

## **Histopathological changes in intestine of chicken (*Gallus domesticus*) infected naturally by *Ascaridia galli***

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**Abstract.** This study was aimed at finding out the histopathological changes of chicken intestines that were naturally infected by *Ascaridia galli*. Ten intestines were obtained from wet market in Banda Aceh then measured in length and divided into three sections: duodenum, jejunum and ileum, of which histologic slides were made. The number of *Ascaridia galli* founded in the lumen and ingesta were counted. Parameter picture of histopathological changes were analyzed descriptively based on desquamation, hiperplasia, and villi fussion occure. Duodenum, jejunum and ileum infected by *A. galli* showed histopathological changes of villi the cover of desquamation, hyperplasia and fusion. Damage level of the small intestine chicken was largely determined by the number of infecting by *A. galli*. It was concluded that the more number of *A. galli* infected to chickens, the higher level of damage would be occurred in the small intestine of chicken.

Keyword: *Ascaridia galli*, chicken, histological changes, intestine

### **Introduction**

The large worms *Ascaridia galli* is the most attacking chickens around the world. According to Soulsby (1982), the morphology of *A. galli* is colored with a yellowish white male worms the size of 50-76 mm and 72-112 mm in females. The egg-shaped oval, measuring 73-92µm and 45-57µm. On the anterior *A. galli* contained a mouth on the anterior part of which is equipped with three lips, the lip on the dorsal and two on lateroventral. There is a narrow wings and stretches along the side of the body (Calneck *et al.*, 1997).

Ascariidiosis in chicken leads to a decrease in the gain of body weight, high mortality and other secondary pathological symptoms. *A. galli* infection caused mortality and sometime morbidity. Chickens infected with *A. galli* also experiencing slowing growth 12.31% (Zalizar *et al.*, 2006), decreased body weight by 38% (Tabbu, 2002). Egg quality is low due to decreased egg weight of 5:35%, decrease in eggshell thickness of 5:55% and decreased levels of calcium in blood serum by 36.26% (Zalizar *et al.*, 2007).

According to Soulsby (1982), young chickens more easily infected by adult *A. galli* than chicken or chicken that had been previously infected. Sensitivity of chickens against *A.*

*galli* infection is strongly influenced by age, type of enclosure, nutrients, the type of chicken (Gauly *et al.*, 2001), the dose of infection (Ikeme, 1971), maintenance systems (Permin and Ranvig, 2001) and weather (Kumari and Thakur, 1999).

*A. galli* infections also cause enteritis cataralis hemorrhagica the gastrointestinal mucosa (Urquhart *et al.*, 1987). Each mucosal inflammation is generally followed by impaired digestion, absorption of nutrients such as electrolytes, vitamins (Anwar and Zia ur-Rahman, 2002) and mineral (Gabrashanska *et al.*, 2004) and secretion of substances that play a role in the process of digestion. *A. galli* infection also causes degeneration and nekrosa on epithelial cells of the small intestine villi and crypt, as well as a decrease in the surface area of the small intestine villi (Zalizar *et al.*, 2007). *A. galli* is a chronic infection that can lead to disease symptoms slowly (Permin *et al.*, 1998).

One of the factors that causes chicken infected with *A. galli* easily as traditional maintenance systems that are exempt chicken hanging around and not kept in special cages (Budiman, 2007). The prevalence rate in young chickens are allowed to roam freely in 48% and 24% of adult chickens (Permin *et al.*, 2002).

Research about histopathological changes in the small intestine caused by *A. galli* has been done by several previous researchers. As in the study Zalizar *et al.* (2006), the occurrence of degeneration and necrose on epithelial cells of the villi and crypt in the small intestine of chicken starter. However, research on the chicken naturally infected with *A. galli* has not been reported. In the study to be observed is the histopathological changes in the small intestine caused by *A. galli* infection that occurs naturally in chicken.

## **Materials and Methods**

### ***Place and Time Research***

This research was conducted in the Laboratory of Pathology and Histology Laboratory of the Faculty of Veterinary Medicine Kuala University in Banda Aceh. The research was conducted in February-April 2013.

### ***Samples***

The sample used in this study is the small intestine of infected chicken is naturally derived from Banda Aceh market.

### ***Equipments***

The tools used in this study is the scissors, cutting board, tweezers, hektek, paperboard, microtome, waterbath, object glass, glass and microscope cover. Materials used in the study was 10% formalin fixative solution, wash solution 5% formalin, chloroform, paraffin and HE dyes.

### **Parameter Study**

Parameters measured were histopathologic changes in intestines of chicken infected naturally by *A. galli*, based on the description of villi covering, i.e desquamation villi is not intact because erosion has absorbtive cells, hyperplasia of the villi and the growing size of the attachment of the fusion each other villi.

### **Research Procedures**

Chicken intestine obtained from merchants measured and then divided into three sections (duodenum, jejunum, ileum), and then on each piece cut 2 cm, section cut paper pasted on cardboard by using hecter, and the section was washed by using formalin 5% for the rest of the food is wasted and put in a fixative solution of 10% formalin for 24 hours. Next step is making preparations histopathologic and stained with haematoxylin eosin staining. In the remaining part of the intestine is opened and counted the number of worms *A. galli*.

### **Data Analysis**

Histopathological observations were analyzed descriptively.

### **Results and Discussion**

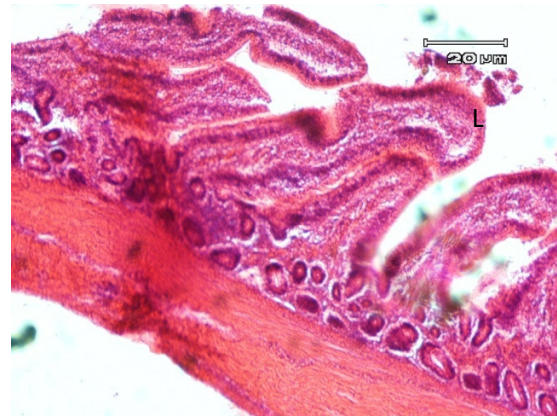
Table 1 shown that epithelial desquamation in the villi more numerous in the duodenum, hyperplasia occurred in all villi, while majority (90%) duodenum and all of jejunum and ileum founded villi fusion. This is in line with research Zalizar *et al.* (2006), worms *A. galli* infection with a heavy dosage (2000 infective eggs) cause degeneration and necrosis of the epithelial cells of the small intestine and cause the villi surface area decreased by 20%. The results of the 10 chicken intestine contained in Table 2.

**Table 1.** The percentage of histopathologic changes in intestine of chickens naturally infected by *A. galli*

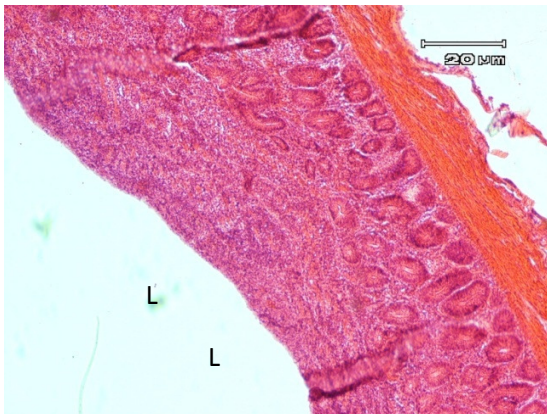
Intestine	Number of samples	Desquamation epitel villi	Hiperplasia villi	villi fusion
Duodenum	10	60%	100%	90%
Jejenum	10	50%	100%	100%
Ileum	10	50%	100%	100%



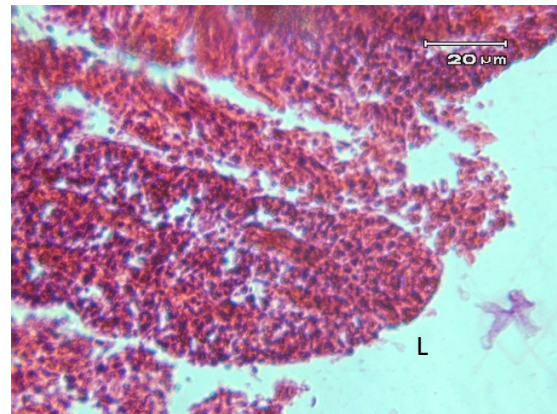
A. Villi epithelial desquamation



B. Hyperplasia



C. Villi fusion



D. Desquamation, hyperplasia, fusion

Figure 1. Desquamation, hiperplasia, and villi fusion in intestine of *Gallus domesticus*

naturally infected by *A. galli* (HE, 400x). L = lumen

Figure 1 seen the presence of villi epithelial desquamation, hyperplasia, and villi fusion. Desquamation of the epithelial villi occurs so that L3 can not get into the duodenal mucosa. While the villi hyperplasia occurs to balance the loss of a number of cells, especially goblet cells that serves to protect the villi of the larvae. In areas experiencing hyperplasia villi contain inflammatory cells, mast cells and goblet cells. It aims to prevent *A. galli* larvae penetrate in the mucosa.

Bahrami (2011) observed that the hypertrophy and hyperplasia occurred in all regions of gastrointestinal tract of lambs infected with multiple nematode *Haemonchus contortus* (50%), *Ostertagia ostertagi* (25%), *Trichostrongylus axie* (12%), *Chabertia ovina* (8%), Cooperia (almost 5%). Histopathological changes also found villi, where as villi were broad and appeared to be flattened in distal regions of small intestine. Hong *et al.* (1997)

suggested that at early stage infection by *Centrocestus armatus*, a minute intestinal trematode of birds and mammals, in albino rats the lesion should be produced by mechanical destruction of the fluke on the enteroepithelial cells. Intestinal pathologic found the stroma of villi around young fluke was edematous and infiltrated by inflammatory cells such as lymphocytes, plasma cells, and eosinophyls. The crypt became mildly hiperplastic and villi were moderately atrophied at four days post infection.

Ferguson *et al.* (1980) showed that the several mice infected with 1000 *Nippostrongylus brasiliensis* larvae had very short or absent villi and long crypts and others had less severe abnormalites. During parasite infection in mice fed an elemental diet increased mucosal damage. Luna-Olivares *et al.* (2012) found that the larvae penetrated in the crypt of jejunal tissue layer pullets seven-week old at three days after infected orally with 20,000 embryonated *A. galli* eggs. Stain *et al.* (1998), states that the abnormality active transfer of ions in the epithelium can result in changes in water flow that contributes to the secretory diarrhea as symptoms of gastrointestinal inflammation. Follow the effects of this phenomenon is the disruption of the intestinal barrier function and abrasion epithelial cells are implicated in desquamation villi.

## Conclusions

Based on the results of this study concluded that the more small intestine of chickens (*Gallus domesticus*) infected by *A. galli* suffered histopathological changes namely desquamation, hiperplasia, fusion that occurs in the jejunum.

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