An Interesting Case of Meningitis:
A Case Report

Background
Meningitis is “acute inflammation of the protective membranes covering the surface of the brain” which can be caused by infection with bacteria, viruses and other microorganisms.

The most common symptoms are headache, neck stiffness and photophobia, associated with fever, confusion and a reduction in conscious level (GCS). Diagnosis is often clinical, supported with results of an assessment of the cerebrospinal fluid (CSF) which is ideally performed prior to the commencement of treatment.

Meningitis can be lethal, and therefore early identification and treatment is required. Below is a case report of an interesting case of meningitis.

Case Report:
A 39 year old Caucasian male was admitted to Emergency department with a few days history of diarrhoea, fever and worsening tiredness. There was no history of travel abroad and he lived alone. Past medical history was of alcoholic liver disease (ALD). On initial examination he was confused. Examination was unremarkable apart from distended non-tender abdominal. Initial investigations showed deranged LFTs, normal inflammatory markers and low sodium. He was treated as decompensated ALD. The same night patient had a fall without loss of consciousness. CT brain was unremarkable. The following day he was found drowsy and irritable. He was pyrexial and demonstrated signs of meningism. Lumbar puncture was performed (table 1). Results were in keeping with a diagnosis of viral meningitis and the patient was commenced on Acyclovir and Ceftriaxone. The patient was transferred to a high dependency bed but GCS and BP continued to deteriorate. The patient was intubated and transferred to ITU towards the end of day 3. He was commenced on inotropic support.

On day 5, repeat CT head examination showed evidence of gross cerebral oedema (Figure 1). His case was discussed with the regional neurosurgical team who recommended a repeat lumbar puncture to check for CSF pressure (table 1).

The patient was continued on acyclovir and ceftriaxone following discussion with the microbiology team. Despite aggressive management there was no improvement in patients condition. On day 10, brain stem testing was performed. The patient was declared Brain Stem Dead and discussions regarding organ donation were done with family and the patient’s kidneys were harvested for donation. The case was discussed with the coroner and no post-mortem was deemed necessary.

CSF PCR (which became available after patients death) was negative for Pneumococcal, meningococcal, VZV, HSV, Enterovirus and Parechovirus. Blood culture was negative on 2 occasions.

Following organ donation, the two recipients of the donated kidneys began with the same neurological symptoms, and were both deceased, with the same clinical picture within two weeks or the donation. Post-mortem examinations reported that both patients brain tissue were “full of nematode larvae” and the parasite was identified as Halicephalobus gingivalis – a very rare cause of eosinophilic meningitis.

Discussion
Eosinophilic meningitis is defined as “a cerebrospinal fluid (CSF) eosinophil count of more than 10/mm³ OR eosinophils representing more than 10% of the CSF leucocyte count, in the presence of clinical signs of meningitis”.

Detection of eosinophils in the CSF requires examination with Wright’s Giemsa (or other appropriate stains) and less than 2% of any antihelminthic drugs in these cases. These infections are not caused by helminths and have a predilection for tissues of the CNS and have a poor response causing progressive mental confusion, lethargy, fever and death as demonstrated in the above case.

Table 1: CSF Findings

<table>
<thead>
<tr>
<th></th>
<th>Lumbar Puncture 1 (Day 1)</th>
<th>Lumbar Puncture 2 (Day 4)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Appearance</strong></td>
<td>Cloudy clear fluid</td>
<td>Yellow thick fluid</td>
</tr>
<tr>
<td><strong>Opening pressure</strong></td>
<td>Not measured</td>
<td>&gt;34mm (off the scale)</td>
</tr>
<tr>
<td><strong>WCC</strong></td>
<td>185</td>
<td>600</td>
</tr>
<tr>
<td><strong>WCC differential</strong></td>
<td>20% polymorphs, 80% lymphocytes</td>
<td>92% polymorphs, 8% lymphocytes</td>
</tr>
<tr>
<td><strong>RBC</strong></td>
<td>5</td>
<td>&lt;1</td>
</tr>
<tr>
<td><strong>Microscopy</strong></td>
<td>No bacteria</td>
<td>No bacteria</td>
</tr>
<tr>
<td><strong>Glucose mmol/mol</strong></td>
<td>2.50 (serum 8.7)</td>
<td>1.60</td>
</tr>
<tr>
<td><strong>Protein g/L</strong></td>
<td>1.51</td>
<td>7.72</td>
</tr>
</tbody>
</table>

Fig 1: CT brain showing marked dilatation of the supra-tentorial ventricular system

References: