Neuroradiology (1989) 31: 129-133

Neuro radiology

Lesions of the brain stem: assessment by magnetic resonance imaging

J. V. Byrne, B. E. Kendall, D. P. E. Kingsley, and I. F. Moseley

Lysholm Department of Radiology, The National Hospitals for Nervous Diseases, London, England

Summary. Ninety-six magnetic resonance imaging (MRI) studies demonstrating solitary brain stem lesions were reviewed in order to establish distinguishing features between tumours and other lesions. Histological confirmation of the radiological diagnoses was obtained in 33% of patients. The morphology of the lesion rather than its signal characteristics was the most useful feature in differential diagnosis, except after haemorrhage. However, areas of abnormal T2 recovery time were significantly more extensive than areas of abnormal T1 recovery time in tumours at presentation. In other lesions and in tumours following radiotherapy induced regression, the extent of abnormal T1 and T2 signal was the same. Possible reasons for this observation are discussed. The accuracy of computed tomography (CT) and MRI were similar for lesions causing brain stem expansion but for small lesions MRI was more sensitive and provided better topographical information.

Key words: Magnetic resonance imaging - Computed tomography - Brain stem tumour - Brain stem haemorrhage - Brain stem infarction

The superiority of magnetic resonance imaging (MRI) over computed tomography (CT) in the diagnosis of brain stem tumours has been demonstrated in a number of reports [1-4] and has been largely attributed to production of primary images in clinically relevant planes and the lack of obscuration by bone generated artefact [5, 6].

Biopsy of brain stem tumour is still considered sufficiently hazardous that many physicians recommend empirical treatment, which inevitably results in a small number of non-neoplastic lesions being inappropriately treated by radiotherapy. Optimum pretreatment assessment is thus mandatory. Accuracy in localisation as well as diagnosis is also important in planning and assessing response to radiotherapy.

We have reviewed in detail a large number of MRI studies showing solitary brain stem lesions. The ability of this technique to locate neoplasms and to differentiate them from other lesions within the brain stem was assessed by comparison of signal changes on T1- (T1W) and T2-weighted (T2W) sequences, in biopsied and unbiopsied lesions. The MRI findings were also compared with those of contempory CT.

Material and methods

Between June 1985 and December 1987, 304 patients with suspected brain stem pathology were referred for cranial MRI scanning at the Queen Square Imaging Centre. Solitary lesions of the brain stem were demonstrated in 80 patients. Four of these were excluded because of incomplete records. Ninety-six examinations were performed in the remaining 76 patients. Six patients, examined for the first time after radiotherapy or a surgical procedure to the posterior fossa, are considered with the follow up examinations.

The MRI examinations were conducted on a 0.3 Tesla superconducting whole body imager (Picker U.K. Ltd.). Generally 3 sequences were employed; a T2-weighted spin echo (SE) sequence (TE 80, TR 2000), a strongly T1-weighted inversion

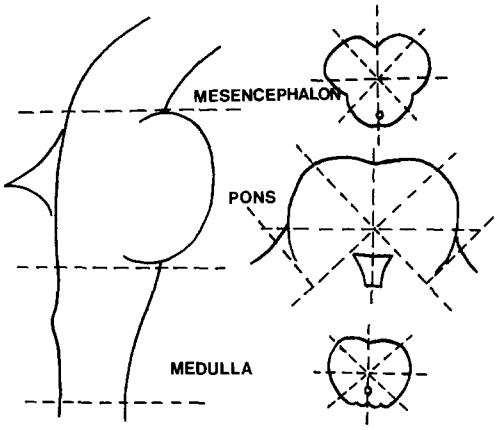

Fig. 1. Scoring of brain stem involvement T1W and T2W images were separately assessed. Involvement at each illustrated level scored 1–8 and extension beyond the brain stem 1–4; maximum possible score 28

recovery (IR) sequence (T1 500, TR 2000) and an intermediate weighted SE sequence (TE 40, TR 500).

The MRI images were reviewed independently by all 4 authors without reference to the clinical details. Based on these interpretations the studies were assigned to one of 3 groups;

Group A. Lesions returning long T1 and long T2 recovery times with some degree of brain stem expansion.

Group B. Lesions returning short T1 and long or short T2 recovery times.

Group C. Lesions in which T1 and T2 recovery times were prolonged but mass effect was minimal or absent.

In order to compare the extent of abnormal signal associated with each lesion, on T1W and T2W sequences, areas of abnormal signal were coded from the images as follows. The brain stem was divided into 3 vertical levels i.e. mesencephalon, pons and medulla, and each level subdivided into 8 segments in the transverse plane (Fig. 1). Areas of abnormal signal were scored 1 to 8 in the transverse plane and values for the three levels summed. Signal change extending supratentorially or into the cervical cord scored one each and into the cerebellum one or 2 depending on whether one or both hemispheres were involved. Thus, a digital profile of each lesion was obtained with a maximum possible score of 28.

The degree of brain stem expansion and associated hydrocephalus were also graded and evidence of exophytic elements and calcification recorded.

Relevant CT studies, available in 53 patients, were also reviewed for mass effect, hydrocephalus, calcification and the presence of exophytic elements.

All patients were followed up through the records of The National Hospitals for Nervous Diseases or by postal inquiry of the referring doctor.

Results

The ratio of male to female patients was as follows: Group A, 24:25, group B, 5:3 and group C, 10:3. The distribution of ages is listed in Fig. 2.

Group A

This group comprised 49 patients examined before any treatment other than a CSF shunt procedure. Histological diagnosis was obtained in 18 patients (group A1): 17 astrocytomas and one ependymoma. The diagnosis in the remainder (group A2) is as yet unconfirmed.

All lesions had prolonged T1 and T2 recovery times; additional areas of short T1 consistent with haemorrhage were demonstrated in 3 patients who are also considered in Group B. Abnormal areas demonstrated on MRI involved the pons in 80% the mesencephalon in 45% and the medulla in 69% of scans. Extension occurred into the cerebrum in 20%, into the cervical cord in 25% and into the cerebellum in 40% of scans. The relative extent of abnormally prolonged T1 and T2 is compared in Table 1 for lesions in this and the following groups. Changes on the T2W images were significantly more extensive than T1W changes in group A1 and A1 + A2 but not in group A2.

CT studies were available for 38 patients, 14 from group A1 and 24 from group A2. There was one false negative CT scan. All the lesions demonstrated were diagnosed as gliomas. The radiological features of these and the MRI studies in the same patients are presented in Table 2.

Group B

This group consists of 11 patients with MRI features highly suggestive of haemorrhage, namely lesions with short T1 and long or short T2 elements. Seven patients scanned between one and 4 weeks of the onset of symptoms showed pronounced hyperintensity on the IR sequence in a small area of the brain stem. In a further patient similar findings one week after the onset of symptoms, had not been apparent at 48 h. The 3 other patients, who are also included in group A, had foci of short T1 within larger areas of prolonged T1 and T2, with considerable

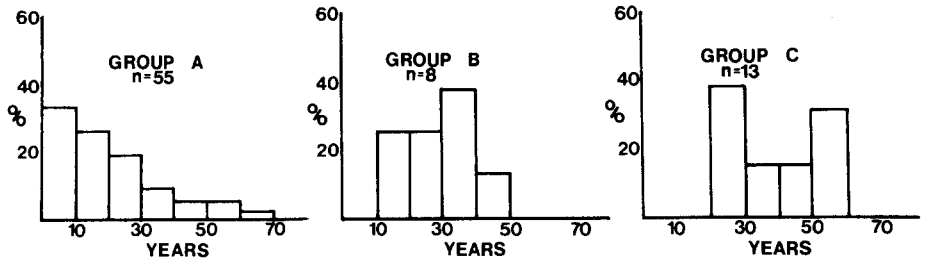

Fig. 2. Age distribution by decade amongst three major groups

mass effect in two patients: one of these lesions was an ependymoma, the other has not been biopsied. Haemorrhage in the third patient was also presumed to be within a tumour, following enlargement of the lesion on a second MRI study 6 months later.

The 8 lesions without evidence of associated tumour were located in the three central levels: pons 54%, mesencephalon 38% and medulla 13%; none extended beyond the brain stem. Areas of abnormal signal were similar in extent for both T1W and T2W sequences (Table 1).

CT studies were available for review in 7 patients: all showed areas of increased attenuation which matched areas of short T1.

Group C

This group consists of 13 patients with lesions giving prolonged T1 and T2 recovery times, without expansion of the brain stem. Histology was obtained in 3 patients: revealing lymphoma, infarction and oedematous white matter respectively. The last patient was subsequently diagnosed as having systemic lupus erythematosis. In the remaining 10 patients the diagnoses made as a result of these studies: infarction 4, multiple sclerosis 2, pontine myelinolysis 2, glioma 1 and arteriovenous malformation 1, remain unconfirmed.

The MRI features were non-specific except for the presence of flow void in a presumed arteriovenous malformation. The diagnosis of pontine myelinolysis was suggested by the symmetry of pontine lesions in two patients, one of whom had an appropriate clinical history of hyponatraemia. Follow up of these and the remaining patients for between 10 months and 2 years has confirmed that these lesions were non-progressive. All lesions were confined to the brain stem, affecting the pons in 54%, mesencephalon in 30% and medulla in 38% of cases. The extent of abnormal signal was similar on T1W and T2W sequences.

CT studies were available in 8 patients and were normal in five, details are included in Table 2. The diagnoses made on the basis of these scans were glioma in two and pontine myelinolysis in one patient.

Table 1. Extent of abnormal signal on T2 and T1 weighted sequences

Group	*n*	T2	T1	T2-T1	*P*
A1	18	11.1±1.2	9.9±1.0	1.8±0.6	<0.007
A2	31	14.0±1.0	13.2±1.0	0.8±0.6	NS
A1+A2	49	13.2±0.8	12.0±0.8	1.2±0.04	<0.005
B	8	3.9±0.8	4.4±0.9	0.1±0.9	NS
C	13	4.7±1.0	4.0±1.2	0.7±0.4	NS
A3	10	16.7±1.9	13.9±1.5	2.8±0.9	<0.01
A3 (after therapy)	10	10.5±1.9	10.5±1.7	0.0±1.3	NS

Mean coded values±SEM; *P* by *t* test

Table 2. Radiological features in patients examined by both MRI and CT

Group	A1		A2		B		C	
n	14		24[a]		7		8[b]	
	MR	CT	MR	CT	MR	CT	MR	CT
Mass								
minimal	0	0	0	0	6	6	8	1
moderate	9	9	11	11	1	1	0	2
severe	6	5	13	12	0	0	0	0
Hydrocephalus	5/14	6/12	8/23	10/22	0	1/7	0	0
Exophytic	6	3	8	5	0	0	0	0
Calcification	0	1	0	4	0	0	0	0
Enhancement	-	3	-	11	-	1	-	1

[a] CT "normal" in 1 patient
[b] CT "normal" in 5 patients

Postoperative and follow up scans

Four patients were first examined by MRI after surgical resection and histological diagnosis of tumours: glioma in 3 and choroid plexus papilloma in one. Two further patients were examined after radiotherapy without biopsy.

Recurrent or residual tumours were diagnosed in three patients, on the basis of considerable tumour mass and/or exophytic elements. In one patient residual tumour, if present, could not be distinguished from postoperative change.

Ten patients from group A (subgroup A3, Table 1) were studied following radiotherapy; be-

tween 2 and 12 months after the initial examination. Two tumours appeared larger, 4 were unchanged and 4 were smaller. Those which had regressed also returned less intense signal from areas of previously prolonged T1 and T2 recovery time. The extent of the lesions on T2W images was significantly larger than on T1W images prior to therapy, in keeping with the majority of group A. After radiotherapy, this difference was not apparent. Subsequent images in two patients, in whom tumour regression had initially occurred, showed an increase in signal intensity coincident with enlargement of the mass and symptomatic deterioration.

Discussion

This study, like most others concerning brain stem lesions, suffers from a lack of histologically proven diagnoses, due to the potential or perceived hazards of biopsy. MRI is generally regarded as the most sensitive radiological means of detecting structural lesions in the posterior fossa [5]. We have used it as such to define our study population and grouped patients on the basis of lesion morphology and signal.

All tumours returned signal indicating prolonged T1 and T2 recovery times, but this alone was not characteristic, since similar changes occurred in group C. However areas of abnormal signal on T2W scans were more extensive than those on T1W scans in the proven tumours, but not in the non-neoplastic subgroups. It is tempting to postulate that the presence of peritumoural vasogenic oedema is responsible for this difference, but although it is frequently stated to be the case, there is as yet no conclusive evidence that T2W sequences are more sensitive to such oedema per se. A linear relationship has been shown between peritumoural brain water content and T1 recovery time [7], prolongation of T1 being attributed to an increase in free mobile water. Lengthening of T2 is more complex; two components can be measured under pathological conditions with quantitative differences between vasogenic and cytotoxic oedema which probably reflect differences in protein content and in location of water protons [8]. It is not possible to draw firm conclusions from scans of mixed weighting but the results do suggest that relatively more extensive T2W changes are a feature of neoplastic activity. The finding that the ratio of T1 and T2 abnormality came to resemble that of the non-neoplastic groups after radiotherapy and symptomatic improvement supports this contention, but the intensity of abnormal signal was less on both sequences. Care must therefore be exercised in interpreting the pathophysiology of these early radiation induced changes.

The morphology of the lesions proved the most useful, but not totally reliable, feature in distinguishing tumours from other lesions. Pronounced brain stem expansion (classed severe on our scale of mass), was only seen in group A patients but the absence of mass effect did not exclude tumour. However the presence of an exophytic element, which MRI demonstrated twice as often at CT, was reliable in making the distinction. In this series calcification was confined to patients in group A though since it may occur in association with non-neoplastic lesions such as arteriovenous malformations it cannot be considered definitive.

Improved definition of lesions may provide information of prognostic significance [9]. It has been suggested that tumours with exophytic elements [10], or those which are hypodense or appear to involve the entire brain stem on CT [11] are associated with reduced survival times.

Patients with haematomas were scanned acutely or sub-acutely while typical high density was still detectable on CT. On images obtained very early or at a late stage, characteristic features of haemorrhage may be absent on MRI and CT respectively. Hyperintensity on T1W sequences, characterising haemorrhage, is delayed until red cell lysis and liberation of methaemoglobin occurs [12] whilst decreasing CT density occurs synchronously, so that the two modalities are to an extent complimentary in the imaging diagnosis of haematomas. The early variations in T2 recovery time are complex and depend on field strength and the timing of the examination relative to the ictus [13]. Morphology as well as signal is important in the recognition of an underlying causative lesion.

The necessity to pursue the diagnosis of many small brain stem lesions is dubious, so that the aetiology of the majority of lesions in group C remains uncertain. However MRI proved a sensitive method of diagnosis and is especially suitable following their evolution.

Follow-up examinations after treatment are frequently ambiguous since signal changes are non-specific and differentiation between postoperative and radiation induced changes from tumour recurrence may be impossible [14]. However, the present study confirms the suggestion that areas of altered signal may return to normal after radiotherapy in association with tumour shrinkage [15] only for the abnormal signal to return with a recurrence.

X-ray CT is the alternative radiological technique generally employed in the detection and localisation of brain stem lesions. In this series we compared the findings with those of MRI but the sensitivity or specificity can not be determined since the latter tech-

nique was used to define the study population. Despite beam hardening artefacts and the inability to perform scans in multiple planes, CT revealed all but one of the lesions associated with brain stem expansion and all the haemorrhages. However, in the absence of mass effect it proved less reliable; 5 of 8 nonexpanding lesions identified on MRI were not detected on CT; all these patients were examined on a modern scanner (GE 9800). Other studies such as positive contrast CT cisternography are unlikely to add further information in such cases.

In conclusion, in the detection of brain stem tumours, MRI and CT were comparable; but for small and non-neoplastic lesions MRI was better and allowed more confident but qualified distinction of neoplastic from non-neoplastic lesions. It is therefore the more reliable means of imaging a structural abnormality in the brain stem. The relative extent of abnormal signal on T1W and T2W sequences may prove useful in tumour recognition.

References

1. Bradac GB, Schorner W, Bender A, Felix R (1985) MRI (NMR) in the diagnosis of brain stem tumours. Neuroradiology 27: 208-213
2. Hueftle MG, Han JS, Kaufman B, Benson JE (1985) MR imaging of brain stem gliomas. J Comput Assist Tomogr 9: 263-267
3. Peterman SB, Steiner RE, Bydder GM, Thomas DJ, Tobias JS, Young IR (1985) Nuclear magnetic resonance imaging (NMR), (MRI), of brain stem tumours. Neuroradiology 27: 202-207
4. Zimmerman RA, Bilaniuk LT, Packer R, Sutton L, Samuel L, Johnson MH, Grossman RI, Goldberg HI (1985) Resistive NMR of brain stem gliomas. Neuroradiology 27: 21-25
5. Lee BC, Kneeland JB, Deck MDF, Cahill PT (1984) Posterior fossa lesions: magnetic resonance imaging. Radiology 153: 137-143
6. Bydder GM, Steiner RE, Thomas DJ, Marshall J, Gilderdale DJ, Young IR (1983) Nuclear magnetic resonance of the posterior fossa: 50 cases. Clin Radiol 34: 173-188
7. MacDonald HL, Bell BA, Smith MA, Kean DM, Tocher JL, Douglas RHB, Miller JD, Best JJK (1986) Correlation of human NMR T1 values measured in vivo and brain water content. Br J Radiol 59: 355-357
8. Naruse S, Horikawa Y, Tanaka C, Hirakawa K, Nishikawa H, Yoshizaki K (1982) Proton nuclear magnetic resonance studies on brain edema. J Neurosurg 56: 747-752
9. Stroink AR, Hoffman HJ, Hendrick EB, Humphreys RP (1986) Diagnosis and management of paediatric brain-stem gliomas. J Neurosurg 65: 745-750
10. Cohen ME, Duffner PK, Heffner RR, Lacey DJ, Brecker M (1986) Prognostic factors in brain stem gliomas. Neurology 36: 602-605
11. Albright AL, Guthkelch AN, Packer RJ, Price RA, Rourke LB (1986) Prognostic factors in pediatric brain stem gliomas. J Neurosurg 65: 751-755
12. Gomori JM, Grossman RI, Goldberg HI, Zimmerman RA, Bilaniuk LT (1985) Intracranial hematomas: imaging by high-field MR. Radiology 157: 87-93
13. Zimmerman RD, Heier LA, Snow RB, Kelly AB, Deck MDF (1988) Acute intracranial hemorrhage: intensity changes on sequential MR scans at 0.5 T. AJNR 9: 47-57
14. Dooms GC, Hecht S, Brand-Zawadzki M, Berthiaume Y, Norman D, Newton TH (1986) Brain radiation lesions: MR imaging. Radiology 158: 149-155
15. Packer RJ, Zimmerman RA, Luerssen TG, Sutton LN, Bilaniuk LT, Bruce DA, Schut L (1985) Brainstem gliomas of childhood: Magnetic resonance imaging. Neurology 35: 397-401

Received: 7 December 1988

Dr. J. V. Byrne
Department of Neuroradiology
Atkinson Morley's Hospital
Wimbledon
London SW20 0NE
England