Emergency cranial computed tomography in the management of acute febrile encephalopathy in children

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Abstract

Objective—Evaluation of the influence of emergency cranial computed tomography on the management of acute febrile encephalopathy in children.

Methods—A retrospective study in children with acute febrile encephalopathy who underwent emergency cranial computed tomography within 12 hours of admission to the paediatric intensive care unit. All scans were evaluated by two independent radiologists.

Results—Thirty nine children were included. Fourteen scans were abnormal and two had clinically insignificant incidental findings. Four children with focal neurological signs had scans demonstrating extra-axial collections. None required neurosurgical intervention. Clinically, raised intracranial pressure was present in 10 patients. Only five had cerebral oedema on computed tomography; these five children died. Emergency cranial computed tomography influenced subsequent management in no child without focal neurological signs and in only one child with focal neurology.

Conclusion—Emergency cranial computed tomography in acute febrile encephalopathy in children without focal neurological signs has little influence on subsequent management. Where cranial computed tomography is thought to be necessary, it should be carried out when the child's clinical condition has been stabilised.

(J Accid Emerg Med 1999;16:403-406)

Keywords: febrile encephalopathy; cranial computed tomography; children

Fever may be associated with a variety of central nervous system (CNS) disorders. In children, acute infection of the CNS is the most common cause of fever associated with signs and symptoms of brain involvement.¹ Regardless of aetiology, most children with acute CNS infection present with similar signs and symptoms, including fever, headache, nausea, vomiting, anorexia, and irritability. Photophobia, myalgia, neck stiffness, obtundation, stupor, coma, seizures, and focal neurological signs may also be noted.

The differential diagnosis of a child who presents with fever and signs of CNS infection is broad, as the complications of CNS infection

include those of raised intracranial pressure (ICP) and the presence of a space occupying lesion.

Fever together with persistent alteration in conscious level, focal neurological signs, and/or seizures (acute febrile encephalopathy) may occur in both acute CNS infection and in non-infectious disorders such as intracranial haemorrhage or malignancy. This definition excludes simple febrile seizures, which are typically generalised tonic-clonic convulsions lasting no more than 10 minutes, followed by a brief period of drowsiness, with rapid resolution of all neurological signs.²

Diagnosis in children who present with acute febrile encephalopathy may be complicated by difficulty in carrying out diagnostic procedures. In particular, lumbar puncture is contraindicated due to clinical evidence of raised ICP or haemodynamic instability.³ Therefore the use of imaging procedures, such as cranial computed tomography, has become commonplace in the emergency management of children with acute febrile encephalopathy, in an attempt to assist in making a definitive diagnosis. Cranial computed tomography is often performed as an emergency procedure shortly after a child with acute febrile encephalopathy has been admitted to hospital or after deterioration. However, there are few published data showing that emergency cranial computed tomography in this population alters subsequent management. The only comparable data are from studies in bacterial meningitis.^{4,5} These studies have shown that in acute bacterial meningitis, clinical management is not influenced by cranial computed tomography findings. Any significant abnormality found on the scan was already clinically suspected.

As emergency cranial computed tomography in children with acute febrile encephalopathy has become part of routine management in many paediatric units, we set out to determine whether this procedure provided any additional, clinically useful information.

Patients and methods

We carried out a retrospective study on a cohort of children presenting consecutively to a regional medical paediatric intensive care unit (PICU) with a clinical diagnosis of acute febrile encephalopathy.

Children were diagnosed with acute febrile encephalopathy if they had fever >38°C, together with persisting depression of conscious level (Glasgow coma score <12), and/or sudden

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Accepted 17 July 1999

Table 1 Discharge diagnoses

Encephalitis	12
Bacterial meningitis	12
Atypical febrile seizure	9
Septicaemia	3
Cerebral malaria	1
Intraventricular haemorrhage	1
Sandhoff's disease	1

onset of seizures, with no obvious precipitating cause evident at the time of admission.

Children admitted to the PICU with acute febrile encephalopathy between February 1994 until January 1996 were included in the study.

All children were referred to the PICU from their local hospital because of deterioration in their clinical condition requiring intubation and ventilation. Obviously, any child who may have been suspected of having a condition requiring neurosurgical intervention may have been referred to a neurosurgical centre rather than our medical PICU.

The indication for referral to our PICU was either intractable seizures or acute reduction in conscious level. All children had a complete physical and neurological examination on admission to their referring hospital and on admission to the PICU. If patients were pharmacologically paralysed for the transfer, details of a history of focal neurological symptoms and signs from parents, and of their neurological examination before paralysis were noted. All patients had a full neurological examination before transfer to the PICU. In addition, muscle relaxation and sedation was weaned off as quickly as possible after PICU admission to allow detailed neurological evaluation.

Clinical features of raised ICP were recorded at the time of scanning. These consisted of persistent reduction or alteration in conscious level together with cardiovascular abnormalities including hypertension and bradycardia with or without pupillary changes.⁶

All children were tracheally intubated, mechanically ventilated, and had pharmacological sedation and muscle relaxation as part of their management during transfer. All were transported to the computed tomography scanner by a specialised paediatric intensive care transfer team.

Only children who had a cranial computed tomography performed within 12 hours of referral to the PICU were included in the study. The duration of illness before PICU admission varied. However, if referral to the PICU was taken to be the time when the child developed neurological failure, all children had

cranial computed tomography performed within 12 hours. The reason for including only these children was that we were evaluating the contribution to management of scans that were carried out acutely. Obviously, children may have had follow up scans later on in the course of their illness. However as these were not carried out in the acute phase, we have not evaluated their contribution to management.

Cranial computed tomography was carried out on a Toshiba Xpress HSI scanner; 5 mm contiguous scans were taken from foramen magnum to vertex. Where necessary, intravenous contrast solution (2 ml/kg Omnipaque 240) was given and before and after contrast scans were performed.

All scans were reviewed by two independent observers: a paediatric radiologist and a neuro-radiologist. The reviewers were blinded to the eventual diagnosis, management, and outcome of the patients.

The scans were assessed for the following: presence of cerebral oedema (compression of cerebral sulci, effacement of ventricles and/or basal cisterns, with or without abnormal grey/white matter differentiation); presence of any extra-axial collection; abnormal meningeal enhancement; evidence of central sinus thrombosis; parenchymal abnormality; and abnormal basal ganglia enhancement.

Decisions regarding influence of the scan on acute management and outcome of the patient were taken by the clinician directly involved in the child's medical management.

Results

Thirty nine children were included in the study (median age 3 years 6 months, range 1 month to 14 years 11 months). The discharge diagnoses are shown in table 1. Only children who had emergency scans because the diagnosis was unclear were included in this study.

Fourteen of 39 (36%) scans were considered to be abnormal. Two of these had what were considered to be incidental findings: one who had an old right occipitoparietal infarct and the other a choroid fissure cyst. Neither of these findings were clinically significant.

Table 2 Relevant cranial computed tomography abnormalities in children with acute febrile encephalopathy

Diagnosis	Computed tomography		Clinical evidence of raised ICP	Outcome
	Findings	Conclusion		
<i>Haemophilus influenzae</i> meningitis	Severely dilated ventricles with debris. Large left frontal subdural collection. Loss of grey/white matter differentiation	Severe cerebral oedema. Large left frontal subdural collection	Yes	Died
Meningococcal meningitis	Moderately dilated ventricles	Moderately dilated ventricles	Yes	Survived
Varicella encephalitis	Loss of grey/white matter differentiation	Mild cerebral oedema	No	Survived
Intracranial haemorrhage	Left thalamic haematoma. Severely dilated ventricles containing blood	Severe cerebral oedema. Intraventricular and parenchymal haemorrhage	Yes	Died
Atypical febrile seizure	Moderately dilated ventricles	Moderately dilated ventricles	No	Survived
Pneumococcal meningitis	Left frontal subdural collection	Left frontal subdural collection	No	Survived
Sandhoff's disease	Ventricular effacement and compression. Marked loss of grey/white matter differentiation	Severe cerebral oedema	Yes	Died
Pneumococcal meningitis	Mild effacement of basal cisterns	Mild cerebral oedema	Yes	Died
Meningococcal meningitis	Loss of grey/white matter differentiation. Left subdural collection	Mild cerebral oedema. Left subdural collection	No	Survived
Atypical febrile seizure	Loss of grey/white matter differentiation	Mild cerebral oedema	No	Survived
Group B streptococcal meningitis	Ventricular compression. Effacement of basal cisterns. Loss of grey/white matter differentiation. Possible central sinus thrombosis	Severe cerebral oedema, possible central sinus thrombosis	Yes	Died
Meningococcal meningitis	Left parietal lobe infarction	Left parietal lobe infarction	No	Survived

Table 2 lists the scans thought to have clinically significant findings. Some scans had more than one abnormality.

Four scans demonstrated extra-axial collections: three subdural collections and one intraventricular haemorrhage. All these children had focal neurological signs on admission. None required acute neurosurgical intervention after consultation with neurosurgeons. All other children had no evidence of abnormal focal neurology.

Eight scans demonstrated cerebral oedema. However, clinical evidence of raised ICP was present in 10 patients. Only seven of these children had abnormal cranial computed tomography (one of these was an incidental finding: an old cerebral infarct); the other had mildly dilated ventricles.

Therefore only five children with cerebral oedema on cranial computed tomography had clinical features of raised ICP. These five children died. Three of these had bacterial meningitis and one had a decompensation of an underlying metabolic disorder (table 2). The fifth child who died had an intraventricular haemorrhage. He had features of severely raised ICP with fixed, dilated pupils, absent brain stem reflexes, and retinal haemorrhages on presentation to his local hospital.

All children with clinical signs of raised ICP were treated acutely for this problem (using standard measures, that is osmotic diuretics, loop diuretics, head elevation to 20 degrees, head kept in the midline, sedation, etc), but those who subsequently died developed signs of brain stem death, either on arrival at the PICU or within 12 hours of their admission to the PICU.

In contrast three children with cerebral oedema on cranial computed tomography had no clinical evidence of raised ICP. They were not treated for raised ICP. These children survived with no neurological sequelae.

No child with cerebral oedema alone on cranial computed tomography had focal neurological signs. In addition, no child with a normal cranial scan (62%) had abnormal focal neurology. All scans were assessed for evidence of bony injury using bone windows, however no case of skull fracture was discovered.

Only in one case was a change in management made after emergency cranial computed tomography. This child had meningococcal meningitis and presented with a right sided hemiparesis. Computed tomography demonstrated a left parietal lobe infarct. After the diagnosis anticoagulant treatment was initiated because it was felt after discussion that the child might benefit from this treatment.

Discussion

Our results have demonstrated that in children with acute febrile encephalopathy without focal neurological signs, performance of emergency cranial computed tomography does not influence subsequent management. There was no change in management after possession of data from the scan in children with a non-focal neurological examination. Even in children with abnormal cranial computed tomography,

only in one case, a child with focal neurology and a parietal lobe infarct, did this influence subsequent management.

Acute neurosurgical intervention was not required for any child in our study. The only case where it may have been beneficial was in a child with clinical signs of acute intracranial haemorrhage, who had physical signs suggesting intracranial haemorrhage on admission to his referring hospital. Unfortunately, by the time the diagnosis was confirmed, this child was already brain stem dead.

Only children with what were thought to be non-surgical causes of their acute encephalopathy were referred to our unit. Therefore there may have been some referral bias in that patients with lesions amenable to neurosurgical intervention may have been under-represented in this sample. The presence of a neurosurgical lesion is extremely unlikely in a previously healthy child with acute onset of fever and encephalopathy and no history of trauma, where a diagnosis of acute bacterial meningitis or encephalitis is much more likely.

While presentation of a child with progressive coma or convulsions is a desperately worrying situation, emergency computed tomography should not be regarded as a priority in management. In these children, secondary neurological damage may be caused by hypoxic-ischaemic encephalopathy due to inadequate respiratory drive or airway obstruction, leading to cerebral oedema. The clinical manifestations of cerebral oedema and raised ICP show a characteristic rostrocaudal progression. Obtundation, confusion, restlessness, agitation, and progressive unresponsiveness are early manifestations. Abnormal respiratory pattern, unilateral or bilateral pupillary dilatation and sluggish response to light, dystonic movements or posturing, or weak or absent doll's eye response represent clinical deterioration, ultimately progressing to apnoea and flaccidity as brain stem compression occurs. Papilloedema and cardiovascular changes usually occur at a later stage of progression. The earlier neurological failure is recognised and treatment is instituted, the better are the chances of a favourable outcome. In these children, the priority is airway control and ventilation, together with control of fluid balance and seizures.³

We have been referred children admitted to their local hospital in coma, where the first intervention was to send the child for cranial computed tomography with inexperienced medical and nursing staff to a radiology department where there are poor monitoring and resuscitation facilities. Unfortunately, intervention to treat neurological failure is not always carried out and children have died in the scanner, with a normal cranial scan. In addition, there are obvious risks in transporting sick children, even the relatively short distances to a radiology department, by non-specialised teams. In one large study, nearly 10% of non-ventilated critically ill children and over one third of ventilated critically ill children had significant physiological deterioration during intrahospital trans-

port, which required major intervention.⁷ All children in our study were transported to the radiology department by a highly trained and specialised paediatric intensive care transport team. There were no transport related adverse events in our children.

If computed tomography is felt to be necessary it should be carried out when clinical stability has been achieved and experienced staff and adequate monitoring is available.

The insensitivity of cranial computed tomography in predicting the presence of raised ICP was confirmed in our study and has been substantiated in several other studies.^{8,9} Of the 10 children with clinically raised ICP, only five showed evidence on computed tomography. The other five scans showed no evidence of cerebral oedema.

Intracranial pressure monitoring was not performed in any of the children in this study. The use of ICP monitoring in children with non-traumatic brain injury has not been shown to be associated with improvement in outcome, and therefore is not routinely performed at our institution.¹⁰ The lack of correlation between physical signs of raised ICP and computed tomography evidence of cerebral oedema once again demonstrates the misapprehension that a normal scan ensures the safety of performance of lumbar puncture, in the absence of focal neurological signs. In these patients a normal scan does not ensure that tonsillar herniation will not occur after lumbar puncture.⁹ Decision to perform lumbar puncture must be clinically based. If there is any doubt, lumbar puncture should be deferred, with presumptive antibiotic and antiviral treatment not delayed. Newer methods of laboratory diagnosis using serum or plasma, such as the polymerase chain reaction, make it less necessary to perform lumbar puncture for diagnostic purposes. In addition, even if lumbar puncture is delayed while clinical stability is achieved, useful information may subsequently be gleaned from the cerebrospinal fluid.

This is not to say that information derived from the scans performed was not useful. Usually a scan is requested in order to rule out an important differential diagnosis and occasionally computed tomography will reveal an abnormality that was not clinically suspected. In addition, the presence of a normal scan may provide some reassurance to the clinicians caring for the child. Even in this scenario, however, this reassurance may be false if there are clinical signs of raised ICP.

In our study no patient who had a non-focal history or physical examination had a significant focal abnormality on cranial computed tomography. This finding is in agreement with other studies carried out in children with acute bacterial meningitis.^{4,5} In all patients where there was a focal abnormality on cranial computed tomography, the presence of focal brain pathology had already been demonstrated clinically.

The findings of our study has altered management in similar patients. We do not now carry out emergency cranial computed tomography on admission. When such children are referred to the PICU, we discuss the above findings to reinforce that computed tomography is not a priority.

In the absence of focal neurological signs we perform standard neurointensive care. When indicated, we perform lumbar puncture to try to reach a diagnosis, followed by cranial imaging when required. In patients who have no cardiovascular instability, we are prepared to postpone imaging until it is felt necessary.

Conclusion

Performance of emergency cranial computed tomography in children with acute febrile encephalopathy without focal neurological signs is not a sensitive aid for the diagnosis of acute brain pathology. In addition, it has poor correlation with the presence of raised ICP and hence the safety of lumbar puncture. In those children with non-focal neurological signs where cranial computed tomography is thought to be necessary, it should be delayed until the child's clinical condition has been stabilised and until experienced staff and adequate monitoring are available.

Presented in part at the 1st Annual Meeting of the Royal College of Paediatrics and Child Health, York, April 1997.

Conflict of interest: none.

Funding: none.

Contributors

Simon Nadel initiated and coordinated the study, collected and collated clinical data, and wrote the manuscript. Rita Joarder and Matthew Gibson collected radiological data, collated results with clinical data, and contributed to writing the results. John Stevens reported on the computed tomography and helped with data interpretation and editing of the manuscript. Joseph Britto and Parviz Habibi collected clinical data, helped with data interpretation, and edited the manuscript. Catherine Owens designed the study, reported on the computed tomography, and helped write and edit the manuscript.

Simon Nadel and Catherine Owens are the guarantors for the study.

- 1 Feigin RD. Fever associated with diseases of the central nervous system. In: Behrman RE, ed. *Nelson textbook of pediatrics*. Philadelphia: W B Saunders, 1992: 654–5.
- 2 Haslam RHA. The nervous system. In: Behrman RE, Kliegman RM, Arvin AM, eds. *Nelson textbook of pediatrics*. Philadelphia: W B Saunders, 1996: 1667–763.
- 3 Berkowitz ID, Berkowitz FE, Newton C, et al. Meningitis, infectious encephalopathies and other central nervous system infections. In: Rogers MC, ed. *Textbook of pediatric intensive care*. Baltimore: Williams and Wilkins, 1996: 1039–90.
- 4 Friedland IR, Paris MM, Rindernecht S, et al. Cranial computed tomographic scans have little impact on management of bacterial meningitis. *Am J Dis Child* 1992;146: 1484–7.
- 5 Cabral DA, Flodmark O, Farrell K, et al. Prospective study of computed tomography in acute bacterial meningitis. *J Pediatr* 1987;111:201–5.
- 6 Sarniak AP, Lieh-Lai MW. Transporting the neurologically compromised child. *Pediatr Clin North Am* 1993;40:337–54.
- 7 Wallen E, Venkataraman ST, Grosso MJ, et al. Intrahospital transport of critically ill pediatric patients. *Crit Care Med* 1995;23:1588–95.
- 8 Rennick G, Shann F, de Campo J. Cerebral herniation during bacterial meningitis in children. *BMJ* 1993;306:953–5.
- 9 Archer BD. Computed tomography before lumbar puncture in acute meningitis: a review of the risks and benefits. *Can Med Assoc J* 1993;148:961–5.
- 10 Filloux F, Dean JM, Kirsch JR. Monitoring the central nervous system. In: Rogers MC, ed. *Textbook of pediatric intensive care*. Baltimore: Williams and Wilkins, 1996: 667–98.