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Original articles

Intracerebral haemorrhage after the neonatal period

J H LIVINGSTON AND J K BROWN

Department of Paediatric Neurology, Royal Hospital for Sick Children, Edinburgh

SUMMARY Intracerebral haemorrhage is rare in childhood. We have reviewed the last 10 years' experience, in our referral area, of parenchymatous intracerebral haemorrhage in children from 1 month to 16 years of age. There were 27 cases, five of which were intracerebellar and two predominantly intraventricular. The commonest aetiology was vascular malformation (10), followed by haemorrhage into tumour (four), and coagulopathies (five).

Clinical features were non-specific, but altered consciousness, headache, vomiting, and focal signs were the most common. Focal signs were, however, rare in the patients with intracerebellar haemorrhage. There was an overall mortality of 54% (14 out of 27). Nine patients were handicapped on follow up, but none severely so. For the diagnosis of intracerebral haemorrhage a high level of clinical suspicion is needed with early use of computed tomography. Maintenance of homeostasis, relief of raised intracranial pressure, and evacuation of haematoma are the aims of management.

Intracerebral haemorrhage in childhood is a rare and often devastating event. There are a large number of published works on this type of haemorrhage in the adult, hypertension with or without atherosclerosis being the major aetiological factor.[1 2]

There have been few studies of intracerebral haemorrhage in childhood and these have been mainly concerned with one aetiological group or have been isolated case reports. In an attempt to obtain an overview of the aetiology, presentation, management, and prognosis of intracerebral haemorrhage in childhood we have reviewed the last 10 years' experience of this type of haemorrhage in Edinburgh.

Patients and methods

Records were reviewed from the Royal Hospital for Sick Children, Edinburgh, and the Regional Neurosurgical Centre. All cases of intracranial haemorrhage in children from 1 month to 16 years of age were sought, including extradural, subdural, subarachnoid, and intracerebral haemorrhages. Cases coded in the records as intracranial aneurysm and arteriovenous malformation were also sought, and in addition the autopsy book kept by the Regional Neuropathology Service was examined to detect those cases who presented and died at other hospitals in the region who were referred for autopsy. The 10 year period from June 1974 to May 1984 was considered.

Results

Over the 10 year period there were a total of 114 cases of intracranial haemorrhage. Of these, 50 were subdural, 34 were extradural, and three were pure subarachnoid.

The remaining 27 cases were parenchymatous intracerebral haemorrhages. There were 20 cases of supratentorial intracerebral haemorrhage, five of intracerebellar haemorrhage, and two of predominantly intraventricular haemorrhage. All subsequent discussion refers to this group of parenchymatous haemorrhages, and the non-parenchymatous haemorrhages will not be discussed further.

Table 1 *Age and sex distribution of the 27 cases of parenchymatous intracerebral haemorrhage*

	No	*%*
M:F	16:11	
Age (years):		
0–2	3	11
2–10	9	33
10–16	15	56
Total	27	100

Age and sex. The age and sex distribution is shown in Table 1. There were three children under the age of 2 years. The aetiology in these cases was; late haemorrhagic disease of the newborn, haemophilia and associated trauma, and ruptured middle cerebral artery aneurysm. There were nine cases between 2 and 10 years and 15 cases between 10 and 16 years. All the cases of intracerebellar haemorrhage occurred in the oldest age group. There was a male preponderence of 16 to 11, a male/female ratio of 1·45/1.

Aetiology. The aetiological groups are shown in Table 2.

Vascular malformation
The largest group were those with haemorrhage due to vascular malformation, which comprised 10 out of 27 (37%) of the total. One of these had Marfan's syndrome, which is a recognised association. The sites of the vascular malformations were as follows: middle cerebral (two), posterior cerebral (two), anterior cerebral (two), around the splenium draining into the vein of Galen (one), microvascular anomaly in the parietal region (one), posterior fossa (one), temporal region of uncertain origin (one), and frontal lobe of uncertain origin (one).

Coagulopathies
As a group, coagulopathies comprised four of the 27 cases (15%). Two of these were thrombocytopenic and one had haemophilia. The other child was a 9 week old baby who had a massive right sided intracerebral haemorrhage associated with prolonged prothrombin and partial thromboplastin time. This was thought to be due to late haemorrhagic disease of the newborn. Autopsy revealed pancreatic changes suggestive of cystic fibrosis, but the relation of this to the haemorrhages is unclear.

Table 2 *Aetiology of the 27 cases of parenchymatous intracerebral haemorrhages*

	No	%
Vascular malformation	10*	37
Aneurysm	3	11
Acute lymphoblastic leukaemia	1	4
Aplastic anaemia	1	4
Haemophilia	1	4
Other coagulopathy	1	4
Meningioma	1	4
Lymphoma	1	4
Astrocytoma	1	4
Cerebellar glioma	1	4
Pontine glioma	1	4
Hypertension	1	4
Unknown	1	4
	27	100

*One patient with Marfan's syndrome.

Aneurysm
Haemorrhage was due to ruptured aneurysm in three cases, two from the middle cerebral and one from the posterior inferior cerebellar arteries.

Haemorrhage into neoplasm
There were five cases of haemorrhage into tumour; one each of meningioma, pontine glioma, lymphoma, cerebellar glioma, and cerebellar astrocytoma. In four of these cases haemorrhage was the initial presenting feature of the tumour.

Others
There were only four cases in whom no cause could be found. One of these was a 4 year old boy who presented with multifocal seizures and drowsiness. His computed tomogram showed small bilateral frontal haematomas. On reviewing the history it became apparent that he had been taking large amounts of aspirin for three days before presentation. A platelet function defect was suspected but never subsequently proven. There was also one case of intracerebral haemorrhage in the region of the right basal ganglia due to hypertension in a teenager with advanced glomerulo nephritis.

Clinical features. The symptoms and signs on presentation are shown in Table 3. Onset of symptoms was sudden in 16 (59%) cases and gradual in 11 (41%). Onset was defined as sudden when symptoms developed over a period of one hour or less. The commonest symptoms were headaches and vomiting, occurring in 19 or 20 (70–75%) cases. Altered consciousness occurred in 25 (92%) cases. Thirteen of these were merely drowsy on presenta-

Table 3 *Symptoms and signs at presentation*

	No	%
Onset:		
Sudden	16	59
Gradual	11	41
Headache	19	70
Vomiting	20	74
Altered consciousness:		
Drowsiness	13	
Stupor	5	
Coma	7	
Focal signs:*	17	63
Hemiparesis	13	48
Focal seizure	5	19
Dysphasia	3	11
Pupillary changes	2	7
Hemisensory	2	7
Other cranial nerve	3	11
Seizures	9	33
Visual symptoms	2	7
Apnoea	2	7
History of trauma	1	4

*Occurred in only one patient after intracerebellar haemorrhage.

tion, five were stuporose, and seven were comatose. Focal signs were common and occurred in 17 (63%) cases. Hemiplegia occurred in 13 (48%), focal seizures in five (19%), dysphasia in three (11%), cranial nerve signs in three (11%), and pupillary changes in two (7%), and two of the older children complained of hemisensory symptoms. Seizures occured in nine (33%) cases and, with the exception of one child who presented in grand mal status, these were always focal. Two children had visual symptoms on presentation, one had visual failure, and one an homonymous hemianopia. Only one child gave an unequivocal history of trauma, this being an 18 month old boy with haemophilia who walked against a cupboard and subsequently died following a massive parieto-occipital haemorrhage.

It is of note that focal signs occurred in only one case of intracerebellar haemorrhage, and this consisted of tonic deviation of the eyes to one side. None of those with intracerebellar haemorrhage had a hemiplegia on presentation.

Investigations. Excluding the patients with coagulopathies, the results of initial investigations on arrival in hospital were not particularly helpful. Neutrophil leucocytosis was present in nine cases, hyperglycaemia in eight, and fever $>38°C$ in seven. Only one patient had a low haemoglobin concentration on presentation, and no patients were thrombocytopenic.

Method of diagnosis. Computed tomography was used for diagnosis in 18 (67%) cases. In two cases diagnosis was made by angiography, both of these presenting in the era before computed tomography. Diagnosis was made at autopsy in five (19%) cases and by head ultrasound in two (7%).

Lumbar puncture was performed in 13 (48%) cases, in all but one of whom the cerebrospinal fluid was blood stained or xanthrochromic. It is of note that four of the five patients with intracerebellar haemorrhage had a lumbar puncture performed and that all these patients subsequently died. Computed tomography was performed in only three of the patients with intracerebellar haemorrhage, the others having died before scanning was possible.

Site of bleed and angiographic findings. There were five posterior fossa and two predominantly intraventricular haemorrhages. The sites of the remaining haemorrhages are shown in Table 4. There were four midline haemorrhages, two midbrain, one pons, and one callosal. Of the remaining 16, 10 were right sided and six left sided. There was only one deep or so called ganglionic haemorrhage, the rest being lobar haemorrhages. There was one case of bilateral frontal haemorrhage and one of multiple haemorrhages in a child with leukaemia.

Table 4 *Site of haemorrhages in the 27 cases with parenchymatous intracerebral haemorrhage*

	No	*%*
Midline:	4	15
Midbrain	2	
Pons	1	
Callosal	1	
Right hemisphere	10	37
Left hemisphere	6	22
Frontal	2	7
Bilateral frontal	1	4
Frontoparietal	2	7
Parietal	3	11
Temporal	4	15
Basal ganglia	1	4
Parieto-occipital	1	4
Multiple	1	4
Massive right sided	1	4
Posterior fossa	5	19
Intraventricular	2	7

Angiograms were carried out in 14 patients. In four cases the angiograms yielded normal results. There was only one false negative; a small vascular malformation was found at surgery in a child whose angiograms had shown normal results. Vascular malformations were found in eight cases and aneurysms in two. The site of these lesions has been discussed above.

Management. Surgery was carried out in 19 (70%) cases. This usually comprised evacuation of haematoma and resection of a malformation if present. In six of the patients surgery comprised emergency insertion of intraventricular drains in an attempt to relieve massively raised intracranial pressure. All of these patients subsequently died. Three of these six patients had posterior fossa haemorrhages, two of them intraventricular haemorrhage, and one a midline intracerebral haemorrhage. Of the patients who had posterior fossa haemorrhages, only one survived long enough for exploration of the posterior fossa to be attempted.

Outcome. There was an overall mortality of 52% (14 out of 27) (Table 5). The mortality after either intracerebellar haemorrhage or pure intraventricular haemorrhage was, however, 100% (seven cases).

The aetiologies of haemorrhage in the remaining seven fatal cases were pontine glioma, angioblastic meningioma, acute lymphoblastic leukaemia, aplastic anaemia, haemophilia, haemorrhagic disease of the newborn and hypertension. Only two of these patients had had surgery. One died shortly after insertion of intraventricular drains with massively raised intracranial pressure after haemorrhage into

Table 5 *Outcome of the 27 cases with parenchymatous intracerebral haemorrhage*

	No	*%*	*Surgical management*	*Conservative management*
Normal	3	11	2	1
Dead	14	52	7	7
Handicap:	9	33	9	—
Hemiparesis	5	19		
Seizures	4	15		
Dysphasia	4	15		
Visual field	4	15		
Clumsiness	1	4		
Memory difficulties	1	4		
Unknown	1	4	1	

Mortality after intracerebellar or intraventricular haemorrhage=100%.

an angioblastic meningioma. The other patient had haemophilia and underwent successful evacuation of a parietal-occipital haematoma. He returned three weeks later with a massive recurrence and died shortly after admission. Four out of five of the patients who did not have surgical management in this group were at the end stages of malignant or progressive diseases and it is uncertain whether surgical intervention would have altered the outcome.

Nine patients were handicapped on follow up and three were normal. Five (19%) have hemiparesis, but in all cases it was mild, all having independent locomotion. Seizures have occurred in four (15%), but in none of these have they been a major problem. Visual field defects and dysphasia have each occurred in four (15%). One patient has memory difficulties and another has slight clumsiness. There are no severely handicapped survivors.

Discussion

There are many causes of intracerebral haemorrhage in childhood. The fairly high proportion of vascular malformations in our study is in keeping with most other reports.[3 4]

In childhood, haemorrhage is the commonest presenting feature of an arteriovenous malfunction. This may be a subarachnoid or intracerebral haemorrhage. These malformations extend deep into the brain parenchyma,[6] and it is not surprising, therefore, that if haemorrhage occurs it is more often intracerebral than purely subarachnoid. Vascular malformations are found in all parts of the central nervous system, including the posterior fossa and spinal cord.[7 8] Margolis *et al* first drew attention to the problem of microvascular malformations as a cause of massive intracerebral haemorrhage, and since that time many reports have confirmed that what had previously been called a spontaneous intracerebral haemorrhage was often a haemorrhage from a microangioma.[9–11] These microangiomas may obliterate themselves after haemorrhage, making subsequent angiographic or histological identification impossible.[12] In our series there were four haemorrhages for which no cause was found. One patient had bilateral haemorrhages and it seems unlikely that these were due to microangiomata. The other three, two of which were cases of intracerebellar haemorrhage, could have been due to microangiomata. In only one case was a microangioma shown, this being in a 7 year old girl with a large left parietal haemorrhage after chickenpox. No malformation was seen at surgery, and histology showed encephalitic tissue. However, angiography subsequently revealed a microangioma in the left parietal region. This raises the interesting possibility that the angioma may have developed secondary to the viral infection.

Aneurysms accounted for only a small proportion (11%) of this group. This is in keeping with the fact that aneurysms rarely bleed in childhood, although they can do at any age from neonate onwards.[4 13 14] The comprehensive study by Sedzimir *et al* revealed a fairly high incidence of aneurysms, 40% of 124 subarachnoid haemorrhages in the 0–20 age range.[15] This was a study of subarachnoid haemorrhage and it is not stated how many of the children also had intracerebral haemorrhage, so it is not directly comparable with this series.

Why aneurysms bleed in childhood is uncertain. It is not associated with atherosclerosis, trauma, exercise, or stress.[4 13] In one study 12% of ruptured aneurysms were associated with coarctation of the aorta and 3·5% with polycystic kidneys. In our series we had one ruptured aneurysm associated with renal artery stenosis and hypertension presenting as a pure subarachnoid haemorrhage.

Histologically, most aneurysms are the congenital type and arise most commonly from the terminal carotid, anterior communicating, and middle cerebral arteries, but they may also arise from the basilar system.

Coagulopathies comprised a major aetiological group in this series (15% of the total). There are many different causes of coagulopathy associated with intracerebral haemorrhage. Intracerebral haemorrhage complicating idiopathic thrombocytopenic purpura occurs in up to 1% of cases.[16] Intracerebral haemorrhage complicating acute lymphoblastic leukaemia occurs in 1% of cases in the acute stage and can be due to thrombocytopenia, disseminated intravascular coagulation, haemorrhage into infiltrating tumour, or treatment with drugs—for example, with L-asparaginase.[17–18] There was one case of haemophilia in our series and as

with other reported cases haemorrhage followed minor trauma.[19]

Apart from the child with haemophilia none of the haemorrhages followed trauma. In a previous review by Ingraham and Matson of 1330 cases of head injury in children they found no intracerebral, 30 extradural, and 319 subdural haemorrhages.[5]

Intracerebellar haemorrhage accounts for a 10th of spontaneous intracerebral haemorrhage in adults and is usually due to hypertension.[20] In this series there were five cases (19%) of intracerebellar haemorrhage. Two were due to haemorrhage into tumour and one to a vascular malformation, and two were of unknown cause. All of these occurred in teenage children. Again microangiomas have been emphasised as a cause of intracerebellar haemorrhage.[21]

The two cases of intraventricular haemorrhage in this series were due to vascular malformation in one and aneurysm in the other. Intraventricular haemorrhage had a uniformally fatal outcome. Both patients were over 7 years of age, and this outcome is in keeping with the high mortality of intraventricular haemorrhage in adults.[22 23] Intraventricular haemorrhage often occurs from ventricular extension of a ganglionic/thalamic haemorrhage. It is thought that it is the proximity to vital centres and the greater density of nerve fibres in the region of the initial haemorrhage with an additional aggravating effect of the intraventricular haemorrhage that accounts for the poor outcome.[24]

Cardinal presenting features of intracerebral haemorrhage (Table 3) were altered consciousness, headache, vomiting, and focal signs. The conscious level ranged from coma to mild drowsiness. None of these features are specific for haemorrhage and all are similar to those occurring in other acute encephalopathies of childhood, such as encephalitis, status epilepticus, and toxic encephalopathy. Likewise, the speed of onset of symptoms was not a reliable diagnostic pointer, onset being sudden in 59% of cases and gradual in 41%. Sudden onset of symptoms is much less likely, however, in the other acute encephalopathies of childhood. The group with intracerebellar haemorrhage is different in that none of the patients had hemiplegia on presentation and only one had focal signs, comprising tonic deviation of the eye to one side. Four out of five patients with intracerebellar haemorrhage had a sudden onset of symptoms.

The presence of neutrophil leukocytosis, hyperglycaemia, or fever was of little help in the diagnosis and as with the clinical features is just as likely to be due to other causes of acute encephalopathy. The method of diagnosis in all recent cases was computed tomography. Before its availability diagnosis was implied by angiography or shown at surgery or autopsy. Computed tomography has a diagnostic accuracy for intracerebral haemorrhage of 94% with virtually no false positives.[25] Taking this fact with the fairly non-specific presenting features, it will be seen that for early diagnosis to be made a high level of suspicion is needed in the clinical situation outlined above with recourse to early computed tomography. In this situation lumbar puncture should be deferred until after the scan is available. In the presence of acutely raised intracranial pressure lumbar puncture is clearly hazardous and all it can show is subarachnoid blood, which tells one nothing about whether intracerebral haematoma is present or not. This point needs to be emphasised to junior doctors receiving emergencies as the diagnosis of meningitis can, in *most* cases, wait a few hours until an emergency scan has been obtained. Our figures add weight to this argument, particularly in relation to posterior fossa haemorrhage. Four out of five of the cases of intracerebellar haemorrhage had lumbar punctures and all of these patients subsequently died with massively raised intracranial pressure and brain stem compression.

There is little uniformity in the management of intracerebral haemorrhage,[26] and there have been no studies specifically addressing this issue in childhood. Adult studies, which are made up predominantly of older patients with hypertension and atherosclerosis, have found that in general early surgical evacuation of haematoma and resection of a vascular malformation (if present) is superior to late surgical or conservative management.[2 15 26] In these studies the best results of early intervention have been in young, non-hypertensive patients.[2] There are theoretical grounds for early clot evacuation in that after formation of haematoma secondary cerebral insult occurs due to perivascular haemorrhage, oedema, and perifocal cortical necrosis.[27] The main indication for early clot evacuation, however, is to relieve the raised intracranial pressure. Until surgery can be arranged, before starting monitoring of intracranial pressure, routine brain orientated intensive care should be begun. Thus the aims of management are control of homeostasis with monitoring of central venous pressure, arterial pressure, and urine output and measurement of electrolytes, glucose, and calcium concentrations. Seizures should be controlled aggressively as these are a major cause of secondary damage. Elective hyperventilation with infusion of mannitol and possibly steroids should be used if there is clinical indication of raised intracranial pressure. Monitoring of intracranial pressure will in general not be possible until neurosurgical intervention is arranged. So, if a delay is likely, full supportive measures, as de-

scribed above, should be begun on clinical grounds alone. Monitoring of cerebral blood flow in this situation using ultrasonographic techniques would clearly provide a rapid non-invasive assessment of cerebral haemodynamics and allow more rational management decisions.

In this series all of the patients with intraventricular or intracerebellar haemorrhage died from massively raised intracranial pressure and brain stem failure. Five out of seven of these patients had intraventricular drains inserted with failure to relieve the raised intracranial pressure.

Only one of the patients with intracerebellar haemorrhage survived long enough for exploration of posterior fossa to be attempted but he died during surgery. In all cases the surgical intervention was within a few hours of presentation. Two of the patients with intracerebellar haemorrhage had to be transferred from another hospital in the region. It is unlikely that earlier surgical intervention would have altered the outcome in any of these cases and it seems that in these cases it is the site of haemorrhage rather than the aetiology that relates to the poor outcome. Only one of the survivors of intracerebral haemorrhage was not surgically managed. This was the child with small bilateral frontal haematomas. The aetiologies in those who died after supratentorial haemorrhage (excluding intraventricular haemorrhage) were tumour (two), coagulopathy (four), and hypertension (one). Only two of these had surgery. Whether the outcome would have been different in the others after surgical intervention is uncertain. Although surgical evacuation of haematoma in tumour and due to coagulopathy has been described,[19 28] prognosis in these groups tends to reflect the prognosis of the underlying disease.

In contrast to this group all patients with vascular malformation or aneurysm underwent surgical evacuation of haematoma, with resection of the underlying abnormality. Only one patient in this group had had a respiratory arrest before surgery, and in general these patients had milder increases in intracranial pressure than the previous group. This is reflected in the outcome in that all of this group survived and morbidity was not severe. Although mild handicap was present in nine out of 13 survivors, all are independent in the activities of daily living. Whether this outcome relates to surgical intervention or not is uncertain. As there is a real chance, however, of re-bleeding from arteriovenous malformation or aneurysm[15] as well as the theoretical secondary damage that can occur after haematoma formation early surgical intervention is preferable.

Prognosis in adult series has been related to the degree of depression of the conscious level on presentation.[1 2 24] In this series, however, no clear relation was shown. Six of those who died were merely drowsy on presentation, and three of those who survived with mild handicap were comatose on presentation. Perhaps it is of importance that none of those who are normal on follow up were comatose on presentation.

For early diagnosis of intracerebral haemorrhage to be made a high level of clinical suspicion is needed. Whether early diagnosis and intervention appreciably alter the ultimate prognosis is not clear from this retrospective study. Survival after haematoma evacuation in sites such as the brain stem and posterior fossa and after haemorrhage into tumour or due to coagulopathy has been described,[2 19-21 28] and further prospective studies addressing the issue of intervention in these situations are needed.

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Correspondence to Dr J H Livingston, The Royal Hospital for Sick Children, Sciennes Road, Edinburgh, Scotland.

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