Table 1 No (%) of times indicator of risk of child abuse marked on checklist during visits by children to accident and emergency at first (1995) and second (1996) audit

Indicator for risk of child abuse	Indicator marked on checklist No (%)*	
	First audit (1995)	Second audit (1996)
Children have all five indicators marked	860/1357 (63.3)	940/988 (95.1)
Child protection register list in accident and emergency checked for all children living in surrounding local authorities	989/1033 (95.7)	801/806 (99.4)
All children with two or more indicators discussed with the on call paediatric registrar	2/52 (4)	8/41 (20)
All children whose names are on the child protection register discussed with the on call paediatric registrar	1/5 (20)	NA
All children < 1 year old with fractures or head injuries referred to the on call paediatric registrar‡	NA	6/12 (50)†

NA=not applicable.

*Target for compliance with checking all indicators was 100%.

†In two cases this was one of two risk indicators; one of these was referred to the paediatrician.

‡Category not included in first audit.

register. This is fewer than would be expected from the local prevalence (47.1 per 10 000) and may reflect variations in prevalence within the boundaries of the local authority.³ The proportion of children with one positive indicator of risk remained constant at 40% (543 children in the first audit; 392 in the second), as did the proportion of those with two (4%; 50 and 40 children respectively), and more than two (0.1%; 2 and 1).

Comment

Clarification of protocols for child protection in the accident and emergency department, regular staff training, and increased communication between paediatricians and nurses in the department led to improvements in identification of children thought to be at risk of child abuse. Improvement was seen in all standards but espe-

cially in those relating to nurse triage. Time spent in training and feedback resulted in the development of good rapport between nursing staff and paediatricians and a commitment to achieve high standards in the child protection procedures. In contrast, the overall rate of referral of children thought to be at risk remained low. Just 3 out of the 57 children (5.3%) identified as being at risk were discussed with paediatricians in the first audit, this increased to 13 out of 51 (25.5%) in the second. Improvements were made in training junior medical staff in issues of child protection; however, with a regular turnover of staff this needs to be sustained and reinforced by close liaison between the accident and emergency and paediatric departments.

This audit showed whether procedures for identifying children thought to be at risk of abuse were being followed. The checklist of indicators of risk does not identify children who have been abused, but merely heightens awareness of those children in whom there are features that might cause concern. What the audit does not show is whether these procedures can accurately identify children who have been abused. Over a longer time it would be possible to review the records of all children identified as having been abused and use any records from visits to the accident and emergency department to determine the predictive value of the indicators of risk.

Funding: None.

Conflict of interest: None.

- 1 Department of Health. *Child abuse. A study of inquiry reports 1980-89*. London: HMSO, 1991.
- 2 Phillips BM, Robson WJ. Paediatrics in the accident and emergency department. *Arch Dis Child* 1992;67:560-4.
- 3 Smith J. *Avon Joint Child Protection Committee, annual report 1995*. Bristol: Avon Social Services Department, 1995.

(Accepted 11 March 1997)

Cerebral and cerebellar atrophy on serial magnetic resonance imaging in an initially symptom free subject at risk of familial prion disease

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BMJ 1997;315:856-7

Prion diseases are transmissible neurodegenerative conditions which occur in sporadic, acquired, or inherited forms. Sporadic Creutzfeldt-Jakob disease presents as a rapidly progressive dementia with myoclonus, most patients showing characteristic pseudoperiodic complexes on electroencephalography. A new variant of Creutzfeldt-Jakob is characterised by a younger age at onset, longer survival, and non-specific initial symptoms including behavioural changes, dysaesthesias, and ataxia but without the expected electroencephalographic changes.¹ Some of these features are also found in 5-15% of all cases of the inherited forms of Creutzfeldt-Jakob disease and are associated with mutations in the prion protein gene.² We report the case of a woman with a family history of prion disease in whom progressive cerebral and cerebellar atrophy was seen when two

magnetic resonance scans were compared using registration.

Case report

A 42 year old woman from a family with histologically confirmed prion disease entered a study of symptom free subjects. She had no complaints of physical or cognitive problems and her lack of symptoms was confirmed by her family. Neurological examination and neuropsychometry gave normal results. She scored 29/30 on the mini-mental state examination. Her magnetic resonance scan was interpreted, by a neuroradiologist (JMS) blind to clinical details, as showing widening of cortical sulci, particularly those of the parietal lobes. In T2 weighted images there were no obvious abnormalities.

Coronal slice from initial magnetic resonance scan overlaid in red to show tissue loss that had occurred in second scan. Green overlay shows points of signal gain in second scan

Nine months later she reported that her balance was not normal and that her legs felt stiff. Over the next six months her balance deteriorated and her family noticed that she was emotionally labile. She was referred for neurological evaluation after a fall. She had a broad based ataxic gait, dysarthria, and jerky pursuit eye movements. No myoclonus was observed; tone, power, and tendon reflexes were normal. She scored 29/30 on the mini-mental state examination. Neuropsychometry showed a decline only in verbal recognition memory and comprehension of abstract words. Laboratory investigations and electroencephalography gave normal results. A magnetic resonance scan showed diffuse atrophy of all elements of the brain including the caudate nucleus, the thalamus, cerebellum, and pons. There was again no detectable signal change in the basal ganglia. A diagnosis of hereditary prion dementia was confirmed by finding a point mutation at codon 102 of the prion protein gene (PrP Leu 102).

The first two magnetic resonance scans were registered and atrophy was detected by subtraction of the second scan from the first.³ The figure shows points of signal loss >20% of the mean (atrophy) in red and points of signal gain in green. Atrophy was widespread, with a total loss of cerebral tissue of 45 ml (4.3% of total brain) and of cerebellar tissue of 9 ml (7.9%).

Comment

Neuroimaging may show atrophy in established prion disease, but magnetic resonance imaging gives normal results in almost 50% of cases.⁴ Signal changes in the basal ganglia on T2 weighted imaging may be a distinctive early sign of Creutzfeldt-Jakob disease,⁵ but in one study such signal abnormalities were reported in only 4 out of 96 cases.⁴

Presymptomatic parietal atrophy was seen in our patient's first magnetic resonance scan, but the appearance of the parietal sulci generally varies widely. In contrast, rapidly progressive cortical and cerebellar

atrophy was seen when the scans were compared using registration. The rate of brain atrophy was much greater than that in normal aging and rapid even for Alzheimer's disease. The pattern of atrophy, affecting the cerebellum and sparing medial temporal structures, differed from that in Alzheimer's disease, in which the cerebellum is usually unaffected.³ The rapid cerebellar atrophy was consistent with the clinical symptoms which included ataxia—often an early feature in familial prion disease associated with the leucine 102 mutation of the prion protein gene. It is also common in iatrogenic and new variant Creutzfeldt-Jakob disease. The technique of registration of serial volumetric magnetic resonance images may prove helpful in diagnosing neurodegenerative disorders.

We thank the family members who generously participated in the longitudinal studies.

Funding: NCF holds an Alzheimer's Disease Society research fellowship; PAF and KFM are supported by the Charles Wolfson Charitable Trust.

Conflict of interest: None.

- 1 Will RG, Ironside JW, Zeidler M, Cousens SN, Estibeiro K, Alperovitch A, et al. A new variant of Creutzfeldt-Jakob disease in the UK. *Lancet* 1996;347:921-5.
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(Accepted 3 February 1997)

Correction

Age related dietary exposure to meat products from British dietary surveys of teenagers and adults in the 1980s and 1990s

An authors' error in this paper by Sheila M Gore and colleagues (16 August, pp 404-5) led to the omission of acknowledgement to Ailsa A Welch, Robert N Luben, and Suzy F Oakes, who provided the EPIC data.

Endpiece

Hopefully, the last word

Since at least the 17th century, certain adverbs ending in -ly have acquired the ability to qualify a predication or assertion as a whole. Such adverbs are elliptical uses of somewhat longer phrases ... In the 20th century there has been a swift and immoderate increase in the currency of [such] adverbs [which] include actually, basically, frankly, hopefully, regretfully, strictly, and thankfully. Suddenly, round about the end of the 1960s, and with unprecedented venom, a dunce's cap was placed on the head of anyone who used just one of them—hopefully—as a sentence adverb ... Conservative speakers, taken unawares by the sudden expansion of an unrecognised type of construction, have exploded with resentment that is unlikely to fade away before at least the end of the 20th century.

Robert Burchfield, *Fowler's Modern English Usage*, Clarendon Press, Oxford (1996)

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