formation of 'growing skull fractures'. The peculiarity of our case is the fact that both mechanisms were operative. All the radiological and surgical findings suggest that the water hammer effect at the level of the dural defect probably caused the separation of the bony fracture, while slow circulation through the diploe of thick occipital bone below the torcula level caused the intradiploic cavitation.

D'Almeida and King² described two adult cases both without any history of head injury similar to our case. Both patients presented with asymptomatic osteolytic skull lesions associated with 'cerebrospinal fluid-diploic fistulas'. It was suggested that the diploe of the skull in each patient gradually expanded over many years resulting in gradual elevation of the outer table and flattening of the inner table. The authors concluded that a congenital aetiology of the lesions would be unlikely and postulated that each patient had probably experienced a forgotten head injury resulting in a small fracture of the inner table of the skull with injury to the dura and subsequent development of an arachnoid pouch through the dural defect.

Table 1 shows that most of the cases were reported before MR imaging became available. Only 2 cases with MR images have been reported so far.^{6,7} MR not only helps to distiguish intradiploic arachnoid cysts from dermoid or epidermoid cysts, eosinophilic granulomas, plasmocytomas and metastatic tumors,⁶ but it also gives more detailed information on cerebral tissue and CSF changes than CT, yet unfortunately less information on bony changes than CT.^{6,7,15}

In the treatment of such patients, craniectomy with careful exposition of the intact dural edges and duroplasty is the gold standard. In cysts located in the posterior fossa, the decompression of the cerebellar tissues is important for clinical improvement. As for our case decompression down to the foramen Magnum was found to be necessary which may not be the case in all occipital cysts. Cranioplasty either with autogenous bone or acrylic material is the choice of the surgeon. In hydrocephalic patients a shunt (or rarely an extraventricular drain) will be helpful not only to decompress the ventricles but also to prevent CSF fistula or a pseudomeningocele.

In conclusion, occipital intradiploic leptomeningeal cysts may present during adulthood due to a head injury in childhood. They may compress the occipital lobe and/or the cerebellum and they have to be treated with bony decompression plus duroplasty and (when necessary) complemented with shunt insertion.

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Multiple cerebral infarcts following septic shock

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Summary A 60-year-old female in septic shock developed neurological signs and symptoms. She had left-sided hemiparesis, left homonymous hemianopia, bimanual coordination disorder, a language dysfunction of anomic aphasic type and a non-aphasic right hemispheric communication disorder. Computer tomography demonstrated bilateral anterior and posterior watershed as well as territorial infarctions. Risk factors included chronic airways limitation, cardiac failure and heavy smoking. Carotid duplex studies were normal. The mechanisms can be explained by flow changes and thrombus formation. © 2002 Published by Elsevier Science Ltd.

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INTRODUCTION

Septic shock is usually due to an overwhelming infection with gram-negative bacteria. The diagnosis of septic shock should be suggested by the occurrence of hypotension in the presence of infection. Recognition of sepsis requires a high index of suspicion and an awareness of those conditions that predispose to sepsis. Black and Daws¹ described a patient with toxic shock syndrome presenting as a cerebral infarct and they attributed this to large vessel spasm.

We describe here a patient with a combination of radiological and clinical manifestations due to multiple cerebral infarcts following septic shock. The areas involved represented border zones between the main arteries in the left hemisphere and multiple branch involvement of the main arteries, the anterior, middle and posterior cerebral arteries in the right hemisphere. We believe that haemodynamic and thrombotic mechanisms were involved.

CASE REPORT

A 60-year-old female of Finnish extraction was hospitalised with a history of being unwell for two days and had become unresponsive on the second day. She was a heavy smoker and suffered from chronic obtructive airways disease and epilepsy, for which she had been on dilantin sodium and thioridazine for paraphrenia. On examination, she was comatose, deeply cyanosed (both central and peripheral) and had cold extremities. Her skin turgor was poor. Her pulse was 86 per minute, temperature 36.5°C and blood pressure 65/30 mmHg. She had a systolic murmur at the apex. The lungs were clear. The liver was palpable five fingerbreadths below the right costal margin. The nervous system was intact other than for the disordered sensorium.

Initial investigations revealed the following: haemoglobin 154 g/l, PCV 50%, WCC 16.4 \times 10/l, N 65%, L 6%, M 5% and band forms 24%, platelets 395. The electrolytes, urea, creatinine and serum phenytoin level were within normal limits. The blood glucose was 3.1 mmol/L, CPK 58 units, AST 39 units and on the following day 212 and 172 units respectively. Urine microscopy revealed evidence of a urinary tract infection and blood culture grew *Escherichia coli*. Arterial blood gas analysis showed pH 7.15. pCO₂ 85, pO₂ 28.0, HCO₃ 32.6, base excess –1.9. The electrocardiogram revealed prominent P waves, incomplete right bundle branch block and T wave inversion in leads II, III, AVF and in the anterolateral chest leads. Computer tomography of the brain done on day 1 revealed no abnormality. She was treated with Haemacel, i.v. fluids, naloxone and gentamycin.

On the next day, she was somewhat lighter in consciousness but was confused. On day 3 her pulse was imperceptible for a few minutes, but the carotid pulse was felt. She was found to have mild-to-moderate weakness of her left arm. The blood pressure ranged from 110/30 to 110/60 mmHg. Chest X-ray revealed enlargement of the cardiac shadow, prominent pulmonary arteries and basal congestion. Thereafter, she steadily improved and by day 7 was seated out of bed when it was noticed that she had a weakness of the left side of her body. A CT scan on day 12 revealed changes described below.

Examination of the nervous system revealed a left-sided hemianopia, a left-sided hemiparesis with weakness of the extensors of the upper arm and lower limb, the leg more than the arm with sparing of the face. Hand squeeze was normal but she had decreased facility with fine finger movements and reciprocal coordination of both hands (simultaneously opening one fist while closing the other) could not be performed. Bimanual tasks such as eating and dressing were not affected. There was no grasp reflex, forced grasping, grasping movements or signs of callosal dysfunction. There was no difficulty in identifying objects by sight. She had speech disorders as described below.

Communication skills were assessed using subtests of the Boston Diagnostic Aphasic Examination² administered through a Finnish interpreter. Speech was fluent and grammatical with occasional verbal paraphasias on testing. Auditory comprehension was reasonably intact, except for difficulties with lengthy test items, the major feature being the word-finding difficulty. Limitation in formal education was reflected in the limited skills which made reading and writing assessment difficult. In conversational contexts, a lack of substantive words were noted at times and there was a tendency for the patient to fail to maintain the conversational topic, characteristics observed in those with right hemispheric lessions.

Transthoracic echocardiography demonstrated right heart volume overload with moderate right auricular and right ventricular enlargement. Moderate tricuspid incompetence was demonstrated on Doppler sampling of the right atrium, which would explain the right heart enlargement. No left-sided valvular abnormality or thrombus was evident. Duplex scan of the carotid arteries revealed no abnormality.

The computerised tomographic (CT) areas were determined by the guidelines of Damasio.³ The cranial computerised tomographic studies on day 12 (subacute stage) showed hypodense areas involving the cortex and deep matter in the right fronto-lateral and left parieto-occipital regions. On the contrast-enhancement CT, enhancement was seen in the grey matter of the left dorso-lateral and right occipital regions (Fig. 1). CT scan done 8 weeks later (chronic stage) later



Fig. 1 CT scan on day 12 shows hypodense areas in the right fronto-lateral and left parieto-occipital regions and contrast enhancement CT shows enhancement in the left dorso-lateral and right occipital regions.



Fig. 2 CT scan 8 weeks later demonstrated in the left dorso-lateral and parieto-occipital regions consistent with borderzone infarction. In the right hemisphere infarction relating to the calcarine and parieto-occipital branches of the posterior cerebral artery, precentral branches of the middle cerebral artery and paracentral and internal frontal branches of the anterior cerebral artery.

revealed two discrete hypodense areas in the left dorso-lateral and left parieto-occipital regions consistent with border zone infarction. In the right hemisphere, the distribution of the infarctions were consistent with occlusion of: (1) the calcarine and parieto-occipital branches of the posterior cerebral artery; (2) the precentral and central sulcal branches of the middle internal frontal branches of the anterior cerebral artery (Fig. 2).

The prolonged hypotension, increased haematocrit and heart disease with decreased cardiac output in our patient have a haemodynamic significance.⁴ Bilateral watershed infarctions are due to a haemodynamic mechanism. In 75% of the patients described by Bogousslavsky and Regli,⁴ they were associated with occlusive or stenotic carotid artery disease. Multiple cerebral lesions have been reported as a sign of 'embolic encephalopathy'.^{5,6} In two studies,^{5,6} there was associated carotid artery disease as well. Although multiple branch occlusions are nearly always embolic in nature, non-embolic multiple cerebral artery branch occlusions could possibly occur.⁵ There was no evidence of carotid artery disease in our patient.

Our patient had an increased haematocrit which not only reduces cerebral blood flow⁷ but also impairs oxygen uptake by the tissues, impairs microcirculation and predisposes to thrombosis⁸ and increases the risk of cerebral infarction.⁹ It would therefore be reasonable to surmise that in our patient haemodynamic and thrombotic mechanisms could be implicated. This is shown in Figure 3. When seen at 8 weeks, she had largely recovered and was ambulating independently. To attain a certain level of functional improvement, it usually takes days or months for patients with ischaemic or haemorrahgic cerebral lesions. It is not unknown for some patients to make full recovery within 24 hours of the insult.¹⁰ It has been shown by Shibasaki et al.¹¹ that there is bilateral activation following cerebral damage in some patients. This could be brought about by either changes in the cerebral blood flow¹⁰ or by the phenomenon of diaschisis.^{12,13} According to Silvestrini et al.,¹⁰ the bilateral activation could arise a few months after the stroke event. The variation in the recovery in the different patients can be explained by the degree of activation in the cerebral areas ipsilateral and contralateral to the lesion.

The patient's linguistic performance came close to anomic aphasia but repetition was slightly poorer than would be expected for this type of aphasia. The major feature being the prominence of word-finding difficulty with relative intactness of auditory comprehension with normal grammar and few



cerebral infarcts.

Our patient also had bilateral motor abnormalities. She had bimanual in-coordination of both hands. Her left occipitoparietal lesion was well suited to induce optic ataxia of both

paraphasias. The underlying brain damage is believed to be in

the most posterior part of the superior temporal gyrus and the

adjacent inferior lobule. Watershed infarctions of the dominant

hemisphere have been associated with a number of language



hands, in the visual fields contalateral to the lesion by interrupting both ipsilateral and contralateral connections linking the visual cortex of the damaged hemisphere with the motor areas of both hemispheres.^{16–18}

In conclusion, we have described a woman with septic shock who had bilateral motor abnormalities, a language dysfunction of anomic aphasic type, a non-aphasic communication disorder with near normal recovery in eight weeks. The events are due to bilateral watershed infarction and a widespread cerebral dysfunction brought about by haemodynamic changes, caused in turn by prolonged hypotension. Her condition was compromised by risk factors, namely her severe chronic airways disease and cardiac failure with proclivity towards thrombus formation.

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Wernicke's encephalopathy presenting with upbeating nystagmus

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Summary The case of a 49-year-old woman with Wernicke's encephalopathy is described, in which primary position upbeating nystagmus was the chief ocular sign. Although there was no history of excessive alcohol consumption, Wernicke's encephalopathy was diagnosed on a background of anorexia nervosa. The diagnosis was supported by the patient's symptomatic and clinical recovery following thiamine therapy. © Elsevier Science Ltd. All rights reserved.

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INTRODUCTION

Wernicke's encephalopathy is a thiamine deficiency syndrome characterised by the acute onset of ocular motor signs, ataxia and a confusional state.^{1–3} The case presented demonstrates upbeating nystagmus, an uncommon ocular presentation of Wernicke's encephalopathy. Only two cases of upbeating nystagmus in Wernicke's encephalopathy have previously been reported and neither of these occurred secondary to anorexia nervosa.^{4,5} Interestingly a recent case of Wernicke's encephalopathy occurred with hyperemesis gravidarum.⁶

Early recognition of the neuro-ophthalmologic signs of Wernicke's encephalopathy is important, as the disease is readily reversible by the early administration of thiamine.^{1–3,7–9}

CASE REPORT

A 49-year-old woman presented with a ten week history of selfinduced starvation and generalised abdominal discomfort, on a background history of a major depressive illness. On examination she was severely emaciated and weighed 34 kg, a substantial reduction from her usual weight of 59 kg. She was fully orientated, but was noted to be apathetic and had a depressed affect. Neurological examination demonstrated gait ataxia and generalised weakness, without focal neurological signs.

Hypokalaemia with a K^+ of 2.6 mmol/l (normal: 3.5–5.5 mmol/l) and hyponatremia with a Na⁺ of 128 mmol/l (normal: 135–145 mmol/l) were noted on investigation.