SHORT REPORTS

Relief of acute pain in herpes zoster ophthalmicus by stellate ganglion block

Stellate ganglion block has been suggested as a useful treatment for trigeminal herpes zoster,[1-4] being most effective if started within two weeks of the onset of the disease.[1] We evaluated the efficacy of this treatment in a randomised prospective controlled study for the first time and assessed its complications.

Patients, methods, and results

Thirty patients aged 60 and over who presented with herpes zoster ophthalmicus were recruited and randomly allocated to one of two groups: treated (group A; mean age 71·5 (range 60-87) years) or untreated (group B; mean age 72·2 (62-92) years). Pain was assessed by visual analogue scale set horizontally for 100 mm.

Patients in group A underwent stellate ganglion block within 14 days of the onset of the rash as previously described.[5] After verbal consent a 0·5 ml test dose, followed 30 seconds later by a 9·5 ml bolus of equal parts 1% lignocaine and 0·5% marcaine, was injected on to the C5/C6 prevertebral fascia. Effect was assessed from miosis, hyperthermia, and pain relief at 60 minutes. Patients were followed up for at least three months.

In group A mean pain score before block was 41·9 mm; at one hour it was 5·2 mm, a highly significant reduction ($p<0·001$, paired t test). Pain scores in subsequent time bands were compared with those at presentation in patients from each group by paired t tests (table). In group A there was a highly significant reduction ($p<0·01$) in pain scores at two weeks to one month and at six weeks to two months ($p<0·05$). In group B no significant reduction in pain occurred during the first two months ($p>0·05$ for each time band). At three months both groups showed significant reductions, reflecting the generally held view that pain decreases with time in this disease.

Mean visual analogue scale pain scores for patients treated by stellate ganglion block and untreated patients at presentation and at subsequent time bands

	Presentation	2 Weeks-1 month	6 Weeks-2 months	3 Months
Treated	42·53	12·43*	31·45†	10·46*
Untreated	26·67	16·45‡	14·85‡	11·71†

*$p<0·01$; †$p<0·05$; ‡$p>0·05$.

It was not appropriate to compare directly the pain scores of the treated and untreated groups at each time band as initial randomisation resulted in an appreciable difference in pain scores at presentation ($p=0·09$). Lower initial scores may have resulted in a different clinical course in each group, and this made interpretation of two sample t tests difficult. None the less, the highly significant reduction in pain scores after treatment indicates a definite effect of treatment.

Seventeen blocks were performed, two being second procedures. Five patients developed a hoarse voice owing to blockade of the ipsilateral recurrent laryngeal nerve, and there was one case of brachial block; as expected these resolved within eight hours. Two blocks were complicated by a bloody first tap: one patient was successfully treated at another site, and the block was abandoned in the other after a test dose at the second site resulted in upbeating nystagmus. Complications occurred in seven patients (41%) but none were permanent.

Comment

This study shows that stellate ganglion block is effective in relieving pain as shown by the appreciable reduction in pain scores at 60 minutes. The effect persists, greatly reducing acute pain for up to three months after treatment, especially between two weeks and one month afterwards.

The method of action of sympathetic block has not been satisfactorily explained, but it clearly modifies both peripheral and central pathways as pain would otherwise return once the effects of anaesthesia wore off.

The acute pain of herpes zoster ophthalmicus is an important contribution to overall morbidity. We think that stellate ganglion block, if used early, plays an important part in greatly reducing or abolishing this pain and suggest that doctors should be aware of the efficacy of this treatment and refer patients early to practitioners experienced in the use of regional blocks. It should be feasible for patients to be treated as a day case by anaesthetists working on an ophthalmic list.

We thank the consultants of St Paul's Eye Hospital for allowing us to study their patients, the junior anaesthetists from the centre for pain relief for performing blocks, and Mrs D Kinsella for secretarial work.

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St Paul's Eye Hospital, Liverpool L3 9PF
S P HARDING, MB, FRCS, honorary senior registrar (also lecturer in ophthalmology, University of Liverpool)
J R LIPTON, MB, FRCS, senior house officer

Centre for Pain Relief, Walton Hospital, Liverpool L9 1EA
J C D WELLS, MB, FFARCS, director and consultant anaesthetist
J A CAMPBELL, MSC, PHD, medical physicist

Correspondence to: Mr Harding.

Urinary sodium excretion in 4-6 year old children: a cause for concern?

There is increasing evidence that a high intake of salt may be important in the development of essential hypertension.[1] Evidence from animals suggests that a high intake of salt early in life may predispose the animal to high blood pressure when it is again exposed to a high intake.[2] A double blind study of babies showed that blood pressure was lower in babies aged 6 months who from birth had been fed a diet low in sodium compared with babies fed a diet with normal sodium content.[3] Little is known about the intake of salt in early childhood, particularly in the age group 4-6 years, when children start expressing preferences for food. Dietary histories are unreliable for estimating intake of sodium; a more accurate assessment can be obtained from measurement of 24 hour urinary sodium excretion. We therefore measured this in a group of primary schoolchildren aged 4-6.

Subjects, methods, and results

Seventeen pupils aged 4-6 from each of two primary schools were randomly selected (20 boys, 14 girls); permission for the study was obtained from the pupils and parents. Written and oral instructions on how to collect urine over two consecutive periods of 24 hours were given to teachers, parents, and children. We supervised the collections during the day at school. We never mentioned that the salt content of the food was being assessed, and the children consumed their normal diet. We measured urinary volume and sodium, potassium, and creatinine concentrations. The children were also weighed.

The mean urinary sodium excretion was 64 (SEM 3·9) mmol(mEq)/24 h (range 20·5—131·0 mmol/24 h). Mean urinary potassium excretion was 34 (1·7) mmol(mEq)/24 h and mean urinary creatinine excretion 1·99 (0·14) mmol/24 h (225 (16·3) mg/24 h). No results were discarded. The mean body weight of the 34 children was 21·2 (0·62) kg.

The ratio of sodium to creatinine excreted over 24 hours was 39, and the ratio of sodium excreted over 24 hours to body weight was 3·1. These ratios are considerably higher than those found in adults. If sodium excretion in an adult is taken as 150 mmol/24 h with a creatinine excretion of 13 mmol/24 h and a body weight of 75 kg, the ratio of sodium to creatinine excreted is 3·5 times higher in children than in adults and the ratio of sodium excreted to body weight is 1·5 times higher in children.

Comment

Our results show that children aged 4-6 who had already started school had a high average sodium excretion of around 64 mmol/day. This if anything is an underestimate because all the 24 hour collections of urine were included. It is difficult to compare excretion of sodium in young children with that in adults, but if the ratio of sodium to creatinine excreted or of sodium excreted to body weight is taken it is clear that these children had a considerably higher intake of sodium than the already high intake of adults.

In the United Kingdom children at school are increasingly consuming snacks rather than meals. These snacks usually have a high content of sodium and saturated fat with little fibre. This change in dietary habit is likely to increase the already high intake of sodium of young children even further. Whether this high intake predisposes these children to develop high

blood pressure later is not known, but circumstantial evidence in man and direct evidence in some animals suggest that it may.[1] We think that the high intake of salt, as well as other aspects of the diet of young children in the United Kingdom, should be a cause for concern and further research.

This study was part of a paediatric project on the paediatric firm, and we gratefully acknowledge the help of Dr David Murdoch and Dr Michael Latter.

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Charing Cross and Westminster Medical School, London W6 8RF
SALLY DE COURCY, MB, BS, house surgeon
HILARY MITCHELL, MB, BS, house surgeon
DAVID SIMMONS, BA, MB, house physician
GRAHAM A MACGREGOR, FRCP, senior lecturer and honorary consultant physician

Correspondence to: Dr G A MacGregor, Blood Pressure Unit, Department of Medicine, Charing Cross and Westminster Medical School, London W6 8RF.

Acute non-A non-B hepatitis after typhoid fever

Ten years after its initial description non-A non-B hepatitis remains a diagnosis of exclusion. Nevertheless, non-A non-B hepatitis includes infections of the liver caused by at least three viruses provisionally designated "blood transmitted," "coagulation factor transmitted," and "epidemic waterborne."[1] Furthermore, there is some evidence of an association between shellfish consumption and some cases of non-A non-B hepatitis,[2,3] and it has been suggested that these cases reported in Western countries may be sporadic cases of epidemic waterborne non-A non-B hepatitis,[1] a hypothesis supported by an analysis of the clinical and biochemical features in 20 cases.[4] Here we describe two patients admitted to our infectious diseases clinic for typhoid fever who then developed acute non-A non-B hepatitis.

Case 1

A 25 year old man experienced an abrupt onset of fever (39°C), headache, and malaise. He was given acetylsalicylic acid (500 mg every 4 hours). Five days later, because of persisting fever, constipation, abdominal pain, and discomfort, he was admitted to hospital. Because of a history of shellfish consumption 15 days before the onset of symptoms blood and stool cultures were performed and Widal reaction was tested. *Salmonella typhi* was isolated from the blood, and an increased titre of 0 agglutinins was recorded during the second week of illness. Chloramphenicol was started and continued according to the standard therapeutic regimen.

Routine alanine aminotransferase determinations showed a nineteenfold increase over the normal values during the second week of illness (four weeks after shellfish consumption), but without any symptoms. Acute non-A non-B hepatitis was diagnosed by exclusion: he was negative for hepatitis B surface antigen, IgM antibody to hepatitis B core antigen, cytomegalovirus, anti-hepatitis A virus, and heterophile antibody to Epstein-Barr virus. In particular, the assay for IgM anti-hepatitis A virus was repeatedly negative. HBsAg and IgM anti-hepatitis A were assayed by Abbott radioimmunoassays, IgM anti-core antigen by enzyme linked immunosorbent assay Corzyme-M (Abbott), IgM anti-cytomegalovirus by Enzygnost-Cytomegalie (Behringwerke), and antibodies to Epstein-Barr virus by Monosticon (Organon).

The patient recovered completely from both typhoid fever and non-A non-B hepatitis: his serum transaminase activity returned to normal within five weeks and remained normal for more than six months (figure).

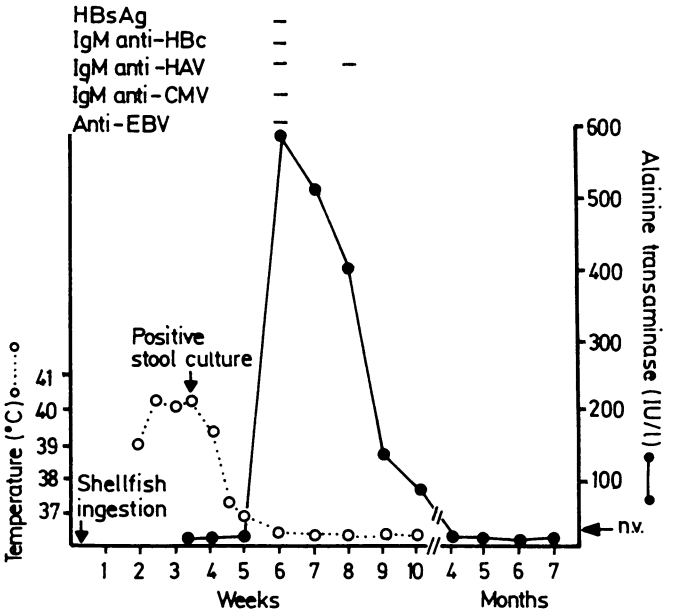
Case 2

A 23 year old man was admitted to hospital with a one week history of high fever (40°C), continuous headache confined to the frontal region, and constipation followed by diarrhoea. He had eaten oysters and mussels 10 days before the onset of symptoms. *Salmonella typhi* was isolated from stools, and treatment with chloramphenicol was started.

At the end of the fourth week of illness (five weeks after shellfish ingestion) there was a sharp rise in his serum transaminase activity with a decrease of prothrombin activity (<70% of normal). The patient was symptom free and anicteric. In this case too acute non-A non-B hepatitis was diagnosed by exclusion. The hepatitis ran an uneventful course with liver function values returning to normal and remaining so over 12 months after the onset of the disease.

Comment

Typhoid fever occurs sporadically in Italy, and shellfish ingestion is often responsible. Occasionally it is associated with acute hepatitis A, which is primarily transmitted by the faecal-oral route. Although faecal-oral non-A non-B hepatitis has been suggested to occur in some patients with biphasic hepatitis A,[5] we are not aware of reports of non-A non-B hepatitis in patients with other stool borne transmitted diseases.



Case 1. Non-A non-B hepatitis after typhoid fever.

The observation of two cases of non-A non-B hepatitis after typhoid fever is intriguing. Although there is no direct evidence that the source of *Salmonella typhi* and non-A non-B virus was the same, the absence in these patients of a history of blood transfusion or admission to hospital within the preceding six months or a history of drug addiction or any other possible parenteral exposure suggests that food contaminated with human excreta might have been the source. In Western countries non-A non-B hepatitis occurs as sporadic cases and such contaminated food could be the source in some of these. This may be a rare event. On the other hand, given the frequent asymptomatic course of the disease, such a source may not be that uncommon since it may not be looked for in a patient with a normal convalescence.

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Infectious Diseases Clinic, University of Milan, L Sacco Hospital, 20157 Milan, Italy
F CAREDDA, MD, clinical research assistant
S ANTINORI, MD, resident
T RE, MD, resident
C PASTECCHIA, MD, resident
M MORONI, MD, director

Correspondence to: Dr Caredda.