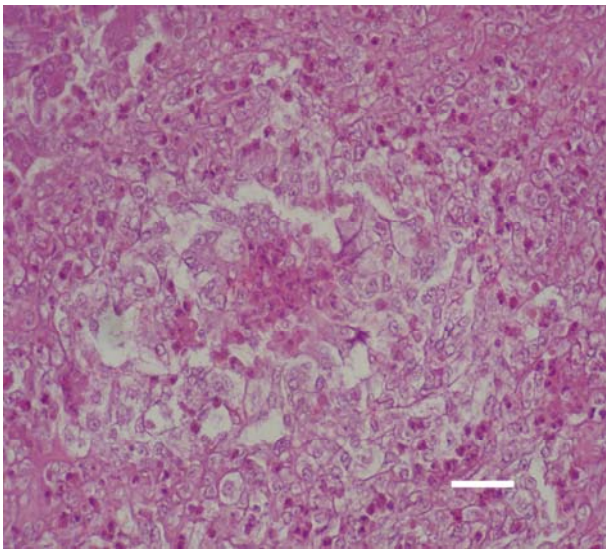

Fig. 5. Liver. Multiple outgrown bile ducts, forming a granuloma structure with a central necrosis. H/E, Bar = 35 μm.

Biliary stasis was present in many bile ducts, with a huge amount of bacilli among the stagnated secretion **(Fig. 6)**. Pericanalicular coagulation necrosis was frequently seen. Among these foci, the necrotic mass was hyalinized and blue-stained microorganisms could be observed. Around the necrotic foci, there was a belt of macrophages, lymphocytes and granulocytes **(Fig. 7)**. Many Gram–positive microorganisms were found among the granulomas and within the lumen of bile ducts and the gallbladder, often accompanied by inflammatory lesions. The mucous coats of bile ducts and the gallbladder were necrotic, and the wall thickened due to connective tissue growth **(Fig. 8).**

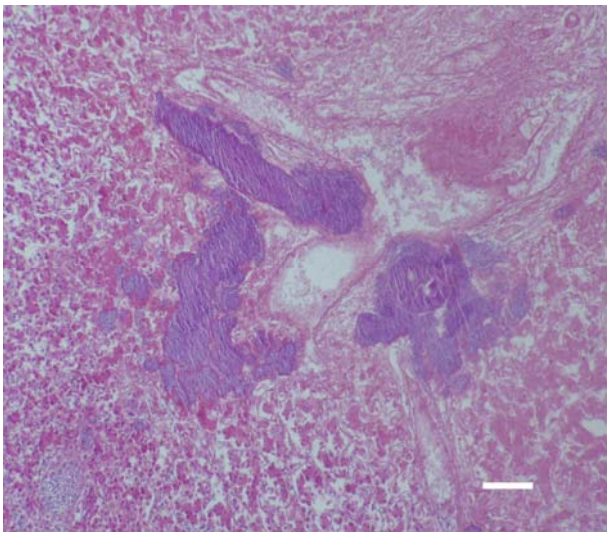

Fig. 6. Liver. A biliary stasis and a huge amount of gram - positive organisms in bile ducts. Gram staining, Bar = 25 μm.

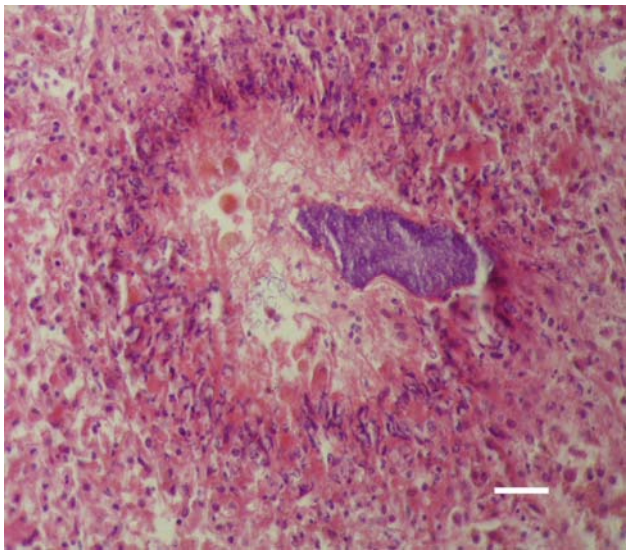

Fig. 7. Liver. Pericanalicular biliary necroses. H/E, Bar = 50 μm.

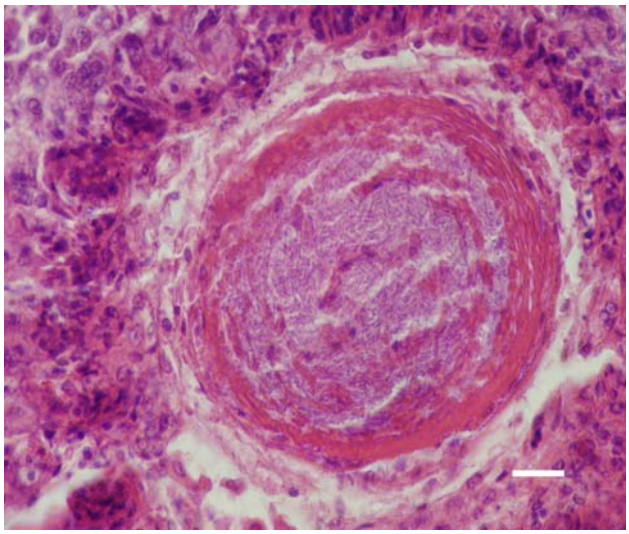

Fig. 8. Liver. Fibrosed and thickened wall of a bile duct, filled with Gram-positive microorganisms after the occurred inflammatory and necrotic changes. H/E, Bar = 35 μm.

DISCUSSION

The gross anatomy results of the present studies (enlarged and hard liver with paler yellow colour, acinar pattern of the surface, necroses) and histopathological (proliferative changes in bile ductules with secondary fibrosis, multiple liver granulomas, cholangitis and cholecystitis) corresponded to signs described in chickens with cholangiohepatitis (5, 7, 8).

At this stage, we could not discuss the involved etiological agent. The results provided strong evidence for the involvement of a rod-shaped Gram-positive microorganism. It was assumed (5, 6, 9, 10) that the infection could be spread by haematogenous,

lymphogenous routes or as an ascendant intestinal bacterial infection. Our observations are in support of the hypothesis for an ascendant intestinal infection due to the fact that major lesions were limited within the hepatobiliary tract.

The presented cholangiohepatitis cases in broiler chickens as also outlined by other researchers were not frequent, with few affected chickens which were sometimes identified at slaughterhouse inspection (5, 10). In one report however (6), the disease was observed as an outbreak, supporting the hypothesis that this was not the case of a sporadic disease.

Our pathomorphological results confirmed entirely that the primary characteristic of cholangiohepatitis in broiler chickens was the extensive proliferation of bile ductules, reported by other investigators (4, 5). This lesion was at the background of the constantly observed cholestasis, whereas pericanalicular necroses were due to bile extravasation and therefore, allowed us to determine them as biliary necroses.

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