Dr Mukerji replies as follows:

I am well aware of the work done by the authors (references 1–3 above) on twitching motility and fimbriation of *A anitratus*. The authors, however, have failed to appreciate my observation regarding the peculiar "pendular type" of motility detected in batch 2 of NCTC 7844 (strain of Schaub and Hauber) which was characteristic of a myxobacterium. Two subsequent batches did not show the same activity, probably because this property was affected by cell wall injury caused by freeze-drying. However they did develop surface gliding movement and other fresh isolates showed flexion and extension motility on our medium. I hope the authors will agree that fimbriae did not produce the latter type of movement? There is other evidence to support NCTC 7844 being a myxobacterium. It secreted viscous gum and showed the characteristic capacity to penetrate soft agar in Stainer's salt agar base on testing for cellulosytic and proteolytic activities. For the former, strips of filter paper were put under the surface of agar and the strain inoculated over the strips. In 2–3 days the strains secreted viscous gum, produced etching around the growth and assumed rod-like morphology with a refractile outline which failed to stain well with Gram's. For proteolytic activity 0-1% skimmed milk was incorporated in the agar; the strain produced superficial pitting of the agar and etching phenomena outside the growth. This occurred within 3–5 days which suggested the capacity of the cells to penetrate soft agar (1%). These findings suggest that NCTC 7844 is a myxobacterium. This could be confirmed by hybridisation experiments with myxobacteria but as far as I know these experiments have not been carried out.

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References


Necrotising granulomatous prostatitis after transurethral resection

We enjoyed the recent article published in the September issue of your Journal by Lee and Shepherd. We would like to add our experience of two cases who also had transurethral resection for benign prostatic hyperplasia and who later had recurrence of obstructive symptoms. Material removed at the second transurethral resection showed an identical histopathological picture to that described by Lee and Shepherd (Figure) and there was also no histological or clinical evidence of other causes of localised or generalised granulomatosis. However, tissue eosinophilia which was mentioned first by Hedelin et al was not a feature in our material. It is of interest to note that the time interval of finding active necrotising granulomata was very variable and in our second case (Table) was after eight years.

As we also support their contention that these granulomata, which bore a striking resemblance to rheumatoid nodules, are the effect of trauma, we would like to postulate that they are related to tissue necrosis caused by diathermy in the initial transurethral resection procedure. The persistence of these necrotising granulomata for a very long time, as in our second and their first case, together with a striking resemblance to rheumatoid nodules, would support the speculation that tissue necrosis is the cause of this reaction as it was hypothesised in the evolution of genuine rheumatoid nodules.

We think by now there is enough evidence in the literature to suggest that the relation of transurethral resection and necrotising granulomatous prostatitis is constant and hope that it will prevent further unnecessary investigations and in some cases empirical antituberculose treatment of the patients in question.

Time intervals between first operation (no granuloma) and second operation (granuloma found) in two cases

<table>
<thead>
<tr>
<th></th>
<th>Case 1</th>
<th>Case 2</th>
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<tbody>
<tr>
<td>Age (yr)</td>
<td>65</td>
<td>78</td>
</tr>
<tr>
<td>Operation 1</td>
<td>Transurethral resection-no granuloma</td>
<td>Transurethral resection-granuloma found</td>
</tr>
<tr>
<td>Interval between first and second operation</td>
<td>3 months</td>
<td>8 yr</td>
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</tbody>
</table>

A large necrotising granulomatous reaction bordered by a pallisading layer of histiocytes (arrows) and a few multinucleated giant cells (arrow heads). Haematoxylin and eosin x70.
References


Granuloma in bladder and prostate after previous operations

I was interested to read the article by Lee and Shepherd describing necrotising granulomas following prostatic ressection, since I have taken a dilettante interest in this subject for some 10 years following the discovery of a similar granuloma in a cystectomy specimen which subsequently proved to be secondary to a previous transurethral resection of a bladder tumour. My experience of prostatic and bladder lesions after transurethral resections gathered over the succeeding eight years was presented at the 1980 International Congress of the International Academy of Pathology in Paris and is essentially in accord with that of Lee and Shepherd.

In my series of 17 cases of tissue from repeat transurethral resections of prostate the time interval varied between three days and four years. In the first two or three weeks areas of frank infarction were seen with adjacent foci of squamoups metaplasia identical to that seen in spontaneously occurring prostatic infarcts. Granulation tissue also present in these early cases contained occasional foreign body giant cells and many eosinophils such that a diagnosis of eosinophilic prostatitis was seriously considered on more than one occasion before the complete history was elicited. Necrotising granuloma identical to those described by Lee and Shepherd were seen in specimens from 18 days onwards and I interpreted the serpiginous central areas of necrosis as the remnants of the areas of infarction and this was borne out by the appearances seen in cystectomy specimens where it was possible to identify the precise location of the granuloma. I agree that such granulomata are sufficiently characteristic to make the true diagnosis and to distinguish them from those seen in granulomatous prostatitis, either tuberculous or idiopathic in nature.

Blood culture symposium

The blood culture symposium in the September 1983 issue of the Journal is of interest. However, may I comment on one aspect of the contribution by BI Duerden on the clinical significance of bacteraemia (p 964). Isolation of Salmonella typhi from the blood of a pyrexial patient is undoubtedly of significance in establishing a diagnosis of acute typhoid fever. However, although not overtly stated, the impression is given that isolation of Salm typhi from blood always has such significance. This is far from being true. Salm typhi may be recovered from whole blood or blood clot cultures in patients suffering from other disease states and from individuals without symptoms.

Such transient bacteraemias occur in typhoid carriers who are intravascular shedders of the organism, probably derived from reticuloendothelial sites where a stable host-parasite relationship has been established. Many of these patients do not excrere organisms either in faeces or in urine. The bacteraemia is not associated with pyrexia and indeed it is quite common to isolate Salm typhi from blood of patients with typhoid fever who are convalescent, apyrexial and symptom free. It is probable that intravascular shedding of organisms from such sites is much more common than is realised simply because routine blood cultures are not done on potential typhoid carriers.

Experimental animal models in mice infected with Salm typhimurium show that an analogous situation exists in this host-parasite relationship.

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References