NOTE *Avian Pathology*

Intestinal Volvulus with Coagulative Hepatic Necrosis in a Chicken

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ABSTRACT. A 7-week-old SPF chicken inoculated at 4 weeks of age with chicken anemia virus was puffed up depressed and had ruffled feathers and a good body condition. Intestinal volvulus involving the jejunum and part of the duodenum forming two loops with one knob was observed. Microscopically, venous infarction of the obstructed loops, periportal and sublobular multifocal coagulative hepatic necrosis and granulomatous inflammation of the cecal tonsils were observed. Gram staining revealed no bacteria in hepatic tissue; however, gram-positive bacilli were detected in the necrotic debris in the intestinal lumen. Immunosuppression might have predisposed the chicken to intestinal and cecal tonsil infection that then progressed to volvulus. Loss of the mucosal barrier in infarction might allow bacterial toxins and vasoactive factors to escape into the systemic circulation (toxemia) and be responsible for the hepatic necrosis.
KEY WORDS: CAV, hepatic necrosis, immunosuppression, intestinal volvulus.

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Of the gastrointestinal tract derangements in fowl, intestinal intussusception [15, 25], intestinal volvulus [1, 5, 11], proventricular intussusception [9, 18, 21] and herniation of the intestine through the post-hepatic septum into the ventral hepatic cavities [8] have been reported. Intestinal disarrangements rarely occur in fowl. There is paucity in reports about pathology and pathogenesis of intestinal derangements in Aves [4, 25]. In horses, intestinal displacements that progress to incarceration or volvulus with strangulation and infarction are a common cause of colic and mortality. Torsion of the long axis of the mesentery is common in suckling ruminants and swine but rarely occurs in dogs and cats. Intestinal intussusception including the small intestine, cecum and colon is common in dogs, lambs, calves and foals [2].

Intestinal volvulus is a twisting across the long axis of the gut characterized by compression of the thin-walled veins and obstruction of the influx of arterial blood, which eventually progresses to dilation, devitalization and venous infarction of the affected segment [2]. Whereas intestinal volvulus rarely occurs and is incidentally observed in chicken carcasses, intussusception occurs relatively frequently in diseases such as coccidiosis, ulcerative enteritis and worm infestation [4, 25]. The present report describes a peculiar case of intestinal volvulus with hepatic necrosis in a chicken anemia virus (CAV)-inoculated SPF (specific-pathogen-free) chicken and introduces a model for a predisposing factor for intestinal volvulus in the avian species.

One 7-week-old SPF male chicken in an experiment concerning the pathology and immunohistochemistry of CAV infection at 4 weeks of age was sitting alone in the corner of a box and looked puffed up. It was reluctant to move (easy to catch) and had ruffled feathers, but its body condition was good. The combs, wattles and eyelids were cyanotic. Its blood was watery and exhibited delayed clotting. Its packed cell volume (PCV) was 27%. The other birds utilized in the experiment were apparently healthy (no diarrhea or any digestive disorders), with a PCV range of 29–37% (mean and standard deviation 32 ± 2.3%). Gross pathological changes were restricted to the intestinal volvulus. The jejunum and lower part of duodenum were involved in the volvulus forming two loops with one knob (Fig. 1). The affected loops were intensely edematous, congested and hemorrhagic. The wall was thickened, deep red-black and loosely adherent to surrounding viscera by fibrinous exudate. Bloody fluid content and gas distended the lumen of the infarcted loops. On incision, bloody ingesta oozed from the lumen. The mesenteric blood vessels were engorged. The venous infarction involved the full thickness of the intestinal wall, and the limits of the infarcted loops were sharply demarcated (Fig. 1). The intestine proximal to the strangulation point was dilated with ingesta, whereas the distal part was empty. Greenish dilated bile ducts were noted in a cross section of the liver.

Microscopically, severe transmural edema, congestion, distension of veins, venous thrombosis and hemorrhages were observed in the affected loops of the intestinal volvulus (Fig. 2). The full thickness of the mucosa exhibited liquefactive necrosis and was filled with hemorrhages, necrotic debris and degraded food material. The submucosa was greatly distended with hemorrhages, necrotic tissue and gaseous bubbles thus the muscularis mucosae and externa were apart. The muscularis mucosae and muscularis externa were devitalized and interspersed with hemorrhages. The hepatic tissue revealed extensive periportal coagulative necrosis (Fig. 3) associated with congestion of blood vessels and dilatation of bile ducts. The engorged hepatic blood vessels contained fibrinous material (according to Azan-

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Mallory staining), and the sinusoids were dilated (no fibrinous thrombi). Hepatocytes had pyknotic nuclei and deep acidophilic cytoplasms (Fig. 4). Multifocal coagulative necroses of sublobular size were dispersed in the hepatic tissue (Fig. 5). No inflammatory cell infiltration was noted in the area of hepatic necrosis. Gram staining revealed no bacteria in the hepatic tissues. The submucosa of the cecal tonsils revealed caseous granulomas (Fig. 6) formed of a central eosinophilic coagulum of tissue debris surrounded by lymphocytes, macrophages, multinucleated giant cells and a thick wall of fibroblasts. Gram staining revealed gram-positive bacilli within the necrotic debris in the cecal lumen, but no bacteria were stained in the granulomatous lesion. There were granulocytic infiltrations in the proventricular submucosa, with heterophilic exudate in the lumen. CAV-specific changes included mild lymphoid depletion of the thymus cortex and bursa of Fabricius.

Intestinal volvulus in chickens is rare and incidentally recorded in dead cases without apparent cause in the autopsy room [1, 5, 11]. However, intestinal and proventricular intussusception are apparently frequently recorded and are etiologically related to intestinal coccidiosis, ulcerative enteritis and worm infestation [4, 9, 15, 18, 21, 25]. The authors of the above reports suggest that disruption of intestinal motility due to infection promotes its intussusception. The presence of an area of induration in the intestinal wall in addition to the predisposing causes may favor its intussusception [25]. However, due to the infrequency of intestinal volvulus in chickens, neither predisposing factors nor their pathogenesis have been studied. In horses, volvulus of the large colon is common and predisposed by its lack of mesenteric anchorage and potential motility [2]. Gas production from highly fermentable feeds or perhaps hypermotility may be a predisposing factor for torsion of the long axis of the mesentery in swine, calves and lambs [2]. CAV infected thymocytes (especially $CD8^+$ T cells) in the cortex resulting in mild to moderate lymphoid depletion of the thymus cortex in 4-week-old inoculated chickens (data not shown). CAV lymphoid depletion of the cecal tonsils [19, 23] predisposes them to infection and formation of a granulomatous lesion. In the present case of intestinal volvulus in a SPF chicken inoculated with CAV at 4 weeks of age, immunosuppression presumably predisposed it to cecal and intestinal infection with inhabitant bacteria (anaerobes), leading to hypermotility or gas formation that favored volvulus. Pneumatosis intestinalis, an uncommon condition characterized by the presence of gas within the bowel wall that predisposes patients to intestinal volvulus, has been observed in humans after treatment with cytotoxic chemotherapy and immunosuppressive drugs [7]. Swollen Payer's patches due to lymphoid hyperplasia and gastroenteritis are the common predisposing factors for intestinal intussusception and volvulus in infancy and childhood [6]. An evidence of association between human acquired immunodeficiency syndrome (AIDS) and intestinal intussusception and volvulus has been reported. Gastrointestinal manifestations of AIDS that may potentially initiate an intussusception and volvulus include lymphoma, lymphoid hyperplasia, cytomegalovirus colitis, Kaposi's sarcoma and bacterial mesenteric lymph node coinfection, i.e., *Mycobacterium avium intracellulare* [3, 12]. In the present case, the presence of a granulomatous lesion in the cecal tonsils and gram-positive bacilli in the necrotic debris in the lumen at the ileo-cecal junction indicates that intestinal infection might predispose chickens to intestinal volvulus.

The clinical signs of intestinal volvulus in animals are severe, intermittent colic, signs of lethargy, weight loss, restlessness and dog-sitting position. The affected chicken was depressed, reluctant to move and had ruffled feathers; these are similar to the signs of endotoxemia in horses [13] and chickens [20]. Gangrene of the gastrointestinal tract in dogs releases vasoactive factors (histamine and serotonin) into the circulation that initiate the pooling of blood and hypotension, resulting in toxic shock [10]. The vascular damage in intestinal volvulus initiates leakage of protein and erythrocytes out of the vascular space into the interstitium, bowel lumen and peritoneal cavity. Loss of the mucosal barrier allows bacteria and endotoxins to escape into the systemic circulation, inducing endotoxemic lesions [24]. Fowl endotoxemia is associated with *Erysipelothrix rhusiopathiae* infection [20]. *Clostridium perfringens*, a gram-positive bacillus, is a normal inhabitant of the intestinal tract that under predisposing conditions, releases exotoxins (enterotoxins) that are then absorbed into the blood stream, inducing toxemia [16]. CAV has been linked with *Clostridium perfringens* as a causative agent of gangrenous dermatitis and as a predisposing factor in necrotic enteritis [17]. The periportal hepatic coagulative necrosis, engorgement of blood vessels with fibrinous materials (no disseminated intravascular coagulation) and gram staining (no bacteria in the liver, but gram-positive bacilli in the cecum) in the present case suggested that these lesions may be induced by toxemia. CAV-induced anemia in 1-day-old chickens predisposes them to centrilobular hepatic ischemic necrosis [23]; however, in 4-week-old chickens neither anemia nor hepatic coagulative necrosis was observed (data not shown). Hepatic blood flow is largely dependant on the portal venous supply balanced by the arterial supply controlled by the sympathetic nervous system. If the portal flow decreases, the hepatic arterial supply is increased and vice versa [22]. A hepatic ischemic necrosis due to reduced mesenteric venous return and decreased portal blood flow accompanying intestinal infarction has not been reported except in a patient with chronic obstructive lung disease that could potentiate the hypoxia of hepatic tissue [14].

In the present case report, we introduced immunosuppression that might enhance intestinal infection with inhabitant bacteria as a predisposing factor for intestinal volvulus. Like in mammals, venous infarction of the intestine and flourishing bacteria (toxins) may induce toxemic lesions in parenchymatous organs (i.e., the liver).

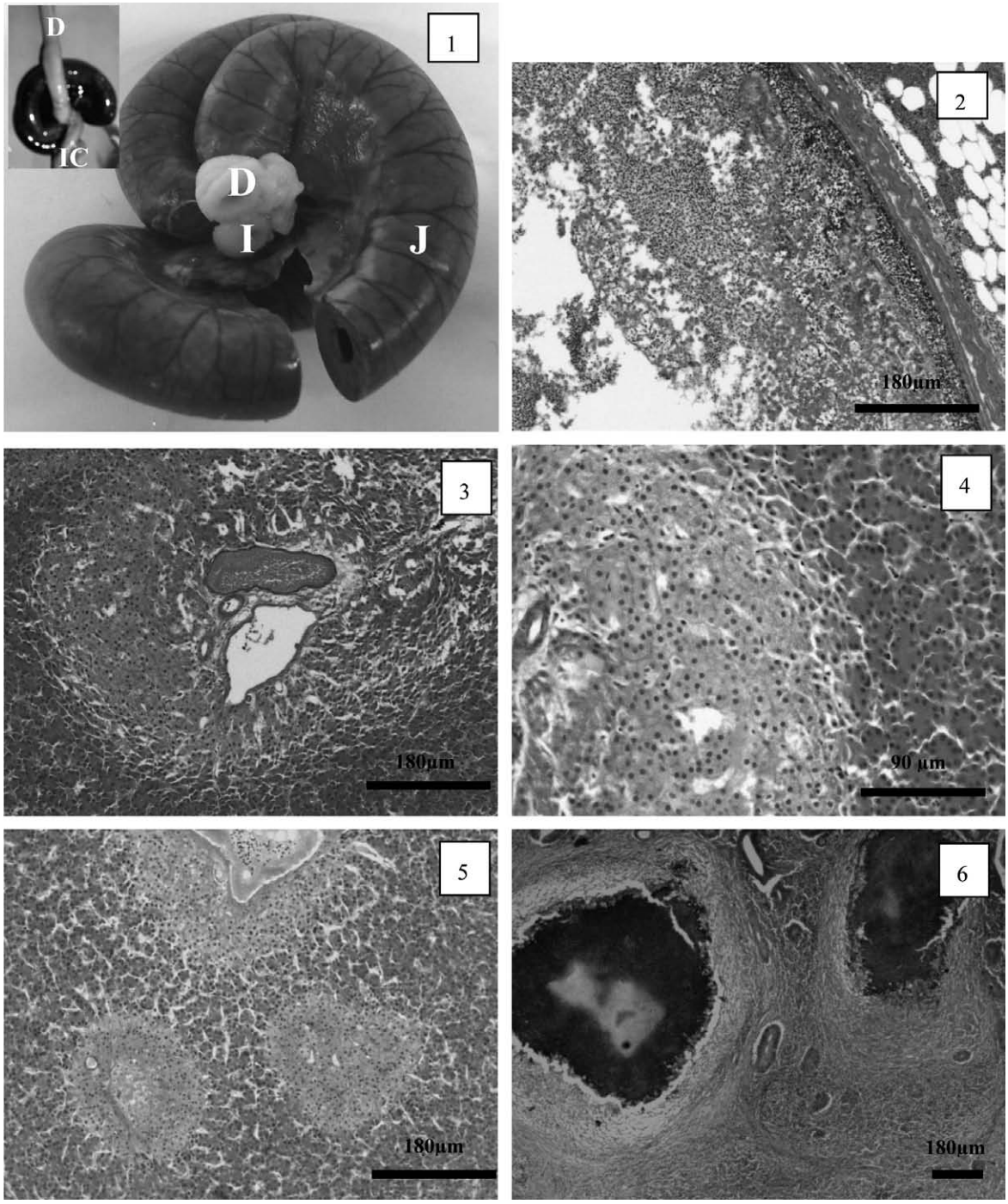

Fig. 1. Venous infarction of two loops of the jejunum and lower part of duodenum that has undergone volvulus (formalin fixed material). Inset: the affected loops are intensely edematous, congested and hemorrhagic (the sample before fixation). D, duodenum. J, Jejunum. I, ileum. C, Cecum.
Fig. 2. Venous infarction of the intestinal wall (HE).
Fig. 3. Periportal hepatic coagulative necrosis (HE).
Fig. 4. Higher magnification of the hepatic coagulative necrosis showing hepatocytes with pyknotic nuclei and deep acidophilic cytoplasms (HE).
Fig. 5. Multifocal sublobular hepatic necrosis (HE).
Fig. 6. Granulomatous inflammation in the cecal tonsils (HE).

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