Table 2  Significant differences found in stroke and control groups between patients with acute and chronic diseases

<table>
<thead>
<tr>
<th></th>
<th>Stroke patients N = 30</th>
<th>Controls N = 30</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>D</td>
<td>PA</td>
</tr>
<tr>
<td>Acute N = 17</td>
<td>0.99 ± 0.45</td>
<td>0.45 ± 0.39</td>
</tr>
<tr>
<td>Chronic N = 13</td>
<td>1.57 ± 0.78*</td>
<td>0.93 ± 0.6*</td>
</tr>
</tbody>
</table>

Student t test *p < 0.05 (two tailed)

insomnia and sleeping disorders. As for the obsessive-compulsive disturbances, whose values are rather high and hence indicate a fairly characteristic pattern, it should be noted that while the scale mainly measures clearly obsessive-compulsive symptoms such as the inability to get rid of undesired thoughts, words or ideas, the need to check and double check what is done, etc, it also contains questions aimed to evaluate more general cognitive difficulties and could hence be influenced, in the stroke group, by organic symptoms due to the lesion of the CNS. Lastly, we draw attention to the fact that with the passing of time phobic type symptoms often tend to appear in stroke patients.

In conclusion, our data confirm depression as the main form of psychological distress appearing after stroke, but also demonstrate the existence of other response patterns. Since some of these patterns appear to vary in time, longitudinal studies would be of great help in increasing our understanding of the problem.

Guido Magni
Fabrizio Schifano
Clinica delle Malattie Nervose e Mentali dell'Università, Via Giustiniani n5, 33100 Padova, Italy.

References


Accepted 8 December 1983

Toxic shock syndrome presenting as cerebral infarct

SIR: Neuropsychological sequelae such as impaired memory, calculation and poorly sustained concentration have been described after the toxic shock syndrome.1 We report a man whose presentation was with a cerebral infarct.

A 38-year-old taxi driver presented with 12 hours of severe diarrhoea, vomiting and myalgia. Immediately prior to admission he suddenly collapsed in his kitchen. On arrival he was semi-conscious and shocked with a pyrexia 40-0°C, pulse 110 and a systolic blood pressure of 80 mm Hg. He had purposeful movement to pain of the left side of his body but not his right, and the right plantar was extensor. All cultures including blood and lumbar puncture were negative. He was treated with intravenous ampicillin, flucloxacillin, gentamicin and steroids. Within 24 hours he developed respiratory failure with bilateral interstitial pulmonary infiltrates and required ventilation for 8 days. His platelet count fell to 69 × 10^9/l and there was a rise in titre of fibrin degradation products. His urea and creatinine both rose to three times normal. After 36 hours his left leg became cold with loss of left femoral and all distal pulses. These returned within 6 hours of full heparinisation. By the third day he had developed a fine erythematous macular rash, and a peeling scrotal abscess with inguinal lymphadenopathy was noticed. 10 ml of pus from the abscess showed no growth but a skin swab grew a coliform and a non-toxin-producing strain of Staphylococcus aureus. Once off the ventilator he was found to have a mixed motor and sensory dysphasia with a right-sided hemiplegia. A CT brain scan showed a left temporoparietal infarct in the middle cerebral artery territory. A repeat scan after one month was unchanged. On the 13th day his soles and palms desquamated. During the illness his antistaphylocoyi titres and antinucleus titres rose four fold. Normal investigations included serum amylase, viral, mycoplasma, legionella and antistreptolysin-O titres. He went home after 6 weeks.

Although we failed to isolate a Staphylococcus aureus able to produce exotoxin F we feel the clinical picture fulfills the case definition of toxic shock syndrome.2 The abscess was sterile as he had received 2 days of effective antistaphylococcal treatment prior to drainage. We believe this is the second case of scrotal infection causing the syndrome in this country.3 It is not clear why this man should have developed a cerebral infarct. There was no clinical evidence of endocarditis nor blood culture.4 Suggestions for mechanism of neurological damage have included direct toxic, altering permeability of the blood brain barrier or an immunologically mediated vasculitis.5 Large vessel spasm in this case might explain the cerebral infarct and the transient loss of leg pulses. The toxic shock syndrome may still have surprising presentations and should be considered in any septic ill patient, not just in menstruating women.

DA BLACK
DSJ MAW
Kent and Sussex Hospital, Tunbridge Wells, Kent TN4 8AT, UK

References


Accepted 8 December 1983

Creatine kinase BB isoenzyme in rugby football players

SIR: Creatine kinase BB (CK-BB) has been found in high concentrations in the brain. It is found in lesser concentrations in the gut.1 Normally, concentrations
Toxic shock syndrome presenting as cerebral infarct.

D A Black and D S Maw

*J Neurol Neurosurg Psychiatry* 1984 47: 568
doi: 10.1136/jnnp.47.5.568

Updated information and services can be found at:
http://jnnp.bmj.com/content/47/5/568.1.citation

Email alerting service

These include:

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/