LUMPY SKIN DISEASE—A REVIEW

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The creeping pandemic

Lumpy skin disease is a serious skin disease of cattle caused by a single strain of capri-pox virus closely allied to the viruses of sheep and goat pox and known as Neethling virus. After a sudden first appearance late in 1929 in Northern Rhodesia (Zambia) it spread throughout Africa over the following 50 years and is still limited to that continent.

Few infectious diseases of livestock have suddenly emerged in recent times with no prior history of their existence. It is now almost sixty years since lumpy skin disease was first seen in central Africa. Towards the end of 1929 and after the onset of the rains a peculiar skin disease of cattle appeared in Northern Rhodesia. Its like had not been seen before neither by the colonial veterinary department nor by native cattle owners. There were no records in the literature of any comparable condition and the disease was treated at the time rather as an oddity as it did not appear to cause much loss. Fourteen years passed before it reappeared in the north west of neighbouring Bechuanaland (Botswana). Here in the cattle raising area of Ngamiland it caused more concern and soon spread throughout the country. It became known for a while as Ngamiland disease without anything further being known of its nature. In 1944 it was seen in the Marico district of the Transvaal. It was now poised at the edge of the Union of South Africa which had good communications and a well developed cattle industry with many exotic cattle. Within three years an explosive epidemic had occurred. All parts of the Union had experienced the disease and around eight million cattle had been infected. The infection spread to the north probably through Mozambique and Tanganyika (Tanzania) and an epidemic occurred in Kenya in 1957. The next 14 years saw no obvious spread in east Africa but by 1971 cases were being seen in south western Sudan. Within three years another explosive leap saw it move through Chad, Niger and Nigeria where it became widespread by 1974; in 1976 it reached the Ivory Coast. Half a century after its first appearance it had covered the continent by a combination of very slow spread and sudden extensive epidemics. The disease is now endemic in many parts of Africa and regional outbreaks occur periodically. There have been no confirmed cases outside the continent of Africa.

Transmission and spread

It has been established that direct contact plays little part in transmission and that a number of genera of biting flies are responsible for spread. Virus has been isolated from Biomyia fasciata and Stomoxys calcitrans caught feeding on infected cattle. Outbreaks of disease in Kenya and Chad have been associated with unusually large numbers of Culex mirificus and Aedes natronius in the former epizootic and flies of the genus Lyperosia in the latter.

Resurgence of disease has been consistently associated with the onset of rains and the emergence of large numbers of vectors. Outbreaks subside and disease

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tends to disappear at the end of the rainy season. Unusually heavy rains have usually preceded epidemics. Early outbreaks in southern Africa were frequently restricted to water courses in dry weather and rapidly spread when heavy rains fell suggesting a close relationship with vector distribution. Cattle movement undoubtedly plays an important part in the spread of disease whether by trek routes or along roads and railways.

It has been a feature of the disease that epidemics recur after an interval of five or six years in an unvaccinated cattle population. No reservoir host apart from cattle has been found to be a likely carrier of infection in the inter-epidemic period. Natural cases have not been seen in game animals although an impala (Aepyceros melampus) and a giraffe (Giraffa camelopardalis) have been successfully infected experimentally. Extensive serology in game animals has given largely negative results and has led to the conclusion that game plays no significant part in the perpetuation of the disease. In the absence of conclusive data on the existence of a reservoir host either vertebrate or invertebrate it must be concluded that infection persists in a region at a low level in the cattle population itself.

Economic significance

Losses mostly accrue from morbidity. Recorded morbidity rates have varied greatly from as low as 5% to 100%. Many have undoubtedly been underestimates. Mortality, except in exceptional circumstances, rarely rises above 5%. There would appear to be no breed resistance and Bos indicus and Bos taurus breeds are equally susceptible. Fever and general malaise cause weight loss and drop in milk yields which can fall by 50% or more in affected herds. Secondary infections of the skin result in further debility and concurrent purulent mastitis accentuates the fall in milk production. Loss of quarters can occur. Lung lesions increase debility and often result in culling. Abortions, probably due to fever, are not uncommon in the early stages. Temporary sterility in bulls has been recorded. Because lesions affect the full thickness of the skin losses through rejection of hides can be considerable. Secondary bacterial infection would seem to be responsible for most of the illness and loss of production. Antibacterial therapy particularly in the early stages is probably beneficial but no record of the economics of such treatment is available.

Diagnosis

As with many animal diseases the clinician can diagnose the condition with a high degree of certainty. Misdiagnosis and misreporting have probably been common over the years due to veterinarians not having had previous experience of the disease. There is really no other skin condition which so resembles lumpy skin disease that confusion should occur. Affected animals should be given a thorough clinical examination and the whole skin surface palpated. This should be followed by a similar examination of the remaining cattle in the herd not obviously affected. Lesions can often be palpated when they cannot be observed. Changes in the skin are preceded by malaise, anorexia and fever of up to 41°C. A generalised lymphadenitis develops. Oedema of the legs and brisket and the occasional abortion may be seen at this stage.

The appearance of the skin lumps which characterise the disease follows within 48 hours. They vary in size but are mostly between 1 and 3 cm in diameter by 1 to 2 cm deep. They are quite hard in consistency and affect the full thickness