Management of traumatic intracranial haematoma

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Abstract

Deciding which head-injured patients should be transferred to a neurosurgical unit can be difficult. Traditional criteria emphasise the development of deteriorating responsiveness but lead to delayed diagnosis and to avoidable mortality and morbidity. To discover if a more liberal admission policy improved results a study was conducted analysing data collected prospectively from 683 patients who had a traumatic intracranial haematoma evacuated in the Glasgow neurosurgical unit between 1974 and 1980. In the first four years, before the change in policy, mortality was 38% but decreased to 29% afterwards. This reflected a reduction in the proportion of patients who talked after injury but who deteriorated into coma before operation—that is, 31% before the change in policy, 16% afterwards.

If the potential benefits of CT scanning in the management of head injuries are to be realised patients must be scanned sooner than in the past. This will usually mean that more patients should go to a neurosurgical unit and that simple criteria for transfer should be established.

Introduction

Reports from centres in which most head-injured patients are admitted directly to neurosurgical care show clearly that the earlier a patient with a traumatic haematoma undergoes evacuation of clot the better is the patient's outcome.^{1,2} Yet head-injured patients throughout most of Britain are admitted initially to a primary surgical ward, and only 5% are subsequently transferred to a neurosurgical unit.³ Unfortunately, according to several studies the criteria widely used to select patients for transfer lead to avoidable mortality and morbidity in those with a traumatic intracranial haematoma.⁴⁻⁷ The reason for this is that it has been customary to suspect that a patient has a significant traumatic intracranial haematoma only after responsiveness deteriorates or if he fails to improve after several days; this leads to delayed diagnosis, so that by the time the clot is evacuated many such patients have already sustained irreversible secondary brain damage.⁴

There are several reasons for restricting severely the number of head-injured patients admitted to neurosurgical units. One is the availability of neurosurgical beds in relation to the total number of head-injured patients. In the past another reason was that the appropriate investigation—that is, angiography—was difficult, invasive, and potentially dangerous and therefore justified only in those patients most obviously suffering from an intracranial complication. The introduction of CT scanning has made the diagnosis of an intracranial haematoma simpler and safer and has shown that a haematoma may be present for some time before clinical signs develop. Scanning can therefore be

justified at a stage that previously would not have been considered to merit angiography. Unfortunately, reports^{8,9} show that the introduction of CT scanning by itself does not lead to improved results so long as patients are still selected by the traditional criteria.

In an effort to use the potential of CT scanning to achieve early diagnosis and thus reduce delays, we proposed that neurosurgical units should be more ready to accept head-injured patients and should investigate those who were considered to be at risk of having a haematoma, even though they had not (at that time) deteriorated.¹⁰ Such patients include those in coma or with neurological signs or persisting confusion, particularly when any of these features is associated with a skull fracture.¹¹ The Glasgow neurosurgical unit began to apply this policy at the end of 1977. To discover how this influenced the management and outcome of patients with a traumatic haematoma we have compared the results in two periods: before and after the change in policy. CT scanning was available in Glasgow throughout both periods.

Patients and methods

We prospectively collected data about patients who had a traumatic intracranial haematoma evacuated in the Glasgow neurosurgical unit between January 1974 and December 1980. Only haematomas requiring craniotomy within two weeks of injury were included. Patients with contusions or small haematomas treated conservatively were excluded, as were those with a chronic subdural collection of fluid. Haematomas were classified as extradural or intradural; patients with both were regarded as having an intradural clot.¹¹ Clinical responsiveness was assessed by the Glasgow Coma Scale, and coma was defined as no eye opening with no comprehensible verbal response and not obeying commands.¹² Pupil reactivity was also recorded. A patient's best level of responsiveness before the onset of any subsequent deterioration was taken as an index of the extent of recovery from any brain damage sustained at the time of injury. The extent of deterioration was indicated by comparing this with the patient's state immediately before operation. Outcome was assessed six months after injury according to the Glasgow Outcome Scale.¹³

Results

Numbers of patients—During the four years 1974 to 1977 the numbers of operations for traumatic intracranial haematoma were 86, 77, 64, and 78, respectively (305 in all); in the next three years the numbers were 115, 159, and 104 (378 patients in all). The yearly rate increased from an average of 76 cases in the first period to 126 after 1978 ($p < 0.001$), but the types of haematoma evacuated and the patients' ages were similar in the two periods (table I).

Responsiveness before deterioration (table I)—Similar proportions of the two series of patients recovered sufficiently from the initial effects of the injury to be able to talk at some stage before operation. Conversely, similar proportions had persisting brain damage from the time of injury and did not talk afterwards. The proportion of patients whose pupils were unreacting from when they were first seen was also similar in the two periods (8% in 1974-7; 11% in 1978-80).

Responsiveness before operation (table II)—The proportion of patients in coma immediately before operation was significantly lower in 1978-80 than in 1974-7 ($\chi^2 = 16.8$; 1 df; $p < 0.001$). This was because fewer patients who had talked after injury subsequently deteriorated into coma (33% of patients in 1978-80 compared with 52% before). Similarly, fewer patients developed non-reacting pupils in the second period (14% in 1978-80 and 30% before).

Outcome (table III)—An unfavourable outcome (death, vegetative stage, or survival with severe disability) was less frequent after 1977

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(40%) than before (51%). Mortality was reduced with both types of haematoma: in patients with an extradural haematoma mortality fell from 25% to 19%, and in those with an intradural lesion the fall was from 47% to 35%. Of the 181 patients who had talked after injury during 1974-7, 47 (26%) deteriorated into coma and died; these accounted for 40% of all deaths in that period. In 1978-80 a total of 211 patients had talked, of whom only 25 (12%) deteriorated into coma and died; these patients accounted for 23% of deaths in the second period. In both periods patients in coma before operation had a higher mortality (1974-7, 53%; 1978-80, 46%) than those with a better level of responsiveness (1974-7, 10%; 1978-80, 12%).

TABLE I—Details of patients studied

	No (%) of patients	
	1974-7 (n = 305)	1978-80 (n = 378)
Age (years) {		
< 39	132 (43)	145 (38)
≥ 40	173 (57)	233 (62)
Extradural clot	119 (39)	127 (34)
Intradural lesion only	186 (61)	251 (66)
Talked	181 (59)	211 (56)
Never talked*	123 (41)	167 (44)

* In one case in 1974-7 it was not known if the patient had talked.

TABLE II—Responsiveness before operation

	No (%) of patients*	
	1974-7 (n = 304)	1978-80 (n = 377)
Not in coma	104 (34)	188 (50)
In coma {		
Had talked	94 (31)	62 (16)
Never talked	106 (35)	127 (34)

* Information missing on one patient in each period. Reduction in proportion of patients in coma was significant ($\chi^2 = 16.8$; 1 df; $p < 0.001$).

TABLE III—Outcome six months after evacuation of traumatic intracranial haematoma

	No (%) of patients	
	1974-7 (n = 305)	1978-80 (n = 378)
Dead	116 { (38)	107 { (29)
Vegetative	1 {	4 {
Severe disability	40 (13)	43 (11)
Moderate disability	39 {	55 {
Good recovery	101 { (49)	138 { (60)
Unknown but out of hospital	8 {	31 {

Reduction in proportion of patients with poor outcome (dead, vegetative, or severely disabled) was significant ($\chi^2 = 8.2$; 1 df; $p < 0.005$).

Discussion

CT scanning is a highly reliable means of detecting a traumatic intracranial haematoma. It provides an exact location so that surgery may be planned precisely, and it has reduced the need for emergency burr holes.⁸ But diagnosis must be earlier as well as more anatomically accurate in order to ensure that as many clots as possible are evacuated before irreversible secondary brain damage develops. Baker emphasised that most scanners in Britain are available easily only to neurosurgeons, whereas most head-injured patients are in primary surgical wards and accident departments.¹⁴ Hence if intracranial clots are to be diagnosed sooner there must be a change in the criteria used to select patients for transfer and investigation.

The introduction of a more liberal transfer policy in the west of Scotland increased the total number of patients admitted to a neurosurgical unit from an average of 223 a year at the beginning of the study³ to 492 a year in 1979-80. The mortality of patients with an intracranial haematoma fell from 38% to 29%.

The increase in the number of patients with a clot that needed to be evacuated was unexpected, and this alone might have influenced the results.

In practice, the additional patients in the second period included both severely and mildly injured people so that overall the two series were closely similar with respect to important prognostic factors such as age, the type of haematoma, and the proportion of patients with severe primary brain damage. The presence of brain damage that is severe from the start is reflected in the patient being in coma from the time of injury, and it is these patients who now account for the major proportion of mortality and morbidity in patients transferred to the neurosurgical unit. In the first period many such patients probably remained in the primary surgical ward and either died or survived with disability. Indeed, in Merseyside, where only 1.2% of patients admitted for head injuries are transferred to the regional surgical unit, deaths from an intracranial haematoma occur as often in general hospitals as in the regional unit. Studies are in progress to establish if the change in admission policy in Glasgow has reduced deaths in the general hospitals in our region.

Although more mildly injured patients were admitted during the second period, the improved outcome cannot be explained by unnecessary operations on patients with a small haematoma who would have recovered without surgery. CT scanning was available in both periods and neurosurgeons in Glasgow have made a point of adopting an expectant policy in the management of these kinds of haematomas.¹⁵ During the second period of the study more than 100 such haematomas were managed without operation. The most likely reason for the improved results in the second series was that a smaller proportion of patients had deteriorated into coma before operation; this indicates that fewer patients had developed irreversible secondary brain damage.

Several different methods are advocated that aim to minimise delay in the detection of a traumatic intracranial haematoma. In North America and in a few British centres the view is that as many head-injured patients as possible, including all those with a mild injury, should be managed by neurosurgeons. Another view is that scanners should be available in general hospitals so that haematomas could be detected there¹⁶; the patient might then be operated on in the general hospital or sent to a regional neurosurgical centre. Both approaches are expensive in resources; moreover, without an alteration in the clinical criteria for investigation neither method would guarantee reduced delay and improved results. Investigation outside a neurosurgical unit might even increase delay. The best method would be to establish simple clinical criteria for identifying before deterioration which head-injured patients have a high risk of haematoma so that these could be transferred for scanning and possible operation.

Even after the change in policy only 7% of head-injured patients admitted to hospital in the west of Scotland were transferred to the neurosurgical unit. The increase in admissions to the neurosurgical unit was feasible because patients were returned rapidly to the referring unit after a significant intracranial lesion had been excluded and also because a few additional beds became available. Avoidable mortality and morbidity are still occurring and their elimination may require the investigation of even more patients—possibly at least 10% of those presently admitted to hospital. Additional neurosurgical facilities for the investigation of head injuries might be provided from the resources that would be released by a reduction in the total number of admissions for head injury. This could be achieved if fewer mildly head-injured patients were admitted for observation than at present. Most of these patients have a negligible risk of haematoma, and studies in Nottingham show that many can safely be discharged.¹⁷

It is essential to break away from the tradition that a traumatic intracranial haematoma is diagnosed only after there has been progressive neurological deterioration. It is also clear that more head-injured patients should go to a neurosurgical unit than occurs in most places in Britain and that they should go there

sooner. We now advocate that a recently head-injured patient should have a CT scan if he has either altered consciousness or other neurological signs or symptoms that do not improve after initial assessment and resuscitation. When there is a skull fracture the indications for scanning are increased; even by itself a fracture probably provides an adequate reason.

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Converting-enzyme inhibitor enalapril (MK421) in treatment of hypertension with renal artery stenosis

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Abstract

Enalapril maleate (MK421), a new inhibitor of angiotensin converting enzyme, in single daily doses of 1.25-40 mg was assessed in five patients with hypertension and renal artery stenosis. Only small falls in plasma angiotensin II concentrations were seen at doses less than 10 mg; even with 10 and 20 mg, angiotensin II concentrations had risen again 24 hours from the last dose. During long-term treatment with 10-40 mg daily all patients achieved good blood-pressure control. No significant changes of body sodium or potassium values were seen. The drug was well tolerated with no serious side effects.

These findings are evidence of the efficacy and acceptability of enalapril in the medical management of hypertension with renal artery stenosis.

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Introduction

We have reported¹ the use preoperatively of the converting-enzyme inhibitor captopril in the treatment of hypertension associated with unilateral renal artery stenosis. Long-term captopril promised well as a predictor of the blood-pressure response to operation, although this requires further study. We have also reported that oral captopril in a dose of 150 mg three times daily produces sustained suppression of the plasma angiotensin II concentration throughout 24 hours.² Captopril, however, has been associated with several toxic effects which may be attributable to the sulphhydryl group in its molecule.³ Although the use of lower doses of captopril may avoid at least some of these unwanted effects, it appears important to consider alternative converting-enzyme inhibitors. Enalapril maleate (MK421),⁴ an orally active converting-enzyme inhibitor devoid of a sulphhydryl group, may permit long-term inhibition of angiotensin II formation without incurring the side effects seen with captopril. We report preliminary results of the use of long-term enalapril in five patients with hypertension and renal artery stenosis. Particular attention was paid to the magnitude and duration of the reduction in plasma angiotensin II concentrations.

Patients and methods

Five patients (two women) aged 38-56 years gave informed consent to the study, which was approved by the hospital's ethical supervisory committee. All patients had unilateral renal artery stenosis shown by intravenous pyelography, renal arteriography, isotope nephrography,⁵ bilateral renal vein renin measurements, and ureteric catheter studies.^{1,6} Four had radiological evidence of atheroma and one fibromuscular hyperplasia. All had normal serum electrolyte values and renal