A MYCOSIS OF TURKEYS.


A specimen of the head of a turkey, showing pathological lesions, was sent to these laboratories by Dr Caverhill, Blantyre Mission, Nyasaland.

The owner of the bird—a poultry breeder in Nyasaland—in a letter to Dr Caverhill stated that many of his young turkeys had suffered from a disease in which lumps appeared on their heads and necks and occasionally on their knee joints. The disease apparently had predilection for young birds, usually attacking those between six days and seven months old, with a case mortality of 75 to 85 per cent.; older birds, however, rarely acquired the malady.

The owner further stated that the prophylactic measures employed, viz., isolation and disinfection of the bird coops, had failed either to prevent or eradicate the disease, and that the various disinfectants applied to the lesions had proved a therapeutic failure.

The specimen sent for examination had been preserved in formalin and represented the head of a turkey, six weeks old, which had been afflicted with the disease for a period of ten days.

Macroscopical examination showed the presence of numerous nodules situated on the supra-, infra-, and inter-orbital regions, as well as on the lower surface of the inferior maxillae. In the inter-orbital region many of the nodules had coalesced to form an irregular-shaped excrescence at the base of the superior maxilla. No lesions were visible in the mouth.
The nodules were of a creamy yellow colour and of a fibro-caseous consistence, being firmly connected to the skin but in no way attached to the subjacent bone. They averaged about half a centimetre in diameter, and were more or less round in shape, somewhat resembling millet seeds (fig. 1).

The older nodules when examined with a hand lens presented a bossed appearance, due to projections and intervening depressions.

**Histopathology.**—Sections of the nodule stained by haematoxylin and eosin showed that there was proliferation of the stratum corneum, but in some parts this stratum had given way owing to the existence of necrosed areas or ulcers which formed depressions on the surface (fig. 2).

Between the stratum corneum and the prickle-cell layer there was an extensive, and, in parts, very dense cell infiltration, consisting chiefly of leucocytes, plasma cells, round cells, and connective tissue cells; in many areas, this cell infiltration had invaded the prickle cells and reached the cutis (fig. 2). There was an increase and hyperplasia of the prickle-cell layer (acanthosis). The more superficial cells of this layer appeared swollen and oedematous and had undergone degenerative changes, while the nuclei of those more subjacent showed mitoses.

A special feature noted in the cutis was the cell infiltration, in which plasma cells predominated.

Sections stained by Gram-Weigert's method gave some indication as to the etiology of the condition. In the necrosed areas or ulcers dense masses of coccal bodies, morphologically resembling the spores of a fungus, could be seen (fig. 3). They retained Gram's stain, were circular in shape, and varied in size from 7 µ to 12 µ in diameter. The larger spores apparently had a double-edged contour, enclosing a paler-staining central area. They were most numerous in the
base and margins of the ulcerated areas, but were also scattered irregularly throughout the lesion, in areas extending between the prickle cells to the dermis proper, where they occurred either singly or in groups of three or four spores (fig. 4). In the more superficial parts of the lesions they were found in dense aggregations limited by a wall or capsule, such aggregations resembling ascospores (fig. 3). Sections from numerous nodules were examined, but fungal filaments were never found.

Remarks.—Macroscopically and histopathologically the lesions showed a striking similarity to those of Epithelioma contagiosum avium, a disease with an obscure etiology, attributed by some workers, notably Marx and Sticker (1), to a filtrable virus, and by other observers—Borrel (2), Burnet (3), and Lipschütz (4)—to the presence of very minute micrococci existent in the lesions. Illustrations of these micrococci are figured in the articles published by Burnet and Lipschütz; their size and resistance to ordinary methods of staining exclude the possibility of their resembling the coccal bodies described in this paper. As already mentioned, these coccal bodies were constantly present in the various nodules examined, and indeed were the only organisms noted—evidence certainly suggestive of their being the causal organism of the disease. In the more superficial areas they were massed in aggregations resembling the ascospores of a fungus. Short of cultivations, however, it was not possible to state what species of fungus they were related to.

The possibility of the condition being one of fowl favus or white comb, caused by Lophophyton gallinarum Matruchot and Dassonville, was considered, but the absence of the typical disc-shaped thick scabs, depressed in the centre, on clinical grounds alone ruled out this disease, while microscopically the histopathological changes and the absence of hyphal filaments formed further points in the differentiation from fowl favus.

With the limited pathological material sent it was impossible to prove the presence of a systemic infection and so account for the high mortality of the birds.
In all probability this mycosis, as in fowl favus, assumed a fatal form when extending to the feathered portions of the skin, death being preceded by cachexia and diarrhoea.

REFERENCES.

MORTALITY IN CAMELS CAUSED BY INGESTION OF SAND.

By Major F. E. Mason, R.A.V.C., Sub-Director and Veterinary Pathologist, Veterinary Service, Ministry of Agriculture, Cairo.

Early in April 1916 I was required to investigate the cause of a high mortality in two Indian camel corps, which were at that time stationed on the sandy desert at Salhia. The mortality started about ten days after arrival in that locality, and a large number of deaths following a short illness had occurred. The rate of mortality at the time of my visit was about one camel per hour.

The symptoms seen during life were: profuse vomiting, diarrhoea, tympanites, colicky pains, and depression. In most cases the temperature remained normal, but in some cases it was elevated. In many fatal cases there was no rise of temperature before death. The temperature was little use as a guide as to termination. In some cases the animal was found dead, no symptoms having been observed.

Autopsies conducted on sixteen camels showed the following lesions:

Peritoneum: slight patchy inflammation. Rumen: contained food mixed with sand; the so-called “water-pouches” were also filled with sand, and in some cases sand was found in these only, and in small quantity, causing inflammation of the lining membrane. Reticulum: in most cases intensely inflamed, with sand present between the papilla. Abomasum: deeply inflamed, particularly at the cardiac extremity, where an accumulation of sand was found and the membrane had an excoriated appearance; contents coated with mucus. Intestines: patchy haemorrhagic inflammation, particularly at those parts which normally contain solid or semi-solid ingesta; contents frequently gritty, but not always. Heart: haemorrhagic patches. Lungs, kidneys, spleen, and liver normal.

Specimens were collected and examined—blood, peritoneal impressions, mesenteric glands, intestinal scrapings, etc.—and all gave negative results for pathogenic organisms.

Enquiry showed that the camels in question came from a region in India where sand does not exist.