Craniectomy for middle cerebral artery infarction

Introduction

Following cerebellar infarction, concerns regarding hydrocephalus and space occupation from oedema and haemorrhage prompt urgent referral to the neurosurgeons. However, there appears to be less awareness of the potential for clinically significant space occupation after cortical stroke. This short case series illustrates the need to consider 'malignant' stroke, discusses the evidence for decompressive surgery in this group and presents the experience of patients transferred to the Birmingham neurosurgical unit from local hospitals over a 1-year period.

Discussion

Massive unilateral hemispheric infarction of the middle cerebral artery accounts for approximately 10–15% of supratentorial infarction (Moulin et al, 1985). Acute occlusion of the major intracerebral arteries produces brain ischaemia and cerebral oedema with an attendant rise in intracranial pressure producing an intracranial compartment syndrome ('malignant' stroke). Patients presenting with malignant infarction are usually first treated with medical therapies (e.g. head elevation, mannitol, sedation) aimed at reducing brain oedema. Although these treatments may be initially successful, maximal medical therapy often fails to prevent herniation, as evidenced by the high associated mortality rate (approaching 80%) in the absence of surgery (Hacke et al, 1996). More so, such efforts may inevitably delay surgical intervention (Rengachary et al, 1981).

In a non-randomized, single centre prospective trial (Schwab et al, 1998), examining early (<24 hours) *vs* late (>24 hours) hemicraniectomy for patients with complete middle cerebral artery infarction, mortality was 16% in the group treated early *vs* 34.4% in the group treated late. The majority (75%) of the late group demonstrated signs of uncal herniation compared to 13% of the patients treated early. Patients treated very early after onset of symptoms (within 6 hours) were found to be significantly less disabled at 6 months (Cho et al, 2003). The cases from the authors' unit showed a similar trend.

European stroke recommendations have recognized that 'surgical decompression in large hemispheric infarction can be a life-saving measure and survivors may have a residual neurological deficit that allows an independent life' (Hack et al, 2003). Results from randomized controlled trials (Hemicraniectomy and Durotomy for Deterioration From Infarction Related Swelling Trial (HeAADFIRST), www.strokecenter.org/trials) are currently awaited and will inform future management.

A national service framework has been introduced for stroke. This has resulted in a greater focus on the management of this condition. Hospitals with stroke units are associated with a better prognosis, which may in part be a result of earlier recognition of stroke complications. Young patients with acute middle cerebral artery territory infarction are particularly at risk of malignant cerebral oedema. The authors would suggest that aggressive medical measures and early referral for close observation in a unit with neurosurgical facilities should be considered if such patients deteriorate with symptoms consistent with progressive cerebral oedema. **BJHM**

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Case Reports

Acute occlusion of the major intracerebral arteries produces brain ischaemia and cerebral oedema with an attendant rise in intracranial pressure producing an intracranial compartment syndrome ('malignant' stroke). Over the course of 2003, three patients referred to the authors' unit underwent craniectomy for space-occupying malignant cerebral oedema following acute middle cerebral artery infarction. Each case deteriorated acutely following the initial event. Details regarding each patient's initial clinical state, mode of deterioration and clinical outcomes are briefly described. On each occasion the preoperative computed tomography (CT) scan showed signs of a large middle cerebral artery territory infarction and was consistent with space occupation and mass effect. The postoperative CT scans demonstrated evidence of expansion of the oedematous brain tissue, most pronounced in case 2. These findings are illustrated in *Figures 1a* and *b*.

Case 1

A 45-year-old woman presented with a dense left hemiparesis and a Glasgow coma score (GCS) of 14. She remained stable for 3 days but then deteriorated acutely over 3 hours. She began vomiting, became increasingly drowsy and eventually exhibited only a withdrawal response to painful stimuli. Her eyes were conjugately deviated to the right but her pupils remained responsive to light. She underwent urgent decompressive craniectomy. Six months following the procedure she was able to mobilize with the help of a stick and her Barthel score was 70.

Case 2

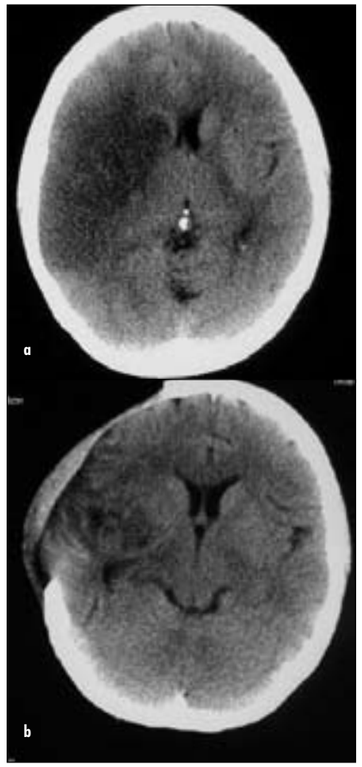
A 23-year-old woman was admitted with a right hemiplegia. The patient remained alert for 2 days after which she became increasingly drowsy. By day 3 she was comatose with non-reacting pupils and required ventilation. The patient was found to have raised intracranial pressure, which proved unresponsive to medical therapy. However, while being ventilated, some purposeful movement of the left hand was noted and a decompressive craniectomy was performed. When reviewed at 6 months, although expressively dysphasic, she was capable of independently caring for herself (Barthel score 60).

Case 3

This 45-year-old male patient had suffered a transient ischaemic attack 11 years earlier and since that time had been placed on aspirin. He presented with a mild spastic left hemiparesis and a GCS of 15 but over the next few hours he became uncooperative and the weakness worsened. Eighteen hours after the initial presentation he was noted to be increasingly drowsy, demonstrating signs of raised intracranial pressure. An urgent craniectomy was therefore performed. At 6 months the patient was walking with assistance and demonstrated no overt cognitive deficits (Barthel score 60).

In each of these three cases there was a significant clinical decline and preoperative CT scan evidence consistent with space occupation. The cause of stroke in case 2 was an arterial dissection, but the aetiology was not clarified in the other cases.

Figure 1. a. Preoperative computed tomography scan of the brain in case 2 demonstrating massive middle cerebral artery infarction. b. Postoperative scan of the same case, showing reduction of the mass effect as a result of cranial decompression.



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The thick of it

I've been wanting to write about BBC4's vicious political satire, *The thick of it*, ever since seeing the first episode. The second series – there have only been three episodes in each series – has just finished, but all six episodes are to be shown on BBC2. Do not miss them.

You could watch *Yes, Minister* and laugh at the way the Civil Service controlled the politicians. It was a feelgood programme. In The thick of it, the spin-doctors are in complete control. Sometimes, screen violence has to be watched from behind the sofa. There is no physical violence in *The thick of it*, but actor Peter Capaldi's Malcolm Tucker, a brutal cross between Alastair Campbell and Peter Mandelson, needs only his face, voice and demeanour to want you to cover your eyes. And its relevance to medicine is Tucker's handling of the hapless Minister for Social Affairs, Hugh Abbot (Chris Langham at his hangdog best), for his seeking of expert advice.

Abbot is saddled with having to close special schools for the educationally subnormal, and support instead the government's option of integrating the children into normal schools. He has seen the special schools, and has first-hand knowledge of how well they work for the children. He has also spoken to an expert, and he tells Tucker so. Tucker carefully explains – he does this in every episode: 'carefully' meaning that he explains as if to a sullen teenager – that you go to an expert only when you've already worked out that they are going to tell you what you want to hear. Reluctantly, Abbot goes to a second expert, who asks Abbot who he's already consulted. Abbot's answer is greeted with a sneer, and a dismissive, 'Well, actually, he's regarded as a bit of joke in educational circles.'

Now, where have I heard that sort of response? Anytime that Allyson Pollock's critique of PFI [the private finance initiative] is raised in discussion with any proponent of government health policy. We're not talking evidence here; we're talking ideology. And I prefer Pollock's ideology to Blair's. Tony Blair has made it known that he is disturbed by some primary care trusts (PCTs) refusing to pay for herceptin for women with breast cancer.

As I write, a breast cancer patient is threatening to take her PCT to the High Court to force treatment that the PCT think is not cost-effective, nor yet proved to be safe. Health Secretary Patricia Hewitt is concerned at the PCT's decision.

It's all very emotive, but someone needs to ask Hewitt, or Blair, which patients will have their treatments curtailed or cancelled so herceptin can be purchased. Shroud wavers can blather on all they like about how it's not right for a life to be costed (let's leave aside that there is no guarantee the herceptin will be effective), but that's what PCTs have to do: they have money, and they have to choose which services to buy. A *BMJ* letter costed herceptin at £460 000 per patient cured. That is a lot of hip replacements or a lot of supportive treatment for women with incurable breast cancer. As the letter writer said, there are no easy answers. But for the politicians there is an easy way out, and Hewitt looks likely to take it. **BJHM**

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