Letters


1 Chalmers WSK, Farmer H, Evans RT, Woolcock PR. Isolation in McCoy cells of Chlamydia psittaci obtained from the domestic
tic duck (Ana platyrhynchos). Avian Pathol-

2 Meyer KF. The host spectrum of psittacosis—
lymphogranuloma venereum (PL) agents.

3 Page LA, Avian (ornithosis). In: Hofsted
ticaemia. Iowa: Iowa State University Press,
1978.

Capnocytophaga ochracea: an unusual
opportunist pathogen

We read with interest the article by Haw-
key et al.1 in which they reviewed Cap-
nocytophaga 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 sep-
ticaemia. 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% glu-
ose broth of one set at 48 hours. On sub-
culture, 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 anaerob-
ically, 0-5-1-0 mm, grey-golden, flat col-
onies were visible that had pitted the sur-
face of the agar. No growth had occurred
on the aerobic blood agar on primary sub-
culture, but subsequent attempts at subcul-
ture 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.,2 who doubted the
degree of dependence on carbon diox-
ide of C. ochracea, but disagrees with the
view of Hawkey et al.,1 who considered car-
bon dioxide to be an absolute requirement.

Table 1 Results of biochemical tests

<table>
<thead>
<tr>
<th>Biochemical reactions:</th>
<th>Acid production from sugars:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Catalase</td>
<td>Glucose</td>
</tr>
<tr>
<td>Oxidase</td>
<td>+ Sucrose</td>
</tr>
<tr>
<td>Aesculin hydrolysis</td>
<td>+ Galactose</td>
</tr>
<tr>
<td>Arginine dihydrolyase</td>
<td>- Lactose</td>
</tr>
<tr>
<td>Ornithine deoxyraseB</td>
<td>- Raffinose</td>
</tr>
<tr>
<td>Lysine deoxyraseB</td>
<td>+ Malte</td>
</tr>
<tr>
<td>Reduction of nitrate</td>
<td>+ Starch</td>
</tr>
<tr>
<td>chlamydiosis urease</td>
<td>- Dextrin</td>
</tr>
<tr>
<td></td>
<td>+ Glycerol</td>
</tr>
<tr>
<td></td>
<td>+ Mannitol</td>
</tr>
<tr>
<td></td>
<td>- Salicin</td>
</tr>
<tr>
<td></td>
<td>- Sorbitol</td>
</tr>
<tr>
<td></td>
<td>- Trehalose</td>
</tr>
</tbody>
</table>

Table 2 Results of disc diffusion tests

<table>
<thead>
<tr>
<th>Resistant to:</th>
<th>Sensitive to:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metronidazole</td>
<td>Fusidic Acid</td>
</tr>
<tr>
<td>Sulphonamide</td>
<td>Cefoxitin</td>
</tr>
<tr>
<td>Ampicillin</td>
<td>Vancomycin</td>
</tr>
<tr>
<td>Piperacillin</td>
<td>Cefamadole</td>
</tr>
<tr>
<td>Azlocillin</td>
<td>Rifampicin</td>
</tr>
<tr>
<td>Methicillin</td>
<td>Chloramphenicol</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>Cefazolin</td>
</tr>
<tr>
<td>Tetracycline</td>
<td>Cephalin</td>
</tr>
<tr>
<td></td>
<td>Moxalatam</td>
</tr>
<tr>
<td></td>
<td>Ciprofloxacin</td>
</tr>
</tbody>
</table>

Table 3 Sensitivity tests in different gaseous environments

<table>
<thead>
<tr>
<th>Aminoglycoside</th>
<th>Oxygen</th>
<th>Carbon dioxide</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gentamicin</td>
<td>Inter-</td>
<td>Resist</td>
</tr>
<tr>
<td>Tobramycin</td>
<td>Medium</td>
<td></td>
</tr>
<tr>
<td>Netilmicin</td>
<td>Medium</td>
<td></td>
</tr>
<tr>
<td>Amikacin</td>
<td>Inter-</td>
<td>Resist</td>
</tr>
</tbody>
</table>

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 anti-
biotic profile obtained.

In contrast to Hawkey et al.1 we found C
ochracea to be sensitive to the range of
ccephalosporins, and this may be an
important therapeutic point as these cidal anti-
biotics are often used to treat neutropenic
patients as part of a regimen giving broad
spectrum cover. This organism needs effec-
tive treatment, particularly as it is said to
produce a toxic inhibitor of neutrophil
activity, which may further reduce the
impaired immunity.4

The only antibiotic results affected by
carbon dioxide were those of the amin-
glycosides 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 consid-
ering 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
cephalosporins or cephaplorins, as this
combination is often used to treat immnosup-
pressed 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.

We agree with Forlenza3 that these results
may vary according to the techniques and
media 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
Postsplenectomy sepsis: the need for lifelong prophylaxis

Dr Evans's report of three cases of late severe postsplenectomy sepsis3 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.4 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,5 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. 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 splenulcus 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, 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.

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.

Glycosylated haemoglobin

The excellent review by Dr Ian Peacock7 contains some biochemical information which, in my opinion, is not completely correct.

The author rightly refers to Hb Aic as $a_2 (\beta - Val-1- deoxyfructose)_2$, but its formation is expressed as:

$$k_1 \text{glucose} + \text{haemoglobin} \rightleftharpoons k_2 \text{labile intermediate} \rightarrow \text{HbA}_{ic}$$

A non-enzymatic reaction should lead to the production of a monoglycosylated compound and, to a much lesser degree, of a diglycosylated one. HbAic is really diglycosylated, while the monoglycosylated haemoglobin, when present, is in low concentration.23 Thus the above scheme is an oversimplification of the reaction and an equilibrium, producing symmetrical haemoglobin, should be postulated:

$$2a_2(\beta \text{Hb}) + 2a_2(\beta \text{Hb}) \rightleftharpoons 2a_2(\beta \text{Hb})$$

It is unclear whether it plays an important part within the erythrocyte or it derives from in vitro manipulation of the samples. A similar equilibrium is well known for some haemoglobin variants, such as HbS, and generally for haemoglobins which are modified near the NH2 terminus of the a 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 simply be collectively indicated as:

$$\alpha \text{Alygly} \beta \text{Alygly}$$

These observations are based principally on theoretical considerations and supported by evidence presented at a meeting of the Haematology Group of the Royal Society of Medicine.