MELIOIDOSIS

REPORT ON A FATAL CASE IN A BRITISH SOLDIER

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Introduction

ALTHOUGH melioidosis may be regarded as endemic in South East Asia, it still appears to be an uncommon disease in Europeans. Recently, cases in Europeans have been reported from Australia by Rimington (1962) but the recorded number is still small. Stanton and Fletcher (1932) in a review of the known cases up to that date found only six Europeans in a total of 83 cases from Burma, Malaya, French Indo-China and Ceylon. The most recent report from Malaya (Khaira et al. 1959) is concerned with three non-European cases and the Annual Reports of the Institute for Medical Research, Federation of Malaya, note only one European case in the years 1953 to 1960. The Annual Report for 1957 mentions the case of a 41-year-old European who presented with osteomyelitis of the femur, toxæmia and a typhoid-like state. Death followed a lung infection and Pfeiferella whitmori was isolated from the sputum.

As far as is known, no case has previously been reported among British troops in this area; this report deals with a fatal case occurring in a British soldier.

Case History

A 38-year-old British N.C.O., who had arrived in Malaya in September, 1961, was admitted to hospital on 23rd November, 1961, with a 48-hour history of severe headache, fever, muscle pains and a slight non-productive cough. He had not left the urban area of Kuala Lumpur and had had no jungle experience.

On admission he had a fever of 101.6°F. and a pulse rate of 116, but, apart from a dry and coated tongue, no other abnormal signs were elicited. In particular the lung fields were clear on auscultation, there was no generalized lymphadenopathy and neither the liver nor the spleen was palpable.

The white blood count (W.B.C.) was 17,200 per c.mm. (neutrophils 94 per cent). Blood slides were negative for malarial parasites. Urinalysis showed albuminuria and culture later was reported as growing staphylocoCci only. Throat swab revealed commensals only. A radiograph of the chest showed a faint opacity in the left lower zone.

There was little change in his condition on 24th November, but he then complained more of muscle pains, especially in the back and calf muscles, and his cough was more prominent although still non-productive.

Routine investigation of the fever continued, blood being withdrawn for culture and for agglutination tests (Widal, Weil-Felix, brucella and leptospiræ). The W.B.C. was now 10,000 per c.mm., with 93 per cent neutrophils. A repeat culture of urine showed a mixed growth of staphylococci, diphtheroids and coliforms sensitive to streptomycin and chloramphenicol.
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Figure 1. Lung (×50) showing muco-abscesses.

Figure 2. Liver (×50) showing muco-abscesses.

Figure 3. Spleen (×50) showing muco-abscesses.
A penicillin regime was commenced on 24th November with an initial dose of 1 mega-unit followed by 500,000 units every 6 hours. There was no improvement on 25th November. Fever up to 103°F continued and muscle tenderness was quite marked. There was also some abdominal tenderness but neither the liver nor the spleen was palpable. A few septic spots were noted on the buttocks and, on this date, crepitations became audible in both lower lung zones.

Vomiting commenced on 25th November and he was incontinent of faeces, the motions being dark and loose. Examination of the stool showed the presence of occult blood, but microscopy revealed no cysts, amebae, etc., and culture was sterile. A further radiograph of chest on this date showed extensive mottling in both mid and lower zones. At this stage chloramphenicol was added to the regimen, 500 mgm. being given intramuscularly every 6 hours. In spite of this, deterioration continued, and on 26th November he was still pyrexial and now disorientated. The pulse rate had increased to 130 per minute and was irregular, and the blood pressure which had previously been recorded as normal showed a systolic fall and was 90/60. Cyanosis of the lips was present. There was slight improvement with decrease of cyanosis when he was nursed in an oxygen tent and was given digoxin intravenously. Some sputum was obtained on this date, and culture showed a mixed growth of coagulase positive staphylococci, monilia and diphtheroids. The staphylococcus was sensitive to chloramphenicol. There was some spread of the rash previously noted on the buttocks to the limbs; this was mainly papular but some purpuric elements were also noted.

As vomiting was troublesome, intravenous fluid replacement and drug therapy was introduced on 27th November and, when there was no apparent response to 6 G. chloramphenicol, erythromycin was substituted in doses of 500 mgm. every 6 hours. Steroid therapy was also given, commencing with 100 mgm. hydrocortisone intravenously and followed by cortisone acetate 50 mgm. intramuscularly 6-hourly. There was no response to this regime and the patient became unconscious on the afternoon of 28th November. Death occurred in the early hours of 29th November. An intra-cardiac sample of blood was obtained immediately after death.

**Autopsy Report (Abridged)**

Both lungs contained multiple firm nodules, mainly superficial. They extended into the upper lobes but were most numerous and larger in the lower lobes. The left lower lobe was almost consolidated. When incised these nodules exuded dirty brown sero-purulent fluid. Histology showed typical abscess formation (Fig. 1).

**Gastro-intestinal Tract.** The stomach wall showed scattered petechial haemorrhages and a small necrotic nodule on the curvature near the cardia.

The ileum contained a dark tarry and bile-stained material, which was not free in the lumen but appeared to be in the submucosa; it gave a strong positive test for occult blood. The jejunum showed patchy inflammation which was in no place intense. There was no melena in the large bowel.

**Liver.** Several small necrotic nodules were present in the right lobe. Histology showed abscess formation (Fig. 2).

**Spleen.** The pulp was friable and contained necrotic nodules. Histology showed abscess formation (Fig. 3).

**Kidneys.** Both showed small abscesses in the cortex.

**Bacteriology.** The same organism was isolated from:

(a) Post-mortem swab of pus from lungs.
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(b) Blood culture of 24th November.
(c) Blood culture of intra-cardiac sample at death (29th November).

The identification characteristics were:

**Morphology:** Motile, Gram negative rods with parallel sides and rounded ends, 1—3\(\mu\) × 0.3—0.5\(\mu\). Non-sporing, non-capsulated, showing bipolar staining and occasional acid-fast granules.

**Culture:** agar plate at 37°C—After 48 hours, round convex greyish umbonate colonies 2 to 3 mm. in diameter with entire edge. On further incubation, size increased, pigmentation became more marked, the surface became honeycombed and the edge dentate.

**Blood agar:** no haemolysis, growth similar to that on agar.

**MacConkey agar:** 48 hours, red opaque umbonate colonies 2 to 3 mm. in diameter.

**Nutrient broth:** after 24 hours at 37°C., even turbidity and surface pellicle was produced. Further incubation led to formation of a heavy viscous deposit.

**Gelatin stab:** fuliform growth and surface pellicle formed, with gradual stratiform liquification.

**Potato:** abundant café au lait growth.

**Metabolism:** aerobic, no growth under anaerobic conditions. Growth at 37°C. more profuse than at 24°C.

**Biochemical reactions:** acid, no gas formed in glucose, mannitol, dulcitol and salicin. Lactose, sucrose, maltose, adonitol and sorbitol were not fermented. Acid and clot formed in litmus milk. Indole, M.R. and V.P. reactions were negative, urea was not split, and H\(^2\)S was produced slowly. Catalase reaction positive.

**Pathogenicity:** guinea pigs were killed in under 48 hours by intraperitoneal inoculation of 0.05 ml. of 1/10 dilution of an overnight broth culture. Strauss reaction was produced in 4 days by 1/100 dilution, and at post-mortem examination the peritoneal cavity contained a small amount of blood-stained fluid from which the organism was recovered. The spleen and transverse colon showed small nodules. The organism was recovered from the heart blood.

**Conclusion:** this organism appears to be *Pfeifferella whitmori*. The organism was resistant to penicillin but sensitive to chloramphenicol, streptomycin and erythromycin.

**Discussion**

Natural infection by *Pfeifferella whitmori* has been reported in many animals. Stanton and Fletcher (1932) mentions rats, rabbits, guinea pigs, dogs, cats and one instance each of horse and cow; in Australia the disease has been found in sheep, goats, pigs, horses and cattle (Rimington 1962).

The normal habitant of *Pfeifferella whitmori* appears to be the soil and stagnant water of swamps and paddy fields. Transmission to domestic animals may be by contaminated food or bites, while man possibly contracts the disease through the medium of infected food. However, the exact mode of transmission is not clear although it seems likely that the route is by ingestion with a subsequent bacteræmic and septicæmic spread.

In the case reported here the appearance of the intestinal tract at post-mortem might suggest this series of events, but the source of infection remains a mystery. The only domestic animal in the household was a dog which belonged to the Chinese amah. Through the courtesy of the State Veterinary Service this dog was examined and found to be healthy; the melioidosis agglutination test and the melioidin test were both negative.

A feature of the disease which has been noted by many authors is the likeness of the acute illness to the typhoid state and the low infectivity for human contacts even when pulmonary signs are prominent, as occurred in this case.
Diabetics may be at greater risk, as one out of three cases quoted by Khaira et al. (1959) and two out of six cases reported by Rimington (1962) were diabetics. Besseige et al. (1959) also note hyperglycaemia in one of their two reported cases.

Although chloramphenicol appears to be the antibiotic of choice in treatment and cure has been obtained following its use, the present case did not respond even though the organism isolated was found to be sensitive to this drug.

**Summary**

A fatal case of melioidosis in a British soldier is reported. The case presented as a septicæmia with severe pulmonary complications and did not respond to chloramphenicol. The association of the disease with known animal infection and a possible association with diabetes is briefly noted.

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The pathology and bacteriology of this case was demonstrated by Lieutenant-Colonel R. P. Bradshaw and Major R. J. G. Hart at a Laboratory meeting of the Royal Society of Tropical Medicine and Hygiene, in March, 1962.

**REFERENCES**


Institute for Medical Research, Federation of Malaya Annual Report for 1957, p. 25.

