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INCLUSION BODY HEPATITIS: A RE-EMERGING DISEASE

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Inclusion body hepatitis (IBH) is an economically important disease of commercial chickens, especially broilers, which was first reported in the USA in 1963.

It was described as a necrotizing hepatitis in 7-week-old chickens associated with intranuclear inclusion bodies in hepatocytes. Subsequently, it was shown that the disease was associated with fowl adenovirus (FAV) infection.

IBH has been recognized in numerous countries/regions of the world and is reappearing as an important contemporary poultry health problem. It is characterized by a sudden onset of mortality which peaks in 3-4 days and usually stops by the 5th day.

However, mortality occasionally continues for 2-3 weeks. Morbidity is low and sick chickens are in what seems to be a crouching position with ruffled feathers and die within 48 hours or recover. Historically, mortality has usually been no more than 10 percent, but occasionally can be as high as 30 percent. However, this feature has changed in the last 10 years. IBH usually is observed in broilers 3-7 weeks of age, but it had been reported in chickens as young as seven days of age and as old as 20 weeks of age.

Twelve serotypes (to date) of FAV have been isolated from chickens. Numbers of FAV have been isolated from both healthy and diseased flocks and this has resulted in differing opinions regarding their role as pathogens. There have been variable results when attempting to reproduce IBH with FAV isolates and marked differences in virulence have been demonstrated among isolates of the same and differing FAV serotypes.

Numerous field and experimental studies have emphasized a predisposing role for infectious bursual disease (IBD) and chicken infectious anemia (CIA) in IBH. Additionally, other studies have shown that simultaneous infection of chickens with FAV and chicken anemia agent (CAA) resulted in hepatitis and anemia, a condition which corresponds well with the IBH that was seen in the 1960s and 1970s.

Based on field and experimental evidence pointing to FAV acting as secondary pathogens (that is, as a result of primary immunosuppression) in a multifactorial disease (for example, IBDV + CAA + FAV = IBH), it is not surprising that the use of IBD vaccines markedly reduced outbreaks of IBH in the USA and other areas of the world.

In the late 1980s, a previously unknown disease having many similarities to IBH spread throughout Pakistan. The disease initially was observed almost exclusively in 3-6 week old broilers, although it has occasionally been seen in breeder pullets and layers. It is characterized by the accumulation of straw-colored fluid in the heart sac, severely affected liver and kidney, minimal morbidity, and 20-75 percent mortality. The heart lesion gave rise to the name "hydropericardium syndrome (HPS)," but it could well be a different form of IBH because the important signs, gross lesions, and histological changes associated with HPS are very similar to IBH.

At about the same time a HPS-like disease was reported in Mexico; hepatitis, hydropericardium, and high mortality being common features. This disease, which was reported as IBH, has subsequently been observed in Venezuela, Ecuador, Peru, and Chile. HPS has also been recently reported in Iraq, Kuwait, and India.

Many of the IBH/HPS outbreaks in these widely separated regions of the world have been found to be associated with serotype 4 FAV. The fact that there has been higher mortality and a much higher incidence of hydropericardium associated with these IBH/HPS outbreaks than has been the case, historically, with classical IBH (that is, primary or concurrent immunosuppression; secondary IBH) supports the need for a better understanding and knowledge of this disease syndrome.

In addition to the apparent changes in the clinical and pathological features of IBH during the past decade, new field and experimental findings have demonstrated that FAV can be primary pathogens. For example, there have been peracute IBH outbreaks (in broilers 7-21 days of age) in Australia and New Zealand in which there is no evidence of concurrent IBD and/or CIA.

Serotype 8 FAV were commonly isolated from affected flocks in both of these locations. Vaccination of broiler breeders with serotype 8 FAV in Australia has successfully protected broilers against peracute IBH outbreaks. What is the significance of the changes observed in this disease syndrome and associated causative agents during the past decade? Are there increasing numbers of highly pathogenic FAV arising in nature?

Do some of FAV serotypes (e.g., serotype 4) have greater tropism for certain organs (e.g., the heart) than other isolates/serotypes, or are they more virulent and heart-associated when interacting with other agents (e.g., IBDV and/or CAA)?

The fact that this disease syndrome(s) is changing and spreading should be of concern for the poultry industry of the USA, as well as other countries, as trade barriers are rapidly disappearing (for example, NAFTA). It is imperative that research on these syndromes and their associated disease agents be initiated so that poultry diagnosticians can rapidly recognize and distinguish these syndromes (if differences truly do exist) and will have the means (e.g., vaccines) to control them.

Research on IBH has been initiated at the Animal Diagnostic Laboratory at Penn State University and will initially be focused on observing and comparing the clinical and pathological features of this disease syndrome in experimentally inoculated chickens.

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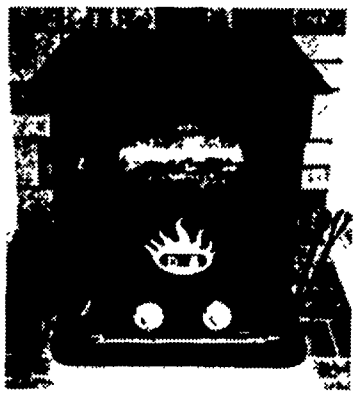

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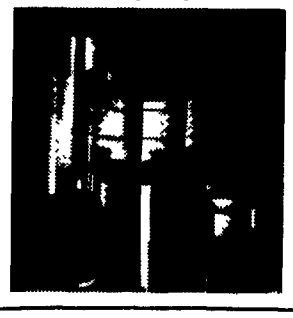

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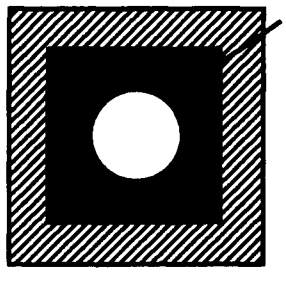

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