Capnocytophaga ochracea: an unusual opportunistic pathogen

We read with interest the article by Hawkey et al in which they reviewed Capnocytophaga ochracea, and we report a similar case that had interesting features. A 48 year old man, who had acute myeloid leukaemia, developed neutropenia and mouth ulcers after cytotoxic treatment. He subsequently became ill with fever and septicaemia. Two sets of blood cultures were taken and a course of piperacillin and cefuroxime was started, after which he quickly recovered.

A slender Gram negative bacillus was isolated from only the aerobic 0-1% glucose broth of one set at 48 hours. On subculture, after 72 hours' incubation in 5% carbon dioxide on chocolate agar, 1-3 mm, grey-golden, moist, flat colonies were grown, which had a spreading edge and some knob like projections; also on the blood agar incubated for 72 hours anaerobically, 0.5-1.0 mm, grey-golden, flat colonies were visible that had pitted the surface of the agar. No growth had occurred on the aerobic blood agar on primary subculture, but subsequent attempts at subculture showed that C ochracea will grow in air, forming pinpoint colonies at 24 hours, which, on further incubation, develop a metallic sheen. This observation agrees with that of Kristiansen et al, who doubted the degree of dependence on carbon dioxide of C ochracea, but disagrees with the view of Hawkey et al, who considered carbon dioxide to be an absolute requirement.

Table 1 shows the results of our identification tests.

We agree with Forlenza that these results may vary according to the techniques used; indeed, our peptone water sugar results to galactose, raffinose, and glycerol differ from those of Hawkey et al, and the explanation may be that, unlike us, they used broth base serum water sugars.

Disc diffusion tests for sensitivity to antibiotics were performed on the isolate by the Stokes method using appropriate sensitive controls: Oxford Staphylococcus (NCT 6571); Haemophilus influenzae (NCT 8143); Clostridium perfringens; and Pseudomonas aeruginosa (NCT 10662). The plates were incubated in air and 5% carbon dioxide for 24 hours. The inoculum was standardised, and the medium was sensitivity test agar (Oxoid) supplemented with 5% lysed blood and Vitox (Oxoid). The discs were of the routine strengths advised for the clinical laboratory. Table 2 summarises the antibiotic profile obtained.

In contrast to Hawkey et al, we found C ochracea to be sensitive to the range of cephalosporins, and this may be an important therapeutic point as these cidal antibiotics are often used to treat neutropenic patients as part of a regimen giving broad spectrum cover. This organism needs effective treatment, particularly as it is said to produce a toxic inhibitor of neutrophil activity, which may further reduce the impaired immunity.

The only antibiotic results affected by carbon dioxide were those of the aminoglycosides whose zones of inhibition were smaller in carbon dioxide than oxygen, as expected, but this influenced interpretation (Table 3).

On the basis of these results and considering the dose related toxicity, it would not be advisable to treat infection with C ochracea with aminoglycosides alone but it would be interesting to find out if the aminoglycosides have synergy with the penicillins or cephalosporins, as this combination is often used to treat immunosuppressed patients.

In conclusion, our case again illustrates how C ochracea can cause septicaemia in the neutropenic patient, the source of which may be mouth ulcers. To isolate the organism the microbiologist may have to culture the specimen in carbon dioxide for more than 48 hours on primary isolation. Further reports of the susceptibility to antibiotics of C ochracea would be of interest.

We thank Mr H Malnick of the National Collection of Tissue Cultures, Colindale, for confirming the identity of our isolate, and Professor Barrett for permission to report on his patient.
Postsplenectomy sepsis: the need for lifelong prophylaxis

Dr Evans’s report of three cases of late severe postsplenectomy sepsis1 rightly draws attention to the fact that such sepsis is often rapidly fatal and can occur as long as 45 years after splenectomy.

I feel, however, that the opportunity to discuss the aetiology of this phenomenon was missed. This is of fundamental importance because successful prophylaxis of overwhelming postsplenectomy sepsis depends on rationally based treatment, and it can be argued that Dr Evans’s recommendations were inadequate.

Overwhelming postsplenectomy sepsis is almost always associated with bacteria such as Pneumococcus meningococcus and haemophilus. These bacteria have a polysaccharide capsule, which evades ingestion by most macrophages and enhances their pathogenicity.2 Specialised dendritic macrophages however, have evolved within the marginal zone of the spleen, and these have the ability to take up and present carbohydrate antigens,3 which are commonly of the T cell independent type.

Evidence from studies on animals shows that a specialised subset of B cells, capable of responding to carbohydrate, exists in close proximity to the marginal zone macrophages.4 This subset of B cells is non-recirculating and resides in the spleen. Gaps in the adjacent endothelium allow direct contact between these specialised immune cells and the blood. Thus it can be seen that the spleen is optimally developed to police the blood for the presence of dangerous encapsulated pathogens.

This explains why the response to infections with encapsulated bacteria or to vaccination with pneumococcal vaccines is inadequate after splenectomy as the response by lymph node macrophages is insufficient. Unless a splenunculus is present the patient is permanently vulnerable to such encapsulated pathogens.

It is well known that postsplenectomy sepsis can be fatal in as little as 12 hours from the onset of symptoms: medical attention, however well informed, may arrive too late. It is therefore my belief that it is the responsibility of the physician or surgeon who recommends splenectomy to discuss the implications with the patient, explaining that lifelong prophylaxis with penicillin will be needed and that pneumococcal vaccine will be given before the operation, although the vaccine may only be beneficial for five years. The family doctor should also be alerted.

I have found that, provided the risks and benefits of splenectomy are clearly explained, patients comply very carefully with prophylaxis with penicillin. For those allergic to penicillin, alternatives are available.

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References

Dr Evans replies as follows:

Dr Kay’s comments are apposite and mainly true. It was a desire for brevity which restricted my report. I am more cynical than he. He believes, however, that it is the responsibility of the doctor recommending or performing the operation to explain that lifelong penicillin will be needed. This may be true; but I do not believe that patients, in the main, will take drugs lifelong, or that general practitioners or specialists will prescribe them. These patients all have an abdominal scar: the evidence of splenectomy is there for everyone to see. What is needed is recognition of the risk, not only by doctors but also by the patients themselves.

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Glycosylated haemoglobin

The excellent review by Dr Ian Peacock1 contains some biochemical information which, in my opinion, is not completely correct. The author rightly refers to HbA\textsubscript{IC} as 
$$\alpha_2(\beta-Val-1-deoxyfructose)_2,$$$ but its formation is expressed as:

$$\begin{align*}
{\text{glucose + haemoglobin} \leftrightarrow \text{labile intermediate} \rightarrow \text{HbA}_k,} \\
{\text{HbA}_k,} \\
{\phantom{\text{HbA}_k,}}
\end{align*}$$

A non-enzymatic reaction should lead to the production of a monoglycosylated compound and, to a much lesser degree, of a diglycosylated one. HbA\textsubscript{IC} is really diglycosylated, while the monoglycosylated haemoglobin, when present, is in low concentration.2,3 Thus the above scheme is an oversimplification of the reaction and an equilibrium, producing symmetrical haemoglobin, should be postulated:

$$\begin{align*}
2\alpha_2\beta_{\text{Rho}} \leftrightarrow 2\alpha_2\beta + 2a\beta_{\text{Glu}} \\
\text{HbA}_{\text{IC}} = \frac{\alpha_2(\beta-Val-1-deoxyfructose)_2}{\alpha_2(\beta-Val-1-deoxyfructose)}.
\end{align*}$$

It is unclear whether it plays an important part within the erythrocyte or it derives from in vitro manipulation of the sample. A similar equilibrium is well known for some haemoglobin variants, such as HbS, and generally for haemoglobinins which are modified near the NH\textsubscript{2} terminus of the \(\beta\) chain.

For the reasons given above the heterogeneous glycosylation on some lateral lysines should provide principally monoglycosylated products. In this case the equilibrium can not be postulated and these haemoglobin components should be collectively indicated as:

$$\alpha_2\text{Lys}_{\text{Glu}} \beta_2 \alpha_2\beta-1\text{Lys}_{\text{Glu}}$$

These observations are based principally on theoretical considerations and sup-
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