



ORIGINAL ARTICLE

Role of magnetic resonance venography in evaluation of cerebral veins and sinuses occlusion

Eman Abd-El Latif Abd-Elaziz El Damarawy ^{a,*},
Abd-El Aziz Mohamed El-Nekiedy ^a, Alaa Mohamed Fathi ^a,
Alaa El-Dein Mohamed Eissa ^b, Reda Mohamed Darweesh ^a

^a Radiodiagnosis Department, Alexandria Faculty of Medicine, Alexandria University, Egypt

^b Neurosurgery, Alexandria Faculty of Medicine, Alexandria University, Egypt

Received 13 July 2011; accepted 5 October 2011

Available online 14 December 2011

KEYWORDS

MRI;
MRV;
Cerebral venous thrombosis

Abstract *Introduction:* Cerebral venous occlusion (CVO) means intraluminal obstruction by cerebral venous thrombosis (CVT) or external compression. The differential diagnosis is broad including stroke, brain capillary telangiectasia, contusion, hypertensive hemorrhage, metastases and venous vascular malformations. MRI in conjunction with MRV is considered the modality of choice in diagnosis of CVO.

Objective: To assess the role of magnetic resonance venography in the evaluation of cerebral veins and sinuses occlusion.

Methods: The study was conducted on 15 patients with cerebral venous occlusion and subjected to MRI and MRV.

Abbreviations: MRI, magnetic resonance imaging; MRV, magnetic resonance venography; SSS, superior sagittal sinus; CVO, cerebral venous occlusion; CVT, cerebral venous thrombosis; DSA, digital subtraction angiography; CT, computed tomography; CTV, computed tomography venography; CVST, cerebral venous sinus thrombosis.

* Corresponding author. Present/permanent address: Faculty of Medicine, Alexandria University, Egypt.

E-mail address: mona_zawawy77@yahoo.com (E.A.-E.L.A.-E.E. Damarawy).

2090-5068 © 2011 Alexandria University Faculty of Medicine. Production and hosting by Elsevier B.V. All rights reserved.

Peer review under responsibility of Alexandria University Faculty of Medicine.

doi:10.1016/j.ajme.2011.10.001



Production and hosting by Elsevier

Results: The study included 13 patients with cerebral venous occlusion due to thrombosis and two patients with occlusion by tumors (meningioma).

Conclusion: MRI in conjunction with MRV is considered an accurate, safe, non-invasive, non-ionizing diagnostic modality in assessing CVO. It can assess parenchymal lesions and detect intraluminal small thrombi and its extension.

© 2011 Alexandria University Faculty of Medicine. Production and hosting by Elsevier B.V. All rights reserved.

1. Introduction

Cerebral venous thrombosis or occlusion by extrinsic compression that subsequently progresses to complete occlusion is an elusive diagnosis because of its non-specific presentation and its numerous predisposing causes.¹ Cerebral (dural) venous sinus thrombosis is an uncommon condition but become more common than previously thought, its clinical presentation is varied and often dramatic and may occur with headache and cranial nerve palsies.² It often affects young-to-middle-aged patients, and more commonly women. It is an uncommon cause of cerebral infarction relative to arterial disease but is an important consideration because of its potential morbidity.³

The most common causes and risk factors associated with CVT are due to: genetic prothrombotic conditions: antithrombin deficiency, protein C and protein S deficiency. Acquired prothrombotic states: nephrotic syndrome, pregnancy, and puerperium.⁴ Infections: otitis, mastoiditis, sinusitis and meningitis. Collagen-vascular diseases: systemic lupus erythematosus, sarcoidosis and Behçet syndrome.⁵ Hematologic conditions: polycythemia, leukemia, anemia and sickle cell disease. Drugs: oral contraceptives and tamoxifen.⁶ Mechanical causes: trauma and neurosurgical procedures and miscellaneous causes: dehydration and cancer.⁷

The imaging characteristics of MRI include: (1) Imaging of the brain parenchyma that appears in the form of non-specific lesions, such as intracerebral hemorrhages or infarcts, edema, isolated or associated with infarcts or hemorrhages, and it can even be normal in up to 30% of patients. (2) Imaging of the cerebral venous system (veins and sinuses thrombus) by direct visualization of a thrombus within the vessel.⁸ MRV features include non-visualization of the vessel (i.e. no flow), flow defect and presence of collaterals at the site of occlusion.⁹

2. Methods

Twelve of the 15 patients with known or suspected CVO were subjected to high field MRI of the brain (1.5 T) by using closed MRI unit (Philips and GE) and three patients by using a low field magnet (0.2 T) MRI unit by using open MRI unit (Hitachi) medical system.

The following protocols were applied: axial T1WI, T2WI and flair sequences, sagittal T1WI sequence, coronal T2WI sequence and MRV for all patients using three dimensional-phase contrast technique (3D-PC).

The following parameters were utilized for closed MRI (1.5 T):

Slice thickness: 6 mm, interslice gap: 0.6 mm, field of view: 230 mm², rectangular field of view (RFOV): 80%, number of signals averaged (NSA): 2, for T1W sequences: repetition time (TR) = 600 ms; echo time (TE) = 15 ms, for T2W sequences:

TR = 5000 ms; TE = 120 ms, for FLAIR: TR = 6000 ms; TE = 120 ms.

The MRV phase contrast parameters were: TR = 500 ms, TE = 50 ms, FOV = 230 mm² and velocity = 15 cm/s.

The following parameters were utilized for open MRI unit (0.2 T):

Slice thickness: 6 mm, interslice gap: 0.6 mm, field of view: 230 mm², rectangular field of view (RFOV): 100%, number of signals averaged (NSA): 3, T1W sequences: repetition time (TR) = 400 ms; echo time (TE) = 17 ms, T2W sequences: TR = 4000 ms; TE = 120 ms and FLAIR: TR = 9000 ms; TE = 140 ms.

The MRV phase contrast parameters were: TR = 40 ms, TE = 12 ms, FOV = 240 mm² and velocity = 6 cm/s.

2.1. Statistical method

Statistical analysis was done using numbers and percentages for descriptive purposes.

3. Results

This study included 15 patients with age range from 40 days to 84 years old as mentioned in (Table 1) and mean age of 33.50 years. Ten patients (66.7%) were females and five patients (33.3%) were males. The most common and earliest clinical presentation in this study was headache followed by convulsion, disturbed consciousness, coma, weakness of one limb, blurring of vision and papilledema. Among the predisposing factors for CVO were post partum complications (four patients – 26.7%), followed by dehydration and post surgery complications, (both included three patients – 20%); however, there was no obvious predisposing factor in five patients (33.3%). The most common cause of cerebral venous occlusion in this study was venous thrombosis, was detected in 13 patients (86.7%) followed by compression from outside by tumor (meningioma) (Fig. 1), that was detected in two patients (13.3%) (Table 2). MRI features in our study were observed in the form of parenchymal changes and abnormal signals in thrombosed veins and sinuses in both T1 and T2, in addition to blooming of them. Parenchymal changes in our study were due to thrombosis in 12 patients and tumor in two patients. There was one patient of thrombosed cases reported no parenchymal changes (Fig. 2). These changes were seen in the form of non-hemorrhagic infarction in six patients (40%), hemorrhagic infarction in four patients (26.7%) and edema in seven patients (46.7%) (Table 3). The commonest MRI findings were replacement of the signal void of sinuses or veins by abnormal signal intensity. It showed three patterns, the first was hyper intense in both T1 and T2 in six patients (46.2%) (i.e. late sub-acute stage) (Fig. 2A-C). In two patients (15.4%), it was hyper

intense in T1 and iso to hypo intense in T2 (i.e. early subacute stage) (Fig. 3A and C) while in four patients (26.7%), it was hypo intense in both T1 and T2 (i.e. chronic stage) (Fig. 4A and B). Finally, there was one patient with equivocal signal in both T1 and T2 (Table 4) but was diagnosed by MRV. The main MRV findings in our study were non-visualization of occluded veins or sinuses due to absent signal, flow defect and presence of collaterals that may form in the dura that surrounds the occluded sinus. These collaterals were noted in three patients in this study (23.1%) (Fig. 4C). MRV successfully diagnosed all cases. The most common non-visualized sinus in this study was SSS that was noted in 86.7%, followed by transverse sinus 73.3%, sigmoid sinus 40% and straight sinus 33.3%. More than one occluded sinus could be found in some patients. Both cortical and deep veins could not be visualized in 33.3% and torcular herophili was included in 13.3% (Fig. 4E and F).

4. Discussion

The diagnosis of CVT is as difficult as its clinical manifestations are non-specific and may be subtle. Several factors combine to create an extremely elusive diagnosis of CVT: wide spectrum of clinical presentations, various predisposing factors, and the fact that it affects individuals of all age groups.¹⁰ CVT may mimic various medical conditions as arterial stroke, tumor, encephalitis, abscess and idiopathic intracranial hypertension (pseudo tumor cerebri).¹¹ So it is a diagnosis of exclusion and imaging investigation. Although most different modalities as DSA, CT, CTV and others help in diagnosis of cerebral venous occlusion (CVO) but each one has specific side effects and some have normal findings.¹² Most of patients in this study were in the age groups of < 1 year and 30–60 years. This agreed with similar literature reported that the most patients with this disease present in the neonates and young-to-middle-aged patients.³ Other literature reported that it is most common in the third decade while it may occur in all age groups.¹³ A Canadian study was done on children in 2001 reported that CVST occurs in 10% in the first year of life and this is a lower percentage in comparison with the percentage in our study that reported three patients (20%) in the first year of life.⁷

Ten patients in the current study were females (66.7%) and five patients were males (33.3%). This agreed with Einhäupl et al.¹³ that reported 75% of patients were females and had been suggested that the use of oral contraceptives in women is behind the disparity between the sexes. According to the

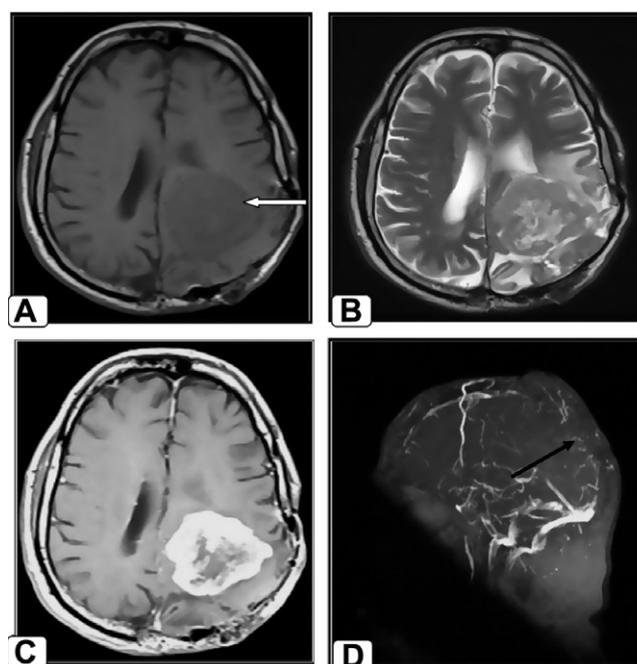


Figure 1 Follow-up of 56-year-old male patient with history of recurrent meningioma after surgery. MRI shows an abnormal signal intensity space occupying lesion (SOL) at the left parietal region (white arrow), surrounded by perifocal edema and exerting mass effect in the form of compression upon the left lateral ventricle. The tumor displays low signal intensity at axial T1WI (A), heterogeneous signal intensity at T2WI (B) with central area of breakdown displaying high signal intensity. Axial post contrast T1WI (C) shows intense enhancement of the tumor sparing the central area of breakdown. Sagittal MRV MIP (D) shows no flow signal at the posterior 2/3 of SSS (black arrow) denoting its compression by the tumor.

Table 2 Distribution of patients according to the causes of cerebral venous occlusion (CVO).

Cause	No.	%
Occlusion by thrombosis	13	86.7
Occlusion by tumor compression	2	13.3

complaints of patients presented with CVT or cerebral venous compression, the current study had shown that headache was the most frequent and often the earliest symptom noted in 10 patients (66.7%). This was in agreement with previous studies of Ameri and Bousser¹⁴ who reported that 75% of the 110 cases complaint of headache. The most common predisposing factor was puerperium and this is agreed with Nagaraj et al.¹⁵ who had found that 200 out of 230 cases (86%) of CVT, seen over eight years, were puerperal in nature. The most common cause of cerebral venous occlusion in the current study was intraluminal thrombus, it occurred in 13 patients (86.7%) and this agreed with Bousser et al.,¹⁶ who reported that all patients in his study of 110 cases had cerebral venous occlusion due to thrombosis.¹⁶ Occlusion by external compression was seen in two patients (13.3%) and was caused by meningioma and this is different from Jeffrey et al.¹⁷ who reported in his study that compression or invasion of cerebral sinuses from

Table 1 Distribution of patients according to age and sex.

Age in years	Male		Female		Total	
	n	%	n	%	n	%
< 1 year	2	13.3	1	6.7	3	20
1 to < 10	–	–	–	–	–	–
10 to < 20	–	–	–	–	–	–
20 to < 30	–	–	2	13.3	2	13.3
30 to < 40	1	6.7	2	13.3	3	20
40 to < 50	–	–	3	20	3	20
50 to < 60	1	6.7	2	13.3	3	20
60 or more	1	6.7	–	–	1	6.7
Total	5	33.3	10	66.7	15	100

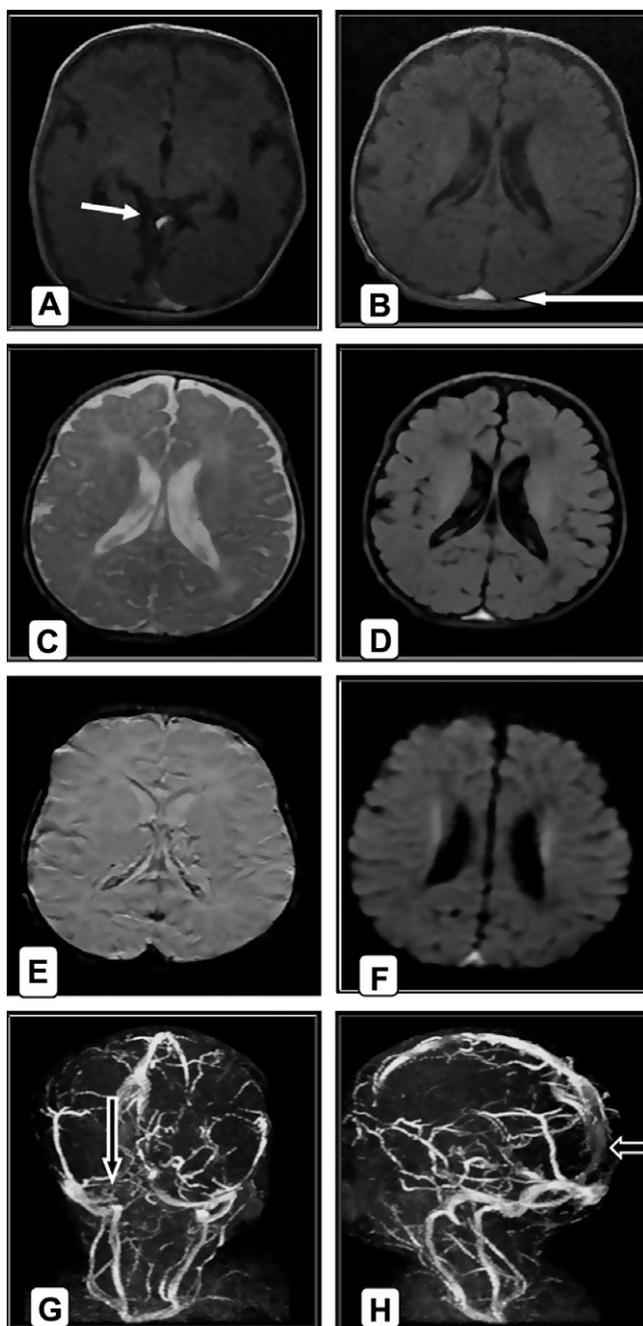


Figure 2 Female patient aged 40 years old complaint of convulsion, (A and B) axial T1WI shows hyper intense thrombus of both veins of Galen and SSS (white arrows) with no parenchymal changes. (C and D) Axial T2 and FLAIR the thrombus appears hyper intense. (E) Axial SWI shows hypo intense thrombus of SSS with blooming of the sinus. (F) Axial DWI the thrombus appears hyper intense. (G and H) Coronal and sagittal MRV MIP show no flow signal in RT transverse sinus and posterior aspect of SSS (black arrows) proved their occlusion.

dural or calvarial metastases was the main cause in those patients with cerebral sinuses occlusion due to solid tumors. Edema was the most common parenchymal change found in the present study, seen in (46.7%) of cases but Simonds et al.¹⁸ observed focal edema in 25% of cases, followed by

Table 3 Distribution of patients according to MR appearance of parenchymal changes.

MR appearance of parenchymal findings	No. of patients	%
Non-hemorrhagic infarction	6	40
Hemorrhagic infarction	4	26.7
Edema	7	46.7

More than one feature may be present in one patient.

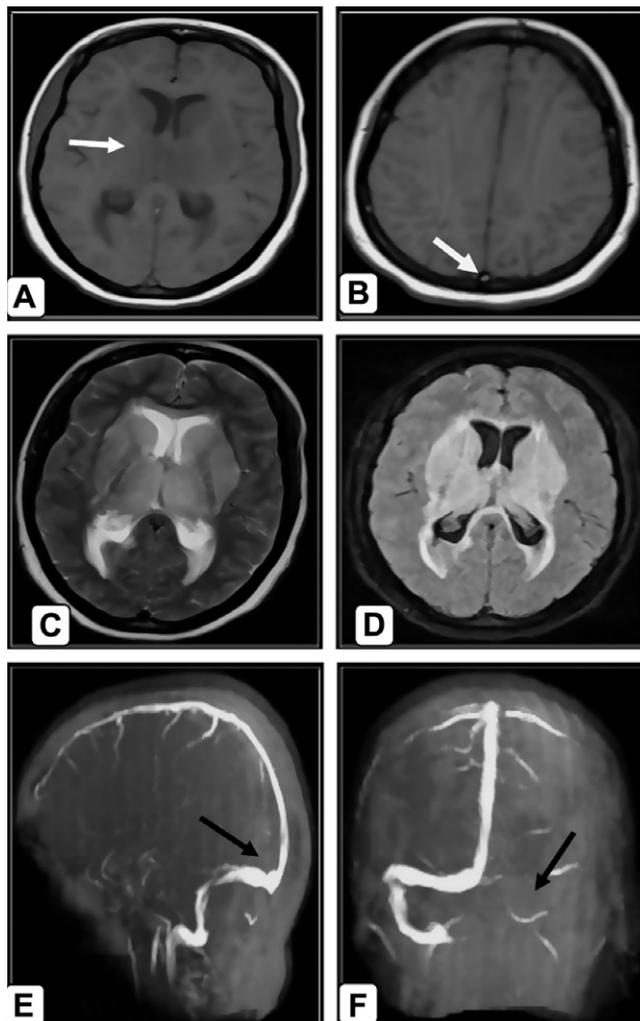


Figure 3 42-year-postpartum female patient complaint of headache, disturbed level of consciousness and coma. (A and B) axial T1WI shows slightly hypo intense area of infarction of bilateral thalami and basal ganglia and hyper intense thrombus in torcular herophili (white arrows). (C) Axial T2WI shows the hyper intense area of infarction of bilateral thalami and basal ganglia but the thrombus appears hypo intense. (D) In axial FLAIR the infarction is hyper intense. (E and F) Sagittal and coronal MRV MIP show no flow in LT transverse, LT sigmoid and straight sinuses with all deep veins and torcular herophili (black arrows) denoting their occlusion.

non-hemorrhagic infarction in (40%) and hemorrhagic infarction found in (26.7%). Nagaraj et al.¹⁵ found hemorrhagic infarction in (40.9%) and non-hemorrhagic infarction in

Table 4 Distribution of patients according to the MRI appearance of signal changes in dural sinuses and veins.

Stage of thrombus	T1WI		T2WI	
	Intensity	PT NO	Intensity	PT NO
Subacute stage (8)	Hyper	8	Hypo	2
Early subacute (2)			Hyper	6
Late subacute (6)				
Chronic stage (4)	Hypo	4	Hypo	4

N.B. There was one patient with equivocal signal intensity in both T1 and T2 but was diagnosed successfully by MRV.

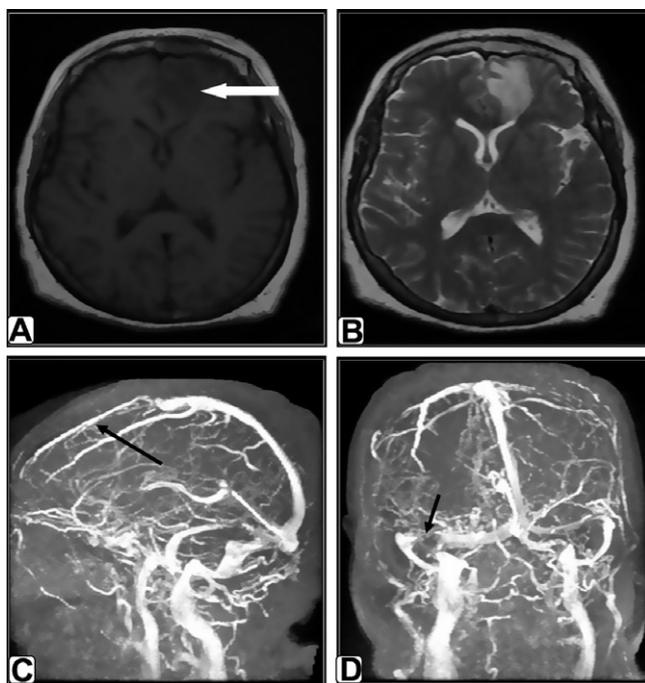


Figure 4 Female patient aged 57-year-old complaint of headache. (A) T1WI shows faint hypo intense area of infarction at the LT frontal lobe (white arrow). (B) T2WI shows LT frontal hyper intense area of infarction without apparent thrombus. (C and D) Sagittal and coronal MRV MIP show no flow in anterior 1/3 of SSS with collaterals formation and distal portion of RT transverse sinus (black arrows) proved their occlusion. Also incidentally noted attenuated LT transverse sinus denoting its hypo plastic nature rather than thrombosis.

(51.6%). Eight patients (61.5%) in our study presented with subacute thrombus. Four cases (26.7%) had chronic thrombus but one patient with equivocal signal. No cases of acute thrombus could be detected. According to some estimates, in 10–30% of cases of sinus thrombosis, the thrombus at initial presentation or imaging examination is in the acute stage of formation.^{18–20} Subacute-stage thrombus has been found in 55% of patients and this nearly agreed with our study.^{21,20} and chronic thrombosis was found in 15% of patients.^{22,20} In the current study, MRV was done in all 15 patients using phase contrast technique in the axial plane with coronal reconstruction. MRV successfully diagnosed occlusion in all

15 cases. It was diagnosed by absence of signal intensity with consequent non-visualization of occluded sinuses or veins in all patients, flow defect in one patient and presence of collaterals in three patients. The superior sagittal sinus was most commonly involved accounting for (86.7%) and this agreed with Ameri and Bousser¹⁴ who reported in their study of 110 cases that SSS accounts for (72%). We finally concluded that MRI and MRV are valuable imaging modalities for the diagnosis of CVO especially as the clinical presentation and CT findings are usually non-specific. Therefore, the radiologist in the emergency department should be familiar with the imaging findings of CVO or CVT, and if it is suspected on CT, MRI and phase contrast MRV should be recommended as they are safer, non-invasive, non-ionizing and highly accurate modalities. Also phase contrast MRV has the ability to differentiate thrombosed sinuses from normal variants and other pitfalls.^{18,23}

References

- Bousser MG. Cerebral venous thrombosis: nothing, heparin or local thrombolysis? *Stroke* 1999;**30**:481–3.
- Lanska DJ, Kryscio RJ. Risk factors for peripartum and postpartum stroke and intracranial venous thrombosis. *Stroke* 2000;**31**:1274–82.
- Ferro JM, Lopes MG, Rosas MJ, et al. Long-term prognosis of cerebral vein and dural sinus thrombosis. Results of the venoport study. *Cerebrovasc Dis* 2002;**13**:272–8.
- Ferro JM, Canhao P, Stam J, Bousser MG, Barinagarrementeria F. Prognosis of cerebral vein and dural sinus thrombosis: results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT). *Stroke* 2004;**35**:664–70.
- DeVeber G, Andrew M. Cerebral sino-venous thrombosis in children. *N Engl J Med* 2001;**345**:417–23.
- Ennaifer R, Moussa A, Mouelhi L, et al. Cerebral venous sinus thrombosis as presenting feature of ulcerative colitis. *Acta Gastroenterol Belg* 2009;**72**:350–3.
- Schievink WI, Maya MM. Cerebral venous thrombosis in spontaneous intracranial hypotension. *Headache* 2008;**48**:1511–9.
- Bousser MG, Ferro JM. Cerebral venous thrombosis: an update. *Lancet Neurol* 2007;**6**:162–70.
- Van Buchem MA, Liauw L, Split A, et al. MR angiography of the intracranial venous system. *Radiology* 2000;**214**:678–82.
- Khealani BA, Wasay M, Saadah M, et al. Cerebral venous thrombosis: a descriptive multicenter study of patients in Pakistan and Middle East. *Stroke* 2008;**39**:2707–11.
- Bousser MG. Cerebral venous thrombosis: diagnosis and management. *Eur J Neurol* 2000;**24**:252–8.
- Duddalwar VA. Multislice CT angiography: a practical guide to CT angiography in vascular imaging and intervention. *BJR* 2004;**77**:27–38.
- Einhäupl K, Bousser MG, de Bruijn SF, et al. EFNS guideline on the treatment of cerebral venous and sinus thrombosis. *Eur J Neurol* 2006;**6**:553–9.
- Ameri A, Bousser MG. Cerebral venous thrombosis. *Neurol Clin* 1992;**10**:87–111.
- Nagaraj D, Haridas T, Taly AB, Veerendrakumar M, Subbukrishna DK. Puerperal cerebral venous thrombosis: therapeutic benefit of low dose heparin. *Neurol India* 1999;**47**:43–6.
- Bousser MG, Barnett HJM. Cerebral venous thrombosis. In: Barnett HJM, Mohr JP, Stein BM, Yatsu FM, editors. *Stroke pathophysiology diagnosis and management*. 2nd ed. New York, NY: Churchill Livingstone Inc.; 1992.
- Jeffrey J, Lisa M, Raiseer DM, et al. Cerebral sinus thrombosis diagnosed by MRI and MRV venography in cancer patients. *Neurology* 2000;**55**:309.

18. Simonds GR, Truwit CL. Anatomy of the cerebral vasculature. *Neuroimaging Clin N Am* 1994;**4**:691–706.
19. Hinman JM, Provenzale JM. Hypointense thrombus on T2-weighted MR imaging: a potential pitfall in the diagnosis of dural sinus thrombosis. *Eur J Radiol* 2002;**41**:147–52.
20. Bergui M, Bradac G. Clinical picture of patients with cerebral venous thrombosis and patterns of dural sinus involvement. *Cerebrovasc Dis* 2003;**16**:211–6.
21. Macchi PJ, Grossman RI, Gomori JM, Goldberg HI, Zimmerman LT, Bilaniuk LT. High field MR imaging of cerebral venous thrombosis. *J Comput Assist Tomogr* 1986;**10**:10–5.
22. Favrole P, Guichard J, Crassard I, Bousser MG, Chabriat H. Diffusion-weighted imaging of intra-vascular clots in cerebral venous thrombosis. *Stroke* 2004;**35**:99–103.
23. Villablanca P, Rodriguez FJ, Stockman T, et al. MDCT angiography for detection and quantification of small intracranial arteries: comparison with conventional catheter angiography. *AJR* 2007;**188**:593–602.