

The Shades of Avian Cellulitis in Meat-Type Chicken

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Mini Review

Received: 04/04/2016

Accepted: 04/09/2016

Published: 05/09/2016

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Keywords: Avian cellulitis, Poultry, Meat inspection, Avian pathology, Broiler pathology

ABSTRACT

Cellulitis of broiler or 'meat-type' chicken is an emerging technopathy, a disadvantageous result of high and intensive animal production, a production cycle disease. Cellulitis causes significant morbidity, mortality and economic losses to the poultry industry: condemnation, downgrading at processing, trimming and reprocessing of affected carcasses. Cellulitis is caused by several different bacteria but most notably by *Escherichia coli*. Cellulitis-affected birds do not exhibit clinical symptoms commonly associated with colisepticemia, and the infection is often detected at slaughter, which results in carcass condemnation at processing. The slaughterhouse, therefore, is a privileged site for the detection and identification of avian cellulitis. The goal of this brief review is a dual approach towards the characterization of avian cellulitis. This clinico-pathologic entity will be described and interpreted in terms of pathology with epidemiological and pathogenetical informations. Then practical cues and skills will be provided for the identification of avian cellulitis during post post-mortem inspection in poultry processing plants.

DEFINITION

Cellulitis is an extra-intestinal infection, affecting integumentary apparatus, caused by avian pathogenic *Escherichia coli* (APEC). Today, *E. coli* strains causing systemic disease in poultry (avian colibacillosis) are termed avian pathogenic *E. coli* (APEC). Avian cellulitis is characterized by the presence of subcutaneous fibrinonecrotic plaques and inflammation of the overlying dermis, resulting in rejection of part or all of the carcasses at processing ^[1,2]. Cellulitis may occur in almost any anatomic area, but most frequently involves the abdomen, breast and thighs, the regions with loose subcutaneous connective tissue. Cellulitis is important not only from the sanitary point of view, since it could potentially represent a public health issue, but is also relevant in terms of animal welfare ^[2,3]. Cellulitis, from the dermatopathological point of view, is a severe inflammation of the dermis and hypodermis, often involving the fascial planes due to an infective, generally bacterial cause ^[4]. Cellulitis is a necrotizing soft-tissue infection caused by a monomicrobial or a mixture of aerobic and anaerobic bacteria that act synergistically.

AVIAN CELLULITIS

Chain of Infection

A technopathy results from the interaction of an agent, a host, and an environment. It is a complex, dynamic, and changeable phenomenon that includes host, microbial and environmental factors. The chain of infection can be illustrated with six different links: pathogen, reservoir, portal of exit, means of transmission, portal of entry, and the new host. Each link has a unique role in the chain and each can be interrupted, or 'broken', through various means (<http://study.com/academy/lesson/chain-of-infection-definition-example.html>).

AGENT

Avian cellulitis in broiler chickens is primarily caused by avian pathogenic *E. coli* (APEC), a monomicrobial necrotizing infection ^[5]. An APEC is defined as an *E. coli* isolated from an extra-intestinal infection of birds. The APEC pathotype possesses a characteristic set of virulence factors that modulates the virulence ^[2,6]. A genotypic assessment of cellulitis isolates revealed that all of them harbour virulence factors related to adhesion, iron acquisition and serum resistance, which are characteristic of the APEC pathotype ^[2].

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The most common bacteria isolates are: *Pasteurella multocida*, *Pseudomonas aeruginosa*, *Enterobacter agglomerans*, *Proteus mirabilis*, *Proteus vulgaris*, *Streptococcus dysgalactiae*, *Trueperella* (formerly *Actinomyces*) *pyogenes*, *Staphylococcus* with different species such as: *S. aureus*, *S. delphini*, *S. intermedius*, *S. xylosum*, *S. saprophyticus*, *S. gallinarum*, *S. lentus*, *S. epidermidis*, *S. haemolyticus*, *S. felis*, *S. capitis*, *S. capitis* sub spp. *urolyticus*, *S. hominis*, *Mannheimia haemolytica*, *Clostridium septicum*, *C. perfringens*, *C. sordelli*. These bacteria have been isolated in association with APEC or in polymicrobial avian cellulitis [7,8].

HOST

Not all poultry are equally susceptible to cellulitis [9]. Different anatomical and functional features have been studied and identified. Host-specific predisposing factors are: the alkaline skin surface pH conditions in certain broiler strains, the mechanical weakness of the dermal layer, demonstrated by loose extracellular matrix components in certain strains. The skin, therefore, is fragile, as the dermis is the structure that confers the biomechanical features to the skin [10]. Furthermore, heterophils isolated from certain broiler breeds tend to have both lowered phagocytic and microbicidal activity [9].

ENVIRONMENT

Most frequently, cellulitis is accompanied by a disruption of the integrity of the skin, such as a traumatic wound or other cutaneous abrasion, enabling a variety of bacteria to enter the wound and colonize the subcutaneous tissue.

Trauma to the skin is necessary for initiating disease, and bruising, mutilation, injurious behavior, cannibalism, hysteria, biting insects, bad litter quality, bad ventilation, high stocking density, poor feed quality and ratio, aggressiveness, neurosis, lameness are all predisposing factors of avian cellulitis [11,12]. Cellulitis has been experimentally induced by artificially causing small dermal scratches on the skin of broiler chickens, which were then placed on litter seeded with avian cellulitis-associated *E. coli* [11].

Avian cellulitis arises when a loss of integrity of the skin (predisposing factor) in a susceptible host (meat-type chicken) is colonized and infected by an APEC characterized by a well-defined pathotype.

GROSS DESCRIPTION

Avian cellulitis is a severe, subacute to chronic, focal to locally extensive fibrinoheterophilic, granulomatous to necrotizing cellulitis, panniculitis, dermatitis and myositis with irregular hyperkeratosis (**Figures 1a–1f**).

The gross features of cellulitis include essentially four parameters that should be taken into account: the colour and texture of the skin, shape and distribution of changes. The colour can be normal, reddened (erythema), purplish (hyperemia and haemorrhage), violaceous (haemorrhage), greenish (chronic haemorrhages or gangrenous exudate) or brown. The presence of scales, fissures and/or a crust or ulcers is frequent. The described discolourations can be associated and irregularly overlapping. The texture, defined as the visual and especially tactile quality of a cutaneous surface, can be irregular, pebbly, or rough. The palpation can also reveal loss of elasticity and mobility, fluctuant turgor, crepitus, rubbery texture, skin sloughing or detachment of the skin from subcutaneous layers, with fetid and foul watery discharge or serosanguineous oozing. In layman's terms, the gross appearance of cellulitic lesions has been defined as 'waffle/honeycombed' [13].

The shape is typically irregular, asymmetrical, poorly demarcated. The most common distribution of cellulitis is the abdominal area [13]. It should be emphasized that in experimental cellulitis, lesions could appear far from the inoculation site, sometimes migrating from the dorsal region to the abdominal area [14]. The removal of skin (epidermis and dermis) reveals the presence of the characteristic plaques, 'flakes' or caseous exudate, yellowish to green, dark red or brown, that can be intensely fetid if gangrenous.

The gross pathological hallmark of avian cellulitis is an asymmetrical change of skin texture with uneven discolouration.

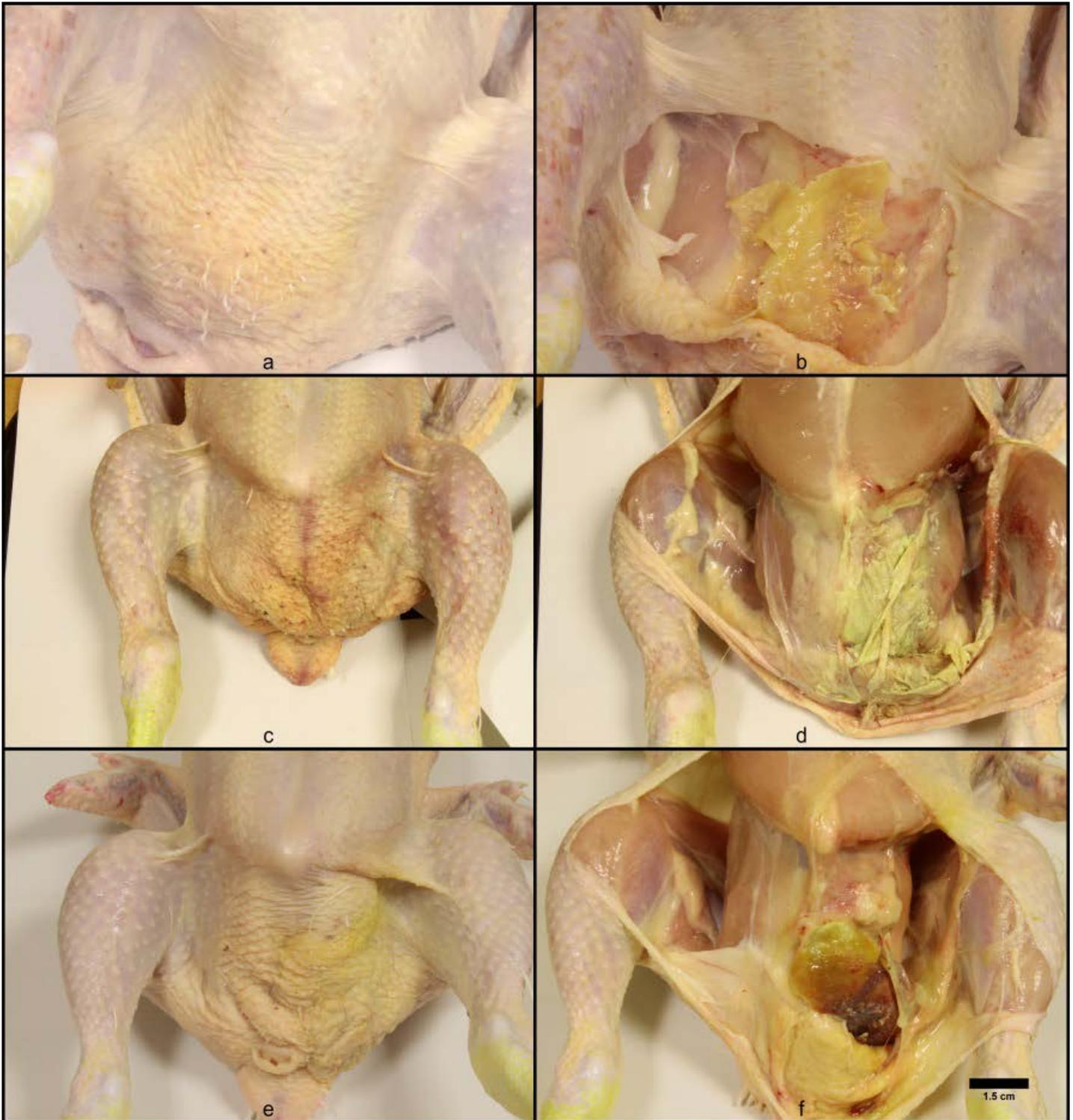


Figure 1. Meat-Type Chicken: (a) Abdominal defeathered skin: classical macroscopic appearance of avian cellulitis, focal yellow asymmetrical discoloration, rubbery texture with hyperkeratosis; (b) Removal of the skin from the abdominal region: revealing a fibrinoheterophilic plaque; (c) Abdominal defeathered skin: variegated and asymmetrical discoloration of the skin in avian cellulitis; (d) Removal of the skin from the abdominal region: revealing a greenish caseous plaque; (e) Abdominal defeathered skin: focal, yellow to green asymmetrical discoloration of the skin; (f) Removal of the skin from the abdominal region: fibrinohaemorrhagic plaque replacing focally the subcutaneous layers.

CONFLICT OF INTEREST

Authors have no conflict of interest to declare.

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