Pakistan Veterinary Journal

ISSN: 0253-8318 (PRINT), 2074-7764 (ONLINE)
Accessible at: www.pvj.com.pk

CASE REPORT

An Outbreak of Erysipelas in Commercial Geese

Kamila Bobrek[1], Marcin Nowak[2], Joanna Borkowska[1], Katarzyna Bobusia[1] and Andrzej Gaweł[1]*

[1]Department of Epizootiology and Clinic of Bird and Exotic Animals; [2]Department of Pathology, Faculty of Veterinary Medicine, Wrocław University of Environmental and Life Sciences, Wrocław, Poland
*Corresponding author: andrzej.gawel@up.wroc.pl

ARTICLE HISTORY (14-539)

Received:	Oct 22, 2014
Revised:	Dec 28, 2014
Accepted:	May 10, 2015
Published online:	July 02, 2016

Key words:
Erysipelas
Erysipelothrix rhusiopathiae
Geese
Sepsis

ABSTRACT

Infections with the Gram-positive bacterium *Erysipelothrix rhusiopathiae* occur in many vertebrate species worldwide. This study describes an outbreak of erysipelas in a flock of geese, which was identified by post-mortem examination, PCR, and microbiological, biochemical, and histopathological examinations. Post-mortem examination and histopathology revealed that the geese were septic. The biochemical profile indicated only 10.6% match with *E. rhusiopathiae* and 88.8% match with *Arcanobacterium haemolyticum*. PCR was used to determine that the etiological agent of the disease was *E. rhusiopathiae*.

To Cite This Article: Bobrek K, Nowak M, Borkowska J, Bobusia K and Gaweł A, 2016. An outbreak of erysipelas in commercial geese. Pak Vet J, 36(3): 372-374.

INTRODUCTION

Infections with the Gram-positive bacterium *Erysipelothrix rhusiopathiae* occur in many vertebrae species worldwide. Its associations with a wide variety of wild and domestic animals, birds and fish may be either pathogenic or commensal (Wang *et al.,* 2010). Because swine erysipelas is highly prevalent and economically important, the domestic pig is the most relevant reservoir of *E. rhusiopathiae*. Swine erysipelas can occur as acute septicaemia or as a chronic disease in which arthritic lesions and endocarditis develop (Ozawa *et al.,* 2009).

Although erysipelas outbreaks in poultry (except turkeys) are economically insignificant and rarely reported, *E. rhusiopathiae* has been isolated from chickens, ducks, geese, emus, pheasants, and pigeons (Bobrek *et al.,* 2013). Recently, many outbreaks in hens in a few European countries have been reported (Mazaheri *et al.,* 2005; Eriksson *et al.,* 2013). Erysipelas in birds usually occurs as an acute, fulminating, septic infection, but the chronic form can arise after an acute outbreak. Outbreaks typically occur suddenly with only a few birds found dead initially, followed by increasing levels of mortality on subsequent days (Bobrek *et al.,* 2013).

This study describes an outbreak of erysipelas in a flock of geese. Erysipelas was confirmed using microbiological, biochemical and molecular methods.

History: Seven dead meat type White Kołudzka® geese from a farm where geese were reared with access to paddocks were delivered to the clinic at the Department of Poultry Diseases within the Department of Epizootiology for Birds and Exotic Animals Wroclaw University of Environmental and Life Sciences. These geese were from an 11 week-old flock comprising of 3,500 birds. Following an interview with the owner of the flock, it was determined that there were no specific premonitory clinical symptoms before death. In addition, there were no other poultry farms, pigs, or cattle within 10 km of this farm. Two days before the geese were delivered, their mortality increased from 6 to 13 birds. None of the other bird facilities in the region noticed any symptoms of distress or increased mortality of their birds. Visual inspection of the delivered geese was conducted on the day of arrival. The Good Management Practice rules were obeyed on the farm.

Clinical examination and post-mortem findings: The bodies of the examined birds were in good condition. The subcutaneous tissues had petechiae, and clearly-visible vessels filled with blood. The pectoral and thigh muscles were dark. Petechial hemorrhages on epicardia were seen in six of the birds. The petechiae ranged from small petechiae on the vestibules of the hearts to strokes on large areas of the heart chambers (Fig. 1A). Livers were swollen, congested, and fragile, and the capsules were tense in all of the examined birds (Fig. 1B). In four of the geese, the lungs were congested and edematous (Fig. 1C). The spleens of all of the examined birds were dark, filled with blood, and oedematous. The pancreas were pale and covered with petechiae (Fig. 1D). Digestive tracts were slightly-filled throughout their entire lengths. In two

geese, the duodena and jejuna were found to be catarrhal. In all the birds examined, the abdominal fat was reddish, the kidneys were slightly congested, and the veins of the brain were filled with blood, but no petechiae were observed.

Diagnostic procedures and Results

Histopathology: Heart, lung, duodenum, proventriculus, gizzard, small and large intestine, liver, pancreas, spleen, kidneys, bursa of Fabricius, and brain samples from all examined birds were collected and fixed in 10% buffered formalin (pH 7.4) for 24 h, dehydrated, and embedded in paraffin blocks. Paraffin sections were stained with haematoxylin and eosin and analyzed with an Olympus model BX 53 optical microscope equipped with a Colorview III digital camera.

Necrosis and lysis of lymphoid elements were observed in the spleen samples. Degenerative changes and necrosis were observed in the pancreas samples. Parenchymal organs showed generalized vascular lesions. In the liver samples, severe congestion and numerous haemorrhages were visible, and small, focal inflammatory infiltrates composed mainly of lymphocytes were localized around the blood vessels. Parenchymal degeneration, fatty degeneration, vacuolisation, and bile pigment deposits in hepatocytes were also visible (Fig. 2A). Massive congestion and interstitial focal lymphocytic infiltrates were also observed in kidney samples.

Numerous small bacterial colonies, peripheral local inflammatory infiltrates, and cardiomyocyte degradation were seen in cardiac muscle samples (Fig. 2B). Numerous lymphocyte and histiocyte infiltrates were observed in the mucosae and submucosae of small intestine samples, what caused damage, necrosis, and shortening of villi. In addition, massive congestion of mucous membranes was observed. Histopathological examination of the central nervous system showed reactivity and amplification of glial cells in periventricular areas. Histology of lung samples showed considerable oedema, atelectasis, and significant congestion with scattered lymphocytic infiltrates (Fig. 2C).

Microbiological and biochemical testing: Liver, lung, and heart samples were removed from each goose for microbiological examination. Each sample was cultured on blood agar, MacConkey's agar, and Chapman agar for 24 h at 37°C under aerobic conditions. Antibiograms and biochemical tests were performed on pure cultures.

After 24 h of incubation on blood agar, all heart and liver cultures contained small (0.3-0.5 mm diameter), transparent colonies with narrow zones of alpha-hemolysis on blood agar (Fig. 2D). No growth was observed on MacConkey's and Chapman agars. Short, gram-positive rods were visible in Gram-stained cultures, and oxidase and catalase reactions were negative. The morphology of bacterium isolate *Erysipelothrix rhusiopathiae*. Disc-diffusion tests of antibiotic susceptibility showed susceptibility to amoxicillin, amoxicillin with clavulanic acid, enrofloxacin, norfloxacin, lincomycin with spectinomycin (lincospectin) and resistance to doxycycline, tetracycline and gentamycin.

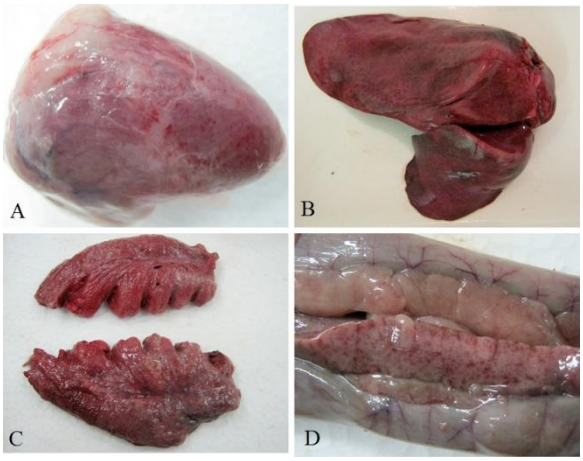

Fig. 1: Gross lesions observed during erysipelas in geese, A) hemorrhages on the surface of the heart muscle, B) enlargement and congestion of liver, C) edema and congestion of lungs and D) petechiae on the pancreas surface.

Biochemical identification was performed using the commercial API Coryne 2.0 system (bioMérieux) according to the manufacturer's instructions. The biochemical profile indicated an 88.8% match with *Arcanobacterium haemolyticum* and a 10.6% match with *Erysipelothrix rhusiopathiae*. There is no poultry disease caused by A. *haemolyticum*, and lesions observed during the necropsy suggest *E. rhusiopathiae* infection. To confirm erysipelas PCR with specific primers was made.

DNA isolation and PCR: *E. rhusiopathiae* IW 445 and *E. tonsillarum* IW 779 from the PIW-PIB strain collection in Pulawy were used as positive controls, and *E.coli* ATCC 25922 was used as a negative control. DNA was isolated from control and test strains using a Genomic Mini Kit (A&A Biotechnology) according to the manufacturer's instructions, and PCR was performed using the primers MO101 - MO102, ER1F - ER1R and ER2F - ER2R according to reaction conditions described previously (Takeshi *et al*., 1999). Positive control PCR products were generated using DNA isolated from *E. rhusiopathiae* IW 445 with primers ER1R and ER1F and using DNA isolated from *E. tonsillarum* IW 779 with primers ER2F and ER2R. Additional positive controls were generated using DNA from *E. rhusiopathiae* IW 445, DNA from *E. tonsillarum* IW 779, and MO101 and MO102 primers. Products were separated on 1.5% agarose by pulsed-field electrophoresis at 110 W and 50 mA for 70 min. The PCR products generated from strains isolated from the dead geese were identical to PCR products generated from *E. rhusiopathiae* IW 445.

DISCUSSION

During this outbreak, 43 geese, which had no premonitory signs, died. In the diseased geese that were examined, vascular changes dominated in parenchymatous organs, and the most affected organs were the liver, spleen, heart, and pancreas. The organs were enlarged, and the hearts were hemorrhagic. Lesions suggested generalized septicaemia, and histopathological examinations found conglomerates of bacteria surrounded by thrombocytes in capillaries and small arteries and veins confirming septicaemia. We found the severity of the lesions to be similar to those observed in previous birds erysipelas investigations (Bobrek *et al*., 2013).

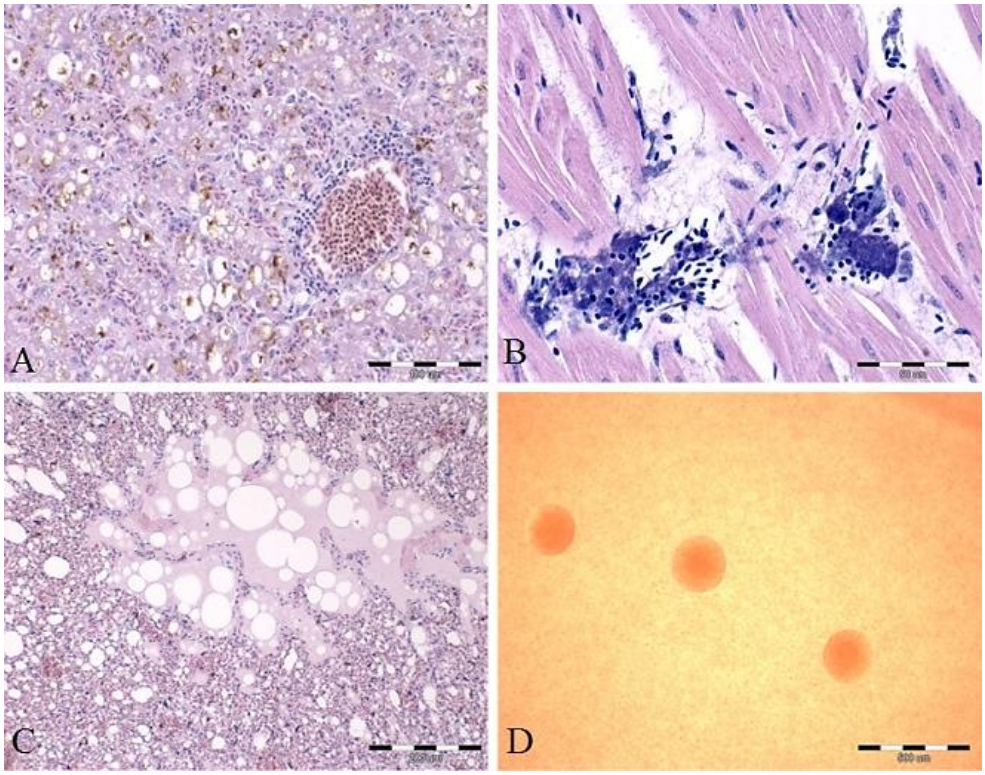

Fig. 2: Histopathologic features of lesions during erysipelas in geese, A) parenchymal and fatty degeneration of liver, vacuolisation, and bile pigment deposits in hepatocytes, B) bacterial colonies, inflammatory infiltrates, and cardiomyocyte degeneration in cardiac muscle, C) significant congestion with scattered lymphocytic infiltrates in lung and D) *E.rhusiopathiae* smooth- form colonies on blood agar after 24h incubation. H & E (A to C) and stain missing (D). Bar equal to 100, 50, 200 and 500µm in A to D, respectively.

Smooth colonies similar to *Erysipelothrix* colonies were visible on blood agar after 24 h of incubation. Gram-staining of these colonies revealed short, gram-positive rods. The API Coryne biochemical system identified the isolate as *Arcanobacterium hemolyticum* and suggested *E. rhusiopathiae* as a possible taxon. PCR identified the isolate as *E. rhusiopathiae.* Based on antibiotic susceptibility tests, the flock was treated with amoxicillin for five days and mortality ceased on the second day of treatment.

This is the first description of an erysipelas outbreak on a farm dedicated to commercial geese production. The geese on this farm had no contact with other animals that could carry *E. rhusiopathiae*, such as pigs, sheep, horses, dogs, chickens and turkeys (Wang *et al.,* 2010).

We suspect that the geese could be infected during being on the pastures, e.g. via contaminated soil, in which *E. rhusiopathiae* can survive for long periods after contamination by the faeces of sick animals, such as wild fowl, rodents, and insects (Eriksson *et al.,* 2013, Eriksson *et al.,* 2014). Further, damaged skin could have facilitated the *E. rhusiopathiae* infections. It has been suggested that the bacteria may gain entry through wounds and damaged mucous membranes after behaviors such as cannibalism and feather pecking (Eriksson *et al.,* 2014). We did not observe that kind of behaviors, but we were not able to exclude some small wounds through which bacterium could get to the circulatory system and cause sepsis. After finished rearing cycle, the pasture was disinfected and another flock was reared without an outbreak.

REFERENCES

Bobrek K, Gaweł A and Mazurkiewicz M, 2013. Infections with *Erysipelothrix rhusiopathiae* in poultry flocks. World Poultry Sci J, 69: 803-812.

Eriksson H, Bagge E, Båverud V, Fellström C and Jansson DS, 2014. *Erysipelothrix rhusiopathiae* contamination in the poultry house environment during erysipelas outbreaks in organic laying hen flocks. Avian Pathol, 43: 231-237.

Eriksson H, Nyman AK, Fellström C and Wallgren P, 2013. Erysipelas in laying hens is associated with housing system. Vet Rec, 173: 18.

Mazaheri A, Lierz M and Hafez HM, 2005. Investigations on the pathogenicity of *Erysipelothrix rhusiopathiae* in laying hens. Avian Dis, 49: 574-576.

Ozawa M, Yamamoto K, Kojima A, Talagi M and Takahashi T, 2009. Etiological and biological characteristics of *Erysipelothrix rhusiopathiae* isolated between 1994 and 2001 from pigs with swine erysipelas in Japan. J Vet Med Sci, 71: 697-702.

Takeshi K, Makino S, Ikeda T, Takada N, Nakashiro A *et al*., 1999. Direct and rapid detection by PCR of *Erysipelothrix sp.* DNAs prepared from bacterial strains and animal tissues. J Clin Microbiol, 37: 4093-4098.

Wang Q, Chang BJ and Riley TV, 2010. *Erysipelothrix rhusiopathiae.* Vet Microbiol, 140: 405-417.