Pulmonary melioidosis in a patient with diabetes mellitus

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A 32-year-old male diabetic developed an acute episode of progressive pulmonary melioidosis while employed in Vietnam. Ketosis and undue glycosuria frequently noted in diabetics with other acute infections were not recognized in this patient. The patient’s response to combined and long-term antimicrobials was excellent. This approach to therapy is recommended in acute cases and refractory cases of chronic disease.

Melioidosis is a disease endemic in south-east Asia. It has achieved increased recognition in the United States since our participation in the Vietnam conflict and merits our enhanced interest (Gilbert, Moore, Hedberg, and Sanford, 1968).

CASE REPORT

A 32-year-old aircraft employee was admitted to the Veterans Administration Hospital, Martinsburg, West Virginia, on 24 July 1969 as a transfer from a service hospital in Vietnam. His military service in 1961 included sporadic trips to south-east Asia. Diabetes mellitus was diagnosed in 1962 and for several years was under control with 40 units of NPH insulin daily. He spent the last six months of 1968 as a civilian employee of an aircraft corporation in Vietnam. Six months later, in June 1969, he returned to the same work in the same area.

Onset of the present illness began one week after his return with an episode of diarrhoea lasting one week. This was followed by a few days of sweats, chills, fever, weakness, and cough productive of greenish-yellow sputum. The patient was admitted to a service hospital on 11 July 1969, and 13 days later transferred to this hospital. Night sweats, haemoptysis, and foul sputum were denied. There had been a 15 lb (6.8 kg) weight loss but neither undue hyperglycaemia nor ketosis was noted.

The radiographic course was initially progressive, reaching a peak by 29 July 1969 when a loculated abscess was evident in the left upper lobe (Figure). It then regressed with therapy. Laboratory studies on admission revealed a white blood count of 13,000 and a haematocrit of 41. Multiple blood cultures were negative. Tuberculin tests, intermediate and second strengths, and fungal serologies for blastomycosis, histoplasmosis, and coccidioidomycosis were negative.

Admission therapy consisting of procaine penicillin, 600,000 units twice daily, failed to alter the patient’s temperature levels of between 102° and 103° F. On 29 July 1969 a report from the service hospital stated that Pseudomonas pseudomallei was recovered from the patient’s sputum. Drug susceptibility studies of this organism are noted (Table I). The patient was afebrile three days after the start of intravenous chloramphenicol, kanamycin, and tetracycline therapy as recommended by Weber, Douglass, Brundage, and Stallkamp (1969). The last positive sputum culture was obtained on 28 August 1969.

<table>
<thead>
<tr>
<th>TABLE I</th>
<th>ANTIMICROBIAL SUSCEPTIBILITY OF ORIGINAL CULTURE OF PSEUDOMONAS PSEUDOMALLEI</th>
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</thead>
<tbody>
<tr>
<td>Drug</td>
<td>Response</td>
</tr>
<tr>
<td>Penicillin</td>
<td>Resistant</td>
</tr>
<tr>
<td>Ampicillin</td>
<td>Resistant</td>
</tr>
<tr>
<td>Streptomycin</td>
<td>Resistant</td>
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<tr>
<td>Tetracycline</td>
<td>Sensitive</td>
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<tr>
<td>Chloramphenicol</td>
<td>Sensitive</td>
</tr>
<tr>
<td>Cephalothin</td>
<td>Resistant</td>
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<tr>
<td>Hexamine</td>
<td>Sensitive</td>
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<tr>
<td>Kanamycin</td>
<td>Sensitive</td>
</tr>
<tr>
<td>Colistin sulphate</td>
<td>Resistant</td>
</tr>
<tr>
<td>Neomycin</td>
<td>Resistant</td>
</tr>
</tbody>
</table>

Peripheral neuropathy attributable to chloramphenicol was noted on 28 August 1969, and resolved one month after this drug was stopped.
At the completion of kanamycin therapy haematuria (100 red cells per high-power field) was noted. This regressed by 23 September 1969. On 9 November 1969, the patient’s sole antimicrobial therapy was tetracycline, 250 mg four times a day. This therapy was maintained for a period of three months after his discharge on 16 December 1969. He was asymptomatic when last seen on 2 June 1970. Periodic haemagglutination and complement fixation titres from 31 July 1969 to 2 June 1970 are reported (Table II).

**DISCUSSION**

Whitmore and Krishnaswami (1912) originally described melioidosis in 1912. The aetiologic agent is *Ps. pseudomallei* (*Malleomyces pseudomallei*), a bipolar pleomorphic Gram-negative rod (Baumann and Morita, 1967; Green and Tuffnell, 1968) said to be related to *Malleomyces mallei*, the causative organism in glanders. *Ps. pseudomallei* is harboured in the soil, vegetation, and water in the rice-growing areas in south-east Asia (Strauss, Groves, Mariappan, and Ellison, 1969b). Transmission to man is said to be direct from these areas. Laboratory transmission has been described (Green and Tuffnell, 1968). Man-to-man transmission has not been recorded but probably merits further scrutiny specifically in areas where the geographic history may be important in the differential diagnosis, such as the United States. Melioidosis has been found to be endemic in parts of the world other than south-east Asia (Rubin, Alexander, and Yager, 1963). Melioidosis reported from the United States, France, England, Africa, India, and Guam has been in soldiers who served in south-east Asia. There are reports of endemic cases originating in Ceylon, Burma, Java, Malaya, the Celebes, North Queensland, Australia, and the Panama Canal Zone (Rubin et al., 1963).

The skin and the respiratory tract are the original sites of patient infection. The clinical status of chronic melioidosis has been likened to tuberculosis and other granulomatous states. Haemagglutination and complement fixation titres for melioidosis may be positive without other clinical evidence of the disease (Nigg, 1963;
Strauss et al., 1969a). In veterans returning with
this disease it is most likely to be found in the
chronic form (Spotnitz, Rudnitzky, and Rambaud,
1967). The disease may evidence itself as an
asymptomatic state with pulmonary findings or
with the presence of fever, cough, sputa, and slow
progression of disease. Acute pulmonary
melioidosis is marked by severe toxicity and rapid
progression (Prevatt and Hunt, 1957; Cooper,
1967; Sheehy, Deller, and Weber, 1967; Spotnitz
et al., 1967; and Weber et al., 1969). The
septicemic or haematogenous form may be
secondary to skin or pulmonary sites of origin.
Subsequent involvement of most of the other
organs has been noted (Baumann and Morita,
1967; Diamond and Pastore, 1967; Patterson,
Darling, and Blumenthal, 1967; Sheehy et al.,
1967; Weber et al., 1969). The impressive
mortality figures associated with melioidosis have
occurred in reports of its haematogenous form
and to a somewhat lesser degree in the acute
pulmonary phase. Urticaria secondary to this
infection has also been described (Steck and Byrd,
1969). In appropriate circumstances the presence
of haematogenous or acute or chronic pulmonary
infection calls for the consideration of Ps.
pseudomallei as the aetiologic agent. A geo-
graphic history, tuberculin and fungal skin tests,
and fungal serologies are excellent screening
points. Blood cultures should be performed for
diagnostic and prognostic purposes and for a
potential therapeutic approach. The organism is
readily identifiable and should be readily cultured
from the sputum. Where biopsy material is available
it should be examined. Complement fixation
and haemagglutination studies are diagnostic but
their titres do not correlate with the severity of
the disease. It should be noted that with prolonged
treatment an impressive drop was noted in this
patient’s complement fixation titre.

The treatment of melioidosis needs clarification.
There is ample evidence that large doses of
tetracycline offer adequate therapy in the chronic
phase of this illness and should suffice when the
disease is recognized in the great majority of our
Vietnam veteran population with melioidosis
(Gilbert et al., 1968). Concern should also be
given to specific organism susceptibility studies.
Weber et al. (1969) reported nine consecutive
cases of the more malignant, acute disease from
Vietnam. Without the rather formidable therapy,
such as was used in this patient, the first four
patients in his series died and the last five
recovered when it was applied. Diamond and
Pastore (1967) confirm a similar picture.

When one reviews the literature there seems to be
an overlapping of the symptomatology ascribed to acute and chronic melioidosis. This patient seems to have had a limited but pro-
gressive acute illness controlled by therapy. We
suggest that in similar situations combined
therapy be used.

Rimington (1962) reports three cases of
melioidosis in diabetics from North Queensland,
Crotty, Bromwich, Quinn, and Brotherton (1963)
from Darwin, Australia report another case. In
none of these patients nor in the present case
could marked aggravation of diabetes be corre-
lated with the presence of Ps. pseudomallei infec-
tion.

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