ASCITES AND THE ANATOMY OF THE PERITONEAL SACS OF BROILERS

A. J. BEZUIDENHOUT, Departement of Anatomy, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort 0110

ABSTRACT


Although the ascites syndrome of broilers is well documented, all the authors fail to describe exactly in which of the various coelomic cavities ascitic fluid is found. Determination of the precise location of this fluid is essential if the pathogenesis of the syndrome is to be understood and explained. Post-mortem examinations were done on 100 broilers that had ascites or had died of the ascites syndrome. In all of them large quantities of fluid were found in the ventral hepatic peritoneal cavities, moderate amounts in the right and left dorsal hepatic peritoneal cavities, and small amounts in the intestinal peritoneal and pericardial cavities. No ascitic fluid was found in the left dorsal hepatic peritoneal cavity. The amount of ascitic fluid effusing from the liver was directly proportional to the surface area of the liver in a given peritoneal cavity.

INTRODUCTION

The ascites syndrome of young broilers is a well recognized condition responsible for great economic losses, especially during the cold winter months. Many research efforts are currently being focused on the possible causes and pathogenesis of the condition. Huchzermeier & De Ruyck (1986) have shown that the ascites in the ascites syndrome is associated with a right cardiac ventricular overload.

Ascites, however, is associated with many different disease conditions and affects various peritoneal cavities of birds. There are many vague and conflicting reports regarding the cavities affected in the various ascites-associated diseases. Bock, Shore, Samberg & Perl (1986), Lekkas, Iordanidis & Artioplos (1986) and Maxwell, Robertson & Glick (1983) found ascitic fluid in the abdominal cavity, and Bergman et al. (1979) describe a highgrade filling of the hepatic peritoneal and other body cavities. Krakower & Goetsch (1945) report a marked ascites in one of the 3 peritoneal cavities, while Pizarro, Salas & Paredes (1970) and Ahmad, Asfhaque & Azam (1986) found fluid in the abdominal cavity and abdominal air sac. Olah, Taylor & Glick (1983) found a small amount of ascitic fluid in the hepato-peritoneal sac, while the visceral-peritoneal cavity contained large amounts of fluid, and Feron & Van Straatum (1966) describe ascites of the cranial and caudal body cavities.

To understand the pathogenesis of ascites in diseased conditions it is necessary to define which of the serious cavities is affected. In the present study it was ascertained which of the cavities are affected in the ascites syndrome of broilers.

MATERIALS AND METHODS

One hundred Ross, Hubbard and Hybro strain broilers between 3 and 6 weeks of age, which had died from the ascites syndrome or were suffering from clinically detectable ascites, were randomly collected over a period of 3 weeks from commercial poultry farms. The animals were humanely slaughtered. The birds were pinned to a board in dorsal recumbency and the skin reflected from the abdomen and thorax. A small incision was made into the left ventral hepatic peritoneal cavity, caudolaterally to the sternum, and the ascitic fluid allowed to drain into a collecting vessel. The incision was then enlarged cranially along the lateral border of the sternum and caudally as far as the caudal limit of the cavity, care being taken not to damage the ventral mesentery that separates the right and left ventral hepatic peritoneal cavities, or the post-hepatic septum that separates the ventral hepatic peritoneal cavities from the intestinal peritoneal cavity.

If any coagulated transudate was present, it was carefully removed and the total volume of ascitic fluid measured. The cavity was then inspected for communications with the right ventral and left and right dorsal hepatic peritoneal cavities, as well as with the intestinal peritoneal cavity. The same procedure was followed on the right side in order to examine the right ventral hepatic peritoneal cavity and its content. An incision was then made into the post-hepatic septum on the right, to expose the intestinal peritoneal cavity for investigation. Any fluid that was present was carefully collected and measured.

The sternum was then removed and the pericardium opened. Any fluid present in the pericardial cavity was collected by aspiration into a syringe and measured. At this stage of the examination the air sacs were also inspected. By lifting the caudal parts of the liver, the small left and right dorsal hepatic peritoneal cavities could be visualized. Both were entered by an incision made in their containing peritoneal membranes, and any ascitic fluid encountered was collected and measured. The cavities were investigated for communications with each other, the ventral hepatic peritoneal cavities and the intestinal peritoneal cavity.

RESULTS

Ventral hepatic peritoneal cavities

In all the broilers examined, the left and right ventral hepatic peritoneal cavities were grossly distended. Each contained between 100 and 150 ml of transudate. In 30 broilers, the 2 ventral cavities communicated freely with each other through a large perforation. All the perforations were located caudally to the liver along the sternal attachment of the ventral mesentery. The right cavity generally contained slightly more fluid than the left.

Right dorsal hepatic peritoneal cavity

In all the broilers examined, the right dorsal hepatic peritoneal cavity was moderately distended and contained between 10 and 30 ml of transudate. No communications between the right dorsal hepatic peritoneal cavity and any of the other peritoneal cavities were found.

Left dorsal hepatic peritoneal cavity

No transudate was encountered in the left dorsal hepatic peritoneal cavity of any of the broilers. The cavity communicated freely with the intestinal peritoneal cavity in all the broilers.

Intestinal peritoneal cavity

Only a small amount of transudate was found in the intestinal peritoneal cavity. This varied between 1 and 10 ml and did not cause abdominal distension in any of the broilers.

Received 29 October 1987—Editor
ASCITES AND THE ANATOMY OF THE PERITONEAL SACS OF BROILERS

Pericardial cavity

The pericardial cavities of all the birds examined contained between 0.5 and 4 ml of transudate. Communications between the pericardial cavity and peritoneal cavities were not found in any of the birds.

Air sacs and pleural cavities

None of the air sacs or pleural cavities in any of the birds contained fluid.

DISCUSSION

The development and topographic anatomy of the coelomic cavities of birds are very well documented by Poole (1909), McClelland & King (1970), Duncker (1979) and McClelland (1979). The five peritoneal cavities (Fig. 1) within the avian coelom are formed by 5 sheets of peritoneum which form partitions within the peritoneal coelom. The combined dorsal and ventral mesentery (Fig. 1) forms a continuous vertical sheet from the dorsal to the ventral body wall as far caudally as the gizzard. Caudal to this level there is only a dorsal mesentery. The left and right sheets of the post-hepatic septum extend cranio-caudally from near the last 2 thoracic vertebrae to the caudal wall of the peritoneal cavity. It divides the peritoneal cavity into 3 principal cavities: a midline cavity, the intestinal peritoneal cavity; and two lateral cavities which enclose the liver. Each lateral cavity is further subdivided by the left and right hepatic ligaments (Fig. 1) into left and right dorsal and left and right ventral hepatic cavities.

All the aforesaid authors state that the ventral mesentery divides the ventral hepatic peritoneal cavity into separate left and right compartments. None of the authors mentions communications between the 2 ventral hepatic peritoneal cavities. In the present study, large perforations or communications between the 2 cavities were found in 30% of the broilers examined. It is assumed that the perforations were pathological and possibly developed through the tremendous filling of the ventral hepatic peritoneal cavities, resulting in an initial stretching and later a localized rupture with perforation of the ventral mesentery.

To understand the pathogenesis of any condition characterized by the development of ascites in birds, it is imperative that the coelomic cavity or cavities be precisely identified. It is therefore surprising to find the descriptions in the existing literature of the peritoneal cavities, in which ascites is formed, very vague and inadequate. All the authors failed to describe exactly in which of the 16 distinct and separate cavities fluid was found. Eight are cavities of the air sacs and the remaining 8 are cavities of the coelom proper. Bergman et al. (1979) describe a high-grade filling of the hepatic peritoneal cavities in the ascites syndrome, while Olah et al. (1983) found little ascitic fluid in the hepatoperitoneal sac, but large quantities in the visceroperitoneal cavity.

In the present study, large quantities of fluid were found in the left and right ventral hepatic peritoneal cavities, a moderate amount in the right dorsal hepatic peritoneal cavity, small amounts in the pericardial and intestinal peritoneal cavities and none in the left dorsal hepatic peritoneal and pleural cavities. The volume of fluid found in each of the 4 hepatic peritoneal cavities was directly proportional to the free surface area of the liver contained within each of these cavities. These observations support the contention (hypothesis) that venous hypertension caused by right ventricular overload, as reported by Huchzermeyer & De Ru yck (1986), could be the cause of such an effusion from the liver. The surface area of the liver contained in the left dorsal hepatic peritoneal cavity is the smallest, and, because this cavity is in direct communication with the intestinal peritoneal cavity, ascitic fluid would not accumulate here but would drain into the intestinal cavity. Only small amounts of fluid were found in the intestinal peritoneal cavity in the broilers. The right lobe of the liver, being also bigger than the left lobe, accounted for the slightly larger volume of fluid found in the right ventral hepatic peritoneal cavity.

Ahmad et al. (1986) and Pizzaro et al. (1970) also reported ascitic fluid in the abdominal air sacs. In the present study, no transudate was found in any of the air sacs and its presence in them cannot be explained in terms of our current understanding of the pathogenesis of the ascites syndrome. Such cases need further investigation.

ACKNOWLEDGMENTS

I wish to thank Dr F. W. Huchzermeyer of the Onderstepoort Veterinary Research Institute for supplying all the material for the study.

REFERENCES


