MRI in 31 patients with Behçet's disease and neurological involvement: prospective study with clinical correlation

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Abstract

Thirty one patients with Behçet's disease and neurological manifestations were prospectively studied with MRI. Cerebral venous thrombosis was diagnosed in 10 patients. MRI performed during the acute illness in eight patients showed an abnormally high signal on the T2 weighted sequences in the occluded sinus. MRI showed minor flow abnormalities suggestive of partial recanalisation of the sinus in two cases at a later clinical stage. MRI can be an alternative, non-invasive, investigation to intravenous cerebral angiography. In 13 patients with central nervous system involvement, MRI performed during the acute illness showed multiple hyperintense lesions on T2 weighted sequences. They were usually less than 5 mm, scattered and confluent, mainly in the white matter, distributed in the hemispheric white matter in nine cases, brainstem in eight, basal ganglia and thalamus in five, and cortex in two. MRI abnormalities were usually associated with appropriate clinical deficits, but were larger and more disseminated than expected.

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Neurological involvement is observed in up to 33% of patients with Behçet's disease (BD) and classically has a poor prognosis.[1–5] The neurological manifestations are variable.[6,7] MRI has been said to be more sensitive than CT scan.[4,7–14] We prospectively studied 31 patients with BD and neurological signs using MRI, and correlated radiological abnormalities and neurological signs.

Patients and methods

From 1985 to 1990 MRI was used to study 31 patients with BD involving the nervous system. All the patients fulfilled the criteria for diagnosis of BD.[15] There were 25 men and six women, mean (SD) age 26 (7·6) at the onset of BD and 33 (7) years when they presented with neurological symptoms. The main features of BD are summarised in table 1. Patients were natives of North Africa (n = 17), Africa (n = 1), Antilles (n = 2), Europe (n = 10), Turkey (n = 1). We tested 28 patients for HLA B5 and it was positive in 10. Neurological involvement was the first manifestation of BD in three patients (cases 7,10,11) and led to diagnosis of BD in 15 cases.

MR scanning was performed on a 0·5 Tesla GE-CGR MR unit. Sagittal T1 weighted spin echo sequence (400/21/1-TR/TE/number of echoes) and axial T2 weighted spin echo sequence (2000–1800/40–60/2–3) were obtained. To detect cerebral venous thrombosis (CVT), frontal T2 weighted (180/60/3) slices were performed. Three successive echoes and a long TE were used in order to avoid flow related enhancement and to differentiate high signal due to thrombosis from even echo rephasing. Each MRI was read by a neuroradiologist (DD) and a neurologist (MV) unaware of the neurological symptoms. When available, CT scans were compared with the MRI scans. Diagnosis of CVT was confirmed on the absence or partial lack of filling of one or several sinuses on at least two projections of bilateral carotid angiography.

Table 1 Main features of Behçet's disease

Case	*Sex/ age**	*Diagnosis criteria*	*Pathergy test*	*Vascular thrombosis*	*Other features*
1	M/18	O-E	ND	Sural vein	Epididymitis
2	W/48	O-G	ND	—	Arthritis
3	M/22	O-S	+	Sural vein and femoral artery aneurysm	
4	M/32	O-G-E-S	ND	Sural vein	
5	M/22	O-G-S	+	Sural vein	
6	W/23	O-G	+	—	
7	W/33	O-E-S	+	—	
8	W/32	O-G-S	+	Sural vein Pulmonary artery	
9	M/27	O-G-S	+	—	
10	M/35	O-E-S	ND	Iliac vein	
11	M/27	O-G-S	ND	—	
12	M/35	O-S	+	—	
13	M/32	O-E-S	+	—	Arthritis
14	M/38	O-E-S	ND	—	Arthritis
15	M/20	O-G-E-S	ND	—	Arthritis
16	M/32	O-G-S	+	—	Arthritis
17	M/21	O-G-S	+	—	Arthritis
18	M/22	O-G-S	+	—	Arthritis
19	W/10	O-G-E	—	Sural vein	Arthritis
20	M/19	O-G-S	+	Femoral vein	
21	M/34	O-G-E-S	+	—	Arthritis
22	M/33	O-G-E-S	ND	—	Arthritis
23	M/26	O-G-E-S	ND	—	Arthritis
24	M/6	O-G-E	—	—	Epididymitis
25	M/27	O-G-E-S	ND	—	Arthritis
26	M/22	O-G-S	ND	Pulmonary artery	
27	M/31	O-G-E-S	ND	—	Arthritis
28	M/37	O-G-E-S	+	—	Arthritis
29	M/29	O-G-S	ND	Sural vein	
30	W/29	O-G-S	+	Brachial and Femoral veins	Arthritis
31	M/25	O-G-E-S	ND	Femoral vein	Arthritis

*Age at onset of disease; O = recurrent oral aphthae; G = genital aphthae; E = anterior or posterior uveitis or retinal vasculitis; S = erythema nodosum, pseudofolliculitis, papular or acneiform lesions; ND = not done.

Table 2 Main characteristics of cerebral venous thrombosis

Case	Symptoms	Angiography	MRI
1	IH	right TS	T1: not done T2: hypersignal right TS
2	IH Monoparesis	SSS right and left TS right jugular vein	T1: isosignal T2: hypersignal SSS right and left TS
3	IH	SSS and left TS	T1: isosignal T2: hypersignal SSS and left TS
4	IH	SSS and right TS	T1: isosignal T2: hypersignal SSS and right TS
5	IH	SSS	T1: isosignal T2: hypersignal SSS
6	IH	SSS and right TS	T1: not done T2: hypersignal SSS and right TS
7	IH Hemiparesis Seizures	right TS	T1: hypersignal right temporoparietal T2: hypersignal right temporoparietal and right TS
8	IH	sinus rectus	T1: isosignal T2: isosignal
9	IH	SSS, right and left TS	T1: normal T2: normal
10	IH	SSS	See table 3

IH = intracranial hypertension; T1 = T1 weighted slices; T2 = T2 weighted slices; SSS = superior sagittal sinus; TS = transverse sinus.

Results

We divided the patients into three groups: patients with CVT, patients with central nervous system (CNS) involvement, and patients with isolated headache.

PATIENTS WITH CVT (CASES 1–9 AND 20)

CVT was diagnosed in 10 patients including six reported in a long term follow up study.[16] Age at onset was 32·2 years [mean; standard deviation: 25·1–39·3]. Neurological signs are summarised in table 2. CVT was the presenting symptom of BD in one patient (case 7) and was followed by CNS involvement after three years in another (case 20). The onset was chronic (>1 month) in all but two patients. The most frequent feature was isolated intracranial hypertension (n = 8). It was associated with focal deficits (n = 2) and seizures (n = 1). CT scans were normal in five cases, showed a "delta sign" in three cases, and a cerebral infarct caused by venous occlusion in one case. The occlusion of one or several sinuses was demonstrated by cerebral angiography: superior sagittal sinus (n = 7), transverse sinus (n = 7), sinus rectus and part of Galen's vein (n = 1). MRI was performed 16 weeks after the first symptom of CVT (3–44) in eight cases (cases 1–8). An abnormal high signal at the level of the occluded sinus was observed on the T2 weighted sequences (fig 1). The thrombus was isodense to the grey matter on sagittal T1 weighted images. The comparison of abnormal angiogram and MRI scans in the patient presenting with CVT of the deep cerebral veinous thrombosis revealed the thrombus on the MRI images. In case 7, a hyperintense lesion surrounded by a rim of hypodensity was observed on both T1 and T2 weighted images and was compatible with haemorragic infarct of venous origin. This image was surrounded by a high signal area on T2 weighted sequences (fig 2). All the patients were treated with anticoagulants and corticosteroids. Neurological symptoms improved within four weeks. Control MRI was repeated 2–20 months later in five patients, who became asymptomatic under treatment. Total (case 6) or partial (cases 1–3) recanalisation was observed. In one case (case 7) the thrombus persisted as a hyperintense signal in the occluded sinus.

MRI was performed three and seven years after the first symptom of CVT in two cases (cases 9 and 20). Minor flow abnormalities were detected and were suggestive of partial recanalisation of the sinus.

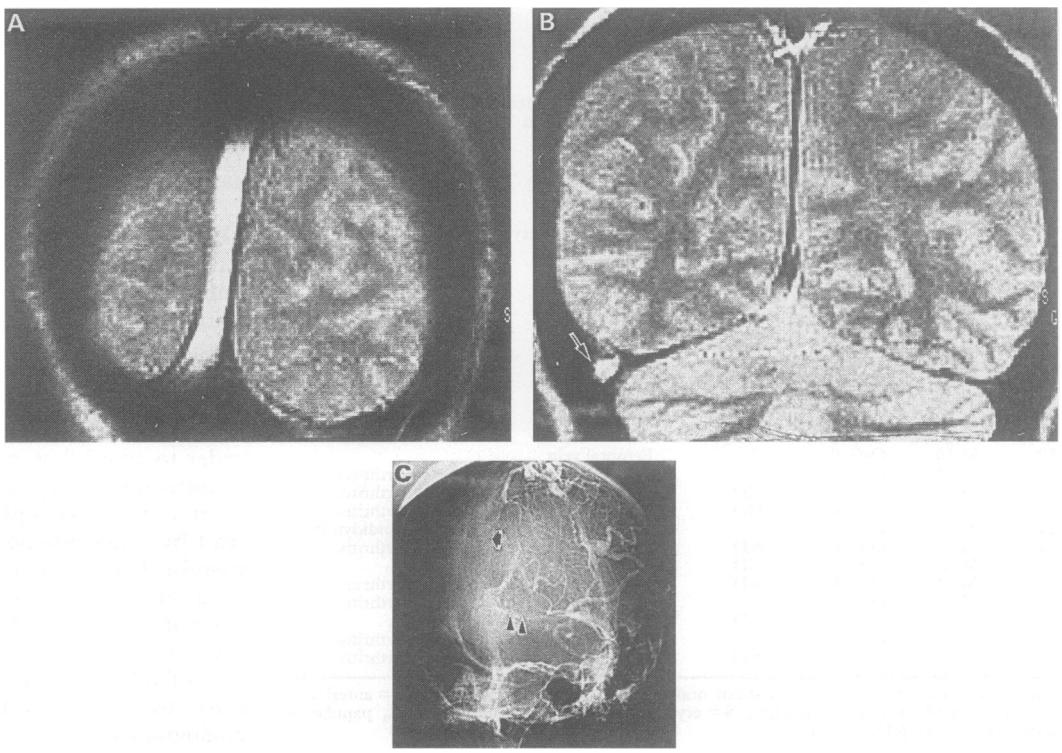

Figure 1 (case 2) Cerebral venous thrombosis with involvement of the superior sagittal sinus (SSS) and of the right lateral sinus (posterior part). 1 (a) Coronal slice through the posterior part of the SSS (T2 weighted image, TR = 1800, TE = 60, first echo), showing hypersignal of thrombosed sinus instead of normal flow void. 1 (b) Same echo, same sequence, coronal slice 3 cm more anterior showing occlusion of right lateral sinus (arrow); flow void is observed in the SSS which was circulating at this level (more anterior than (a)). 1 (c) Left carotid angiogram (oblique view) showing occlusion of posterior part of SSS (broad arrow) and of right lateral sinus (small arrow) and partial occlusion of left lateral sinus (arrowhead).

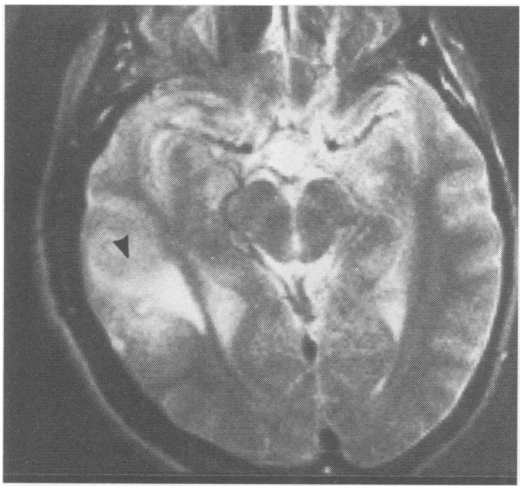

Figure 2 Venous infarction in right temporal lobe associated with right lateral sinus thrombosis (case 7). Axial T2 weighted image (TR = 2000, TE = 40) shows a high signal intensity in the temporal lobe (arrow).

PATIENTS WITH CNS INVOLVEMENT (CASES 10–24)

CNS involvement was diagnosed in 15 patients. The neurological features are summarised in table 3. Age at onset of neurological symptoms was 33·5 years [mean; standard deviation: 27·1–39·9]. In all patients funduscopy was normal. CSF characteristics were: proteins 50 mg/dl (46–91), lymphocytes 112 × 10^6/l (5–295). Cerebral angiography, performed in five cases, was normal. CT scan was normal (n = 7), showed mild cerebral atrophy (n = 1), focal hypodensity in the temporal lobe (n = 1), or in the internal capsule (n = 2).

MRI was performed during the acute illness and was abnormal in 13/14 cases. In seven patients T1 weighted sequence MRIs were considered to be normal or showed mild atrophy (n = 1). Nevertheless T2 weighted sequences demonstrated lesions in all the cases but one. They appeared as hyperintense areas and were usually small (<5 mm), scattered, confluent particularly in white matter (figs 3, 4, and 5). MRI lesions were distributed as follows: hemispheric white matter in nine patients (70%) (internal capsule n = 6, corona radiata n = 4, periventricular white matter n = 4), brainstem in eight patients (60%) (midbrain n = 5, pons n = 8, medulla n = 1), basal ganglia and thalamus in five patients (40%), cortex in two patients. Hemispheric involvement was isolated in only four cases. There was no predilection for the periventricular regions and linear periventricular hyperintensities (cases 11, 16, 23, 24) were always associated with another localisation (table 3). Brainstem lesions always involved the pons (figs 6 and 7). They were isolated in one case and associated with hemispheric lesions in seven cases. Both confluent and punctiform lesions were observed irrespective of vascular territories.

MRI abnormalities were usually associated with appropriate clinical deficits (table 3) but they were larger and more disseminated than expected. Cerebellar signs were related to lesions of the afferent or efferent pathways and the cerebellum itself was spared. Lesions of the brainstem were consistently associated with severe neurological impairment such as tetraparesis, pseudobulbar palsy, and bilateral cerebellar signs. Isolated hemispheric involvement was associated in two cases with contralateral hemiparesis or monoparesis. When lesions were bilateral, tetraparesis and pseudobulbar palsy were also observed. In three patients the neurological symptoms could not be related to abnormalities on the MRI. In case 19 the MRI was thought to be normal but the scan was of poor quality because of the patient's excessive movements. A control MRI was obtained (cases 15, 20) at four months and four years after onset of the

Table 3 Main characteristics of central nervous system involvement

Case	*Clinical features*	*MRI*	*Delay*
10	Headache, R hemiparesis	Normal	13 years
11	Dementia, tetraparesis Cerebellar alaxia, pseudobulbar palsy	T1: diffuse atrophy T2: HS periventricular + bilateral internal capsule	3 years
12	Headache, R hemiparesis	T1: normal T2: HS left internal capsule + cerebral peduncle + pons	6 weeks
13	R lower limb monoplegia	T1: normal T2: left corona radiata	2 months
14	Headache, L hemiparesis	T1: hyposignal pons T2: HS R thalamus and cerebral peduncle + pons	2 months
15	Headache, tetraparesis pseudobulbar palsy	T1: hyposignal pons + midbrain T2: HS L internal capsule + midbrain + pons	1 month
16	Headache, tetraparesis cerebellar ataxia	T1: normal T2: HS periventricular + corona radiata + R internal capsule and basal ganglia	3 years
17	Headache, L hemiparesis cerebellar ataxia, R III palsy	T1: not done T2: diffuse atrophy	10 months
18	Dementia, tetraparesis, pseudobulbar palsy bilateral cerebellar syndrome	T1: hyposignal pons T2: HS L internal capsule + basal ganglia + pons	6 months
19	Headache, tetraparesis, pseudobulbar palsy dementia, bilateral cerebellar syndrome	Normal	11 months
20	Headache, tetraparesis, pseudobulbar palsy bilateral cerebellar syndrome R III+ VII + L VI palsy	T1: normal T2: HS L basal ganglia + midbrain + pons	6 weeks
21	Headache, R hemiparesis, L VI palsy	T1: normal T2: HS R cerebral peduncle + pons + medulla	4 months
22	Headache, L hemiparesis cerebellar ataxia	T1: hyposignal pons T2: HS bilateral corona radiata + pons	18 months
23	Headache, tetraparesis L cerebellar syndrome	T1: hyposignal bilateral corona radiata T2: HS bilateral corona radiata + parietal cortex + L internal capsule + basal ganglia + periventricular	2 years
24	Headache, L Hemiparesis	T1: normal T2: HS periventricular + bilateral parietal cortex	7 months

R = right; L = left; HS = hypersignal; T1 = T1 weighted slices; T2 = T2 weighted slices.
*Patient with a history of CVT 3 years before (superior sagittal and transverse sinus are normal); delay = delay between neurological symptoms and performance of MRI

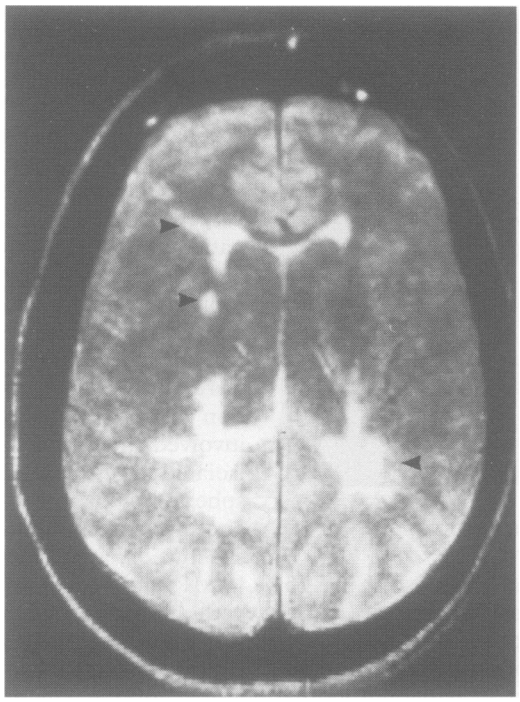

Figure 3 CNS involvement (case 16). Axial T2 weighted image (TR = 2000, TE = 40) shows foci of high signal intensity in the white matter (arrows).

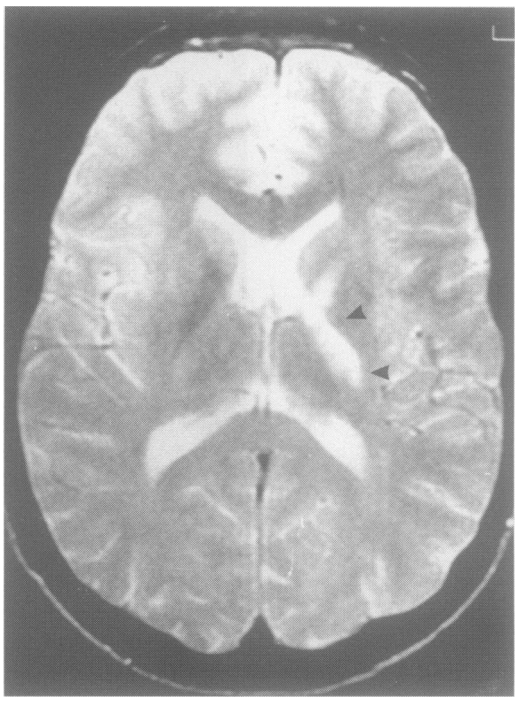

Figure 5 CNS involvement (case 18). Axial T2 weighted image (TR = 2000, TE = 40) shows high signal intensity in the left pyramidal tract (arrows).

treatment. No change of the lesions was observed.

In a patient seen 13 years after the onset of the neurological symptoms (case 10), the MRI scan was normal.

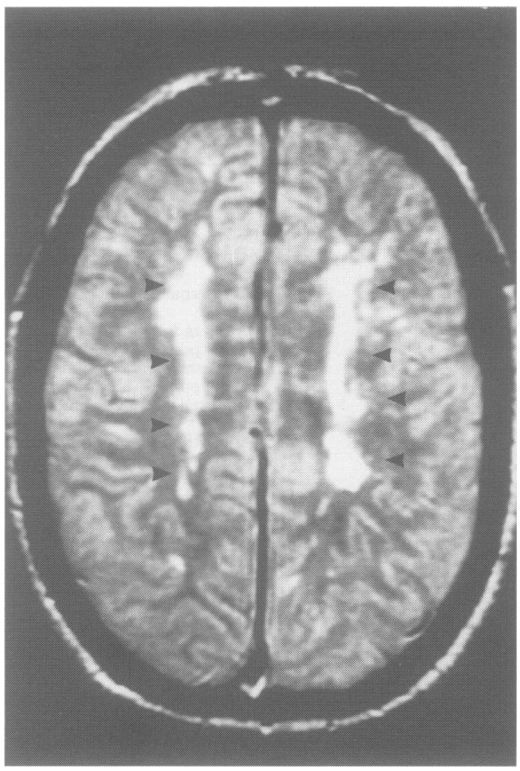

Figure 4 CNS involvement (case 23). Axial T2 weighted image (TR = 2000, TE = 40) shows multiple bilateral foci of high signal intensity involving the periventricular white matter (arrowheads).

PATIENTS WITH ISOLATED HEADACHE (CASES 25–31)

Isolated headache, without neurological symptoms or papilloedema, was observed in seven patients. Three had CSF pleiocytosis (lymphocytes 28 × 10^6/l 10–60): CT scan and MRI were normal. Four patients presented with chronic intermittent headache for 10 months to five years. MRI was normal in three. In one patient, a small hyperintense signal was noticed in the left frontal lobe but no neurological symptom appeared during one year of follow up.

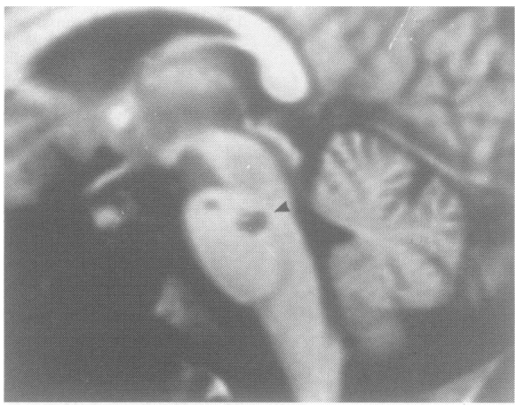

Figure 6 CNS involvement (case 14), sagittal T1 weighted image (TR = 540, TE = 10) shows a large hyposignal in the pons (arrow).

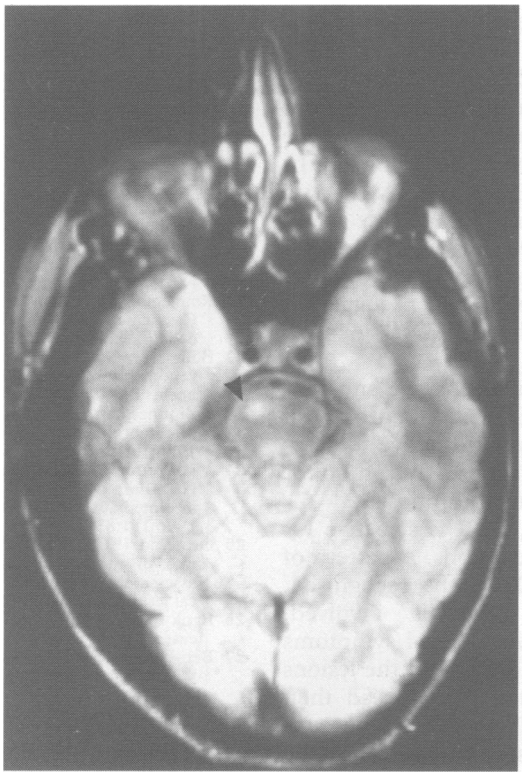

Figure 7 CNS involvement (case 20). Axial T2 weighted image (TR = 2000, TE = 40) shows high signal intensity in the right part of the pons (arrow).

Discussion

BD, originally described as a syndrome of aphthous stomatitis, genital ulceration, and uveitis, is now recognised as a systemic vasculitis.[17 18] In retrospective studies neurological involvement occurs in 10–50% of cases.[5 17] In a recent Turkish prospective study, Serdaroglu *et al* estimate the incidence at 5·3%.[19] It is the presenting symptom in up to 5% of cases.[20 21] Neurological manifestations of BD can be divided into three categories[21]: CVT, CNS involvement, and isolated headache.

CVT is observed in 35% of patients with neurological manifestations.[5] Conventional angiography is still required for diagnosis[22] but MRI has been shown to be a safe and reliable technique in the diagnosis of dural sinus thrombosis.[4 23–25] In this series clinical features and imaging are similar to those of patients with CVT of other origin.[22] Isolated IH is the usual presentation. Occlusion of several sinuses (including the SSS) is frequent.[14 19 21 26–31] MRI shows direct signs of thrombosis. On T1 weighted slices hypersignals of the occluded sinus have been reported during the first 15 days of evolution.[23 24] On T2 weighted sequences, abnormal high signal intensity within the affected dural sinuses is observed a few weeks after thrombosis. In pure cortical venous thrombosis or deep cerebral venous thrombosis, the use of contrast agents should be emphasised. More recently, MR angiography has proved helpful in identifying thrombosis with a good correlation with conventional angiography.[32] Follow up with MRI can demonstrate recanalisation of the occluded sinus and can be used to monitor anticoagulant therapy.[33]

CNS involvement is a severe and devastating manifestation of BD. CT scan may be abnormal in patients with neurological involvement from BD[20 34–36] but fails to demonstrate small infarcts.[37] Indeed, MRI is much more sensitive than CT scan.[4 21] When CT scan is normal despite neurological symptoms MRI can reveal abnormal signals. When lesions are seen on CT scan, they are larger and more widespread on MRI.[7 9 38–40] The results of our study are consistent with a pattern suggestive of BD.

1 Lesions are usually multiple small foci of high intensity on T2 weighted sequences. They are usually extensive, confluent and distributed over white matter without predilection for the periventricular regions.[41]

2 The lesions are within the white matter (70%), the brainstem (60%), the basal ganglia and the thalamus (40%).

3 Isolated lesions of the cerebral hemispheres can be observed.

4 Lesions of the brainstem are rarely isolated and usually involve the pons.

5 Neurological symptoms are generally correlated with radiological findings as previously reported.[21] Hypointensity on T1 and hyperintensity on T2 weighted images can be observed on MRI. When CT scan is normal despite neurological symptoms, MRI can reveal abnormal signals; however, the lesions are more extensive than clinically expected. MRI lesions occur with the same anatomical distribution as the pathological lesions related to vasculitis.[9] This vasculitis consists of disseminated meningoencephalitis with perivascular cell infiltration, infarction with small necrotic areas surrounding blood vessels, haemorrhage, loss of myelinated fibres, and gliosis.[9 12 37 42] However, the MRI findings are not specific to BD and can be observed in other vasculitis of the CNS such as systemic lupus erythematosus[12]; but involvement of the brainstem is rarely observed in systemic lupus erythematosus.[43 44] BD is also considered in the differential diagnosis of multiple sclerosis (MS) when neurological features are seen in isolation.[12] The most striking MRI findings are multiple irregular and extensive periventricular lesions.[25 45 46] Nevertheless to differentiate MS from BD on the basis of MRI is difficult. In our experience some features are in favour of BD: MR involvement of basal ganglia and thalamus; the absence of periventricular predominance of the white matter lesions; and the involvement of the pons where hypersignals are mostly observed in the centre part. This is the opposite to MS, where lesions of the brainstem involve the floor of the fourth ventricle and the middle cerebellar peduncles. It must be emphasised that both diseases are diagnosed on a clinical basis. In vasculitis the periventricular changes are often rather mild, hemisphere lesions can be disclosed without periventricular changes, and involvement of grey matter is frequent.[12]

Clinical and radiological correlations have been shown[41] and resolution of abnormal signals on MRI after treatment has been demonstrated and correlated with clinical improvement.[7 11 21 43 47] It has been postulated

that at an early stage of the disease, reversibility of MR lesions in BD may reflect a reversible breakdown in the blood-brain barrier rather than gliosis or infarction.[47] We did not observe such imaging reversibility on the two MRIs that we performed on follow up. In patients with isolated headache, MRI was normal in all cases but one. In one case a very small lesion was noticed on MRI and was clinically asymptomatic on follow up without therapy. Nevertheless, it has been suggested that headache[17] could herald CNS involvement.

In conclusion, MRI is a sensitive and reliable procedure in BD with neurological involvement. It can be an alternative to cerebral angiography to diagnose and monitor therapy of cerebral venous thrombosis.

In CNS involvement, MRI is sensitive for the early detection of the lesions. A pattern of lesions evocative of BD can be recognised, especially when the pons is involved. Correlations between neurological symptoms and MRI lesions can be drawn but the lesions are often larger and more widespread than clinically expected. Further studies with consecutive MRI are necessary to appreciate the effects of treatment on abnormal imaging.

In case of isolated headache, MR helps to rule out neurological involvement, but the abnormalities can be seen in asymptomatic patients.

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1 Kawakita H, Nishimura M, Satoh Y, Shibata N. Neurological aspects of Behçet's disease. *Jpn J Neurol Sci* 1967;5:417–39.
2 Lakhanpal S, Tani K, Lie JT, Katoh K, Ishigatsubo Y, Ohokubo T. Pathologic features of Behçet's syndrome. *Human Pathol* 1985;16:790–5.
3 Schotland DL, Wolf SM, White HH, Dubin HV. Neurologic aspects of Behçet's disease. *Am J Med* 1963;34:544–53.
4 Willeit J, Schmutzhard E, Aichner F, Mayr U, Weber F, Gerstenbrand F. CT and MR imaging in neuro-Behçet disease. *J Comput Assist Tomogr* 1986;10:313–5.
5 Wolf SM, Schotland DL, Phillips LL. Involvement of nervous system in Behçet's syndrome. *Arch Neurol* 1965; 12:315–25.
6 Chajek T, Fainaru M. Behçet's disease. Report of 41 cases and a review of the literature. *Medicine* 1975;54: 179–95.
7 Patel DV, Neuman MJ, Hier DB. Reversibility of CT and MR findings in neuro-Behçet disease. *J Comput Assist Tomogr* 1989;13:669–73.
8 Besana C, Comi G, Del Maschio A, *et al.* Electrophysiological and MRI evaluation of neurological involvement in Behçet's disease. *J Neurol Neurosurg Psychiatry* 1989;52:749–54.
9 Fukuyama H, Kameyama M, Nabatame H, *et al.* Magnetic resonance images of neuro-Behçet syndrome show precise brain stem lesions. *Acta Neurol Scand* 1987;75:70–3.
10 Kataoka S, Hirose G, Tsukada K. Brain stem type neuro-Behçet's syndrome. *Neuroradiology* 1989;31:258–62.
11 Kermode AG, Plant GT, Mac Manus DG, Kendall BE, Kingsley DPE, Moseley IF. Behçet's disease with slowly enlarging midbrain mass on MRI: Resolution following steroid therapy. *Neurology* 1989;39:1251–2.
12 Miller DH, Ormerod IEC, Gibson A, Du Boulay EPG, Rudge P, MacDonald WI. MR brain scanning in patients with vasculitis: differentiation from multiple sclerosis. *Neuroradiology* 1987;29:226–31.
13 Shakir RA, Sulaiman K, Kahn RA, Rudwan M. Neurological presentations of neuro-Behçet's syndrome: clinical categories. *Eur Neurol* 1990;30:249–53.
14 Snyder TC, Sachdev HS. MR imaging of cerebral dural sinus thrombosis. *J Comput Assist Tomogr* 1986;10: 889–91.
15 International Study Group for Behçet's Disease. Criteria for diagnosis of Behçet's disease. *Lancet* 1990;335: 1078–80.
16 Wechsler B, Vidailhet M, Piette JC, *et al.* Cerebral venous thrombosis in Behçet's disease: clinical study and long term follow up of 25 cases. *Neurology* 1992;42:614–8.
17 O'Duffy JD, Goldstein NP. Neurologic involvement in seven patients with Behçet's disease. *Am J Med* 1976; 61:170–8.
18 Shimizu T, Ehrlich GE, Inaba G, Hayashi K. Behçet disease. *Semin Arthritis Rheum* 1979;8:223–60.
19 Serdaroglu P, Yazici H, Ozdemir C, Yurdakul S, Bahar S, Atkin E. Neurologic involvement in Behçet's syndrome. *Arch Neurol* 1989;46:266–9.
20 Kozin F, Haughton V, Bernhard GC. Neuro-Behçet disease: two cases and neuroradiologic findings. *Neuroradiology* 1977;27:1148–52.
21 Rougemont D, Bousser MG, Wechsler B, Blétry O, Castaigne P, Godeau P. Manifestations neurologiques de la maladie de Behçet. *Rev Neurol* 1982;138:496–505.
22 Bousser MG, Chiras J, Bories J, Castaigne P. Cerebral venous thrombosis. *Stroke* 1985;16:199–213.
23 Macchi PJ, Grossman RI, Gomori JM, Goldberg HI, Zimmerman RA, Bilaniuk LT. High field MR imaging of cerebral venous thrombosis. *J Comput Assist Tomogr* 1986;10:10–5.
24 Mac Murdo SK, Brant-Zawadzki M, Bradley WG, Chang GY, Berg BO. Dural sinus thrombosis: study using intermediate field strength MR imaging. *Radiology* 1986;161:83–6.
25 Ormerod IEC, Miller DH, Mac Donald WI, *et al.* The role of nuclear magnetic resonance imaging in the assessment of multiple sclerosis and isolated neurological lesions. *Brain* 1987;110:1579–616.
26 Bank I, Weart C. Dural sinus thrombosis in Behçet's disease. *Arthritis Rheum* 1984;27:816–8.
27 Ben-Itzhak J, Keren S, Simon J. Intracranial venous thrombosis in Behçet's syndrome. *Neuroradiology* 1985; 27:450.
28 Brissaud P, Laroche L, de Gramont A, Krulik M. Digital angiography for the diagnosis of dural sinus thrombosis in Behçet's disease. *Arthritis Rheum* 1985;28:359–60.
29 Feuilhade C, Touboul JL, Wechsler B, Guillard A. Thrombophlébite cérébrale au cours de la maladie de Behçet. *Presse Med* 1982;11:3351.
30 Harper CM, O'Neill BP, O'Duffy JD, Forbes GS. Intracranial hypertension in Behçet's disease: demonstration of sinus occlusion with use of digital subtraction angiography. *Mayo Clin Proc* 1985;60:419–22.
31 Masheter HC. Behçet's syndrome complicated by intracranial thrombophlebitis. *Proc Roy Soc Med* 1959; 52:1039–40.
32 Payadachee TS, Bingham JB, Graves MJ, *et al.* Dural sinus thrombosis: diagnosis and follow-up by magnetic resonance, angiography and imaging. *Neuroradiology* 1991;33:165–7.
33 Einhaeupl KM, Villringer A, Meister W, *et al.* High dose heparin treatment in cerebral sinus venous thrombosis. *Lancet* 1991;338:597–600.
34 Dobkin BH. Computerized tomographic findings in neuro-Behçet's disease. *Arch Neurol* 1980;37:58–9.
35 Herskovitz S, Lipton RB, Lantos G. Neuro-Behçet's disease: CT and clinical correlates. *Neurology* 1988;38: 1714–20.
36 Litvan I, Roig C, Rovira A, Ruscalleda J. Behçet's syndrome masquerading as tumor. *Neuroradiology* 1987; 29:103.
37 Kuroiwa Y, Tohgi H, Kanayama H, Fujimori M, Aoki H. Neuro-Behçet's disease with alternating hemiparesis. *Neuroradiology* 1986;28:284.
38 Vidaller A, Carratala J, Moreno R, Arbizu T, Rubio F. Magnetic resonance imaging in neuro-Behçet's disease. *Br J Rheum* 1987;27:79–80.
39 Wechsler B, Dormont D, Lubetski C, *et al.* Magnetic resonance imaging in Behçet's disease. In: JD O'Duffy, E Kokmen, eds. *Behçet's disease, basic and clinical aspects.* New York: Marcel Dekker, 1991:331–4.
40 Zuheir Al Kawi M, Bohlega S, Banna M. MRI findings in neuro-Behçet's disease. *Neurology* 1991;41:405–8.
41 Banna M, El Ramahl K. Neurologic involvement in Behçet disease: imaging findings in 16 patients. *Am J Neuroradiol* 1991;2:791–6.
42 Iseki E, Iwabuchi K, Yagishita S, Amano N, Matsushita M. Two necropsy cases of chronic encephalomyelitis: variants of neuro-Behçet's disease? *J Neurol Neurosurg Psychiatry* 1988;51:1084–7.
43 Mc Abee GN, Barasch ES. Resolving MRI lesions in lupus erythematosus selectively involving the brain stem. *Pediatr Neurol* 1990;6:186–9.
44 Suzuki K, Hara M, Nakajima S, *et al.* Analysis of systemic lupus erythematosus (SLE) involving the central nervous system by magnetic resonance imaging (MRI). *Ryumachi* 1989;29:88–96.
45 Paty DW, Oger JJ, Kastrukoff LF, *et al.* Nuclear magnetic resonance imaging in the diagnosis of multiple sclerosis. *Neurology* 1988;38:180–5.
46 Steward JM, Houser OW, Baker HL, O'Brien PC, Rodriguez M. Magnetic resonance imaging and clinical relationships in multiple sclerosis. *Mayo Clin Proc* 1987; 62:174–84.
47 Pamir MN, Kansu T, Erbengi A, Zileli T. Papilledema in Behçet's syndrome. *Arch Neurol* 1981;38:643–5.