Journal of Clinical Neuroscience 19 (2012) 1525–1529

Contents lists available at SciVerse ScienceDirect

Journal of Clinical Neuroscience

journal homepage: www.elsevier.com/locate/jocn

Clinical Study

Imaging predictors of clinical deterioration in cerebral venous thrombosis

Irene Y.L. Yii [a,c], Peter J. Mitchell [b], Richard J. Dowling [b], Bernard Yan [a,c,*]

[a] Department of Medicine, The University of Melbourne, Parkville, Victoria, Australia
[b] Department of Radiology, The Royal Melbourne Hospital, Parkville, Victoria, Australia
[c] Department of Neurology, The Royal Melbourne Hospital, Grattan Street, Parkville, Victoria 3050, Australia

ARTICLE INFO

Article history:
Received 11 February 2012
Accepted 14 February 2012

Keywords:
Sinus
Stroke
Thrombosis
Venous
Venous infarction

ABSTRACT

Cerebral venous thrombosis (CVT) is a rare stroke subtype with a highly variable clinical course. There is limited information on clinical deterioration in these patients, and imaging predictors of deterioration have not been studied adequately. Therefore, we aimed to investigate the radiological predictors of clinical deterioration in patients with CVT. We conducted a retrospective study of 106 consecutive patients from 1997 to 2010. All patients were confirmed as having CVT using imaging techniques. The following clinical data were collected: patient demographics, clinical presentation, radiological findings, treatment and clinical deterioration. Of the 106 patients, there were 77 females and 29 males, with a mean age of 43 years (range 19–79 years). The common symptoms of clinical presentation included headache (72%), seizure (29%) and severe motor impairment (20%). Overall, 34% of the patients with CVT developed clinical deterioration during hospital admission. Univariate analysis showed venous infarcts and hyperintensity on diffusion-weighted imaging (DWI) as predictors of clinical deterioration. Parenchymal haemorrhage, vasogenic oedema, midline shift and thrombosis location were not predictive of clinical deterioration. In conclusion, our study showed that venous infarcts and hyperintensity on DWI were associated with clinical deterioration in patients with CVT. These findings suggest that close monitoring is necessary in these groups of patients as they may require more aggressive therapy.

1. Introduction

Cerebral venous thrombosis (CVT) is uncommon, accounting for only 0.5% of all strokes.[1] The annual incidence of CVT is three to four cases per million in adults, and it more often affects young people and females.[2] Due to the rarity of this disease, there is limited information on CVT in Australia. To our knowledge, only one study has been conducted on CVT in Australia in the last decade,[3] but the diagnosis and management of CVT may have changed since then.

Most studies have focused on the long-term prognosis of patients with CVT, and limited data are available on clinical deterioration. According to a report by the International Study on Cerebral Venous Thrombosis (ISCVT), 23% of patients with CVT may experience clinical deterioration.[4] However, no information was available on predictive factors for clinical deterioration in patients with CVT, in particular imaging predictors.

We aimed to investigate the imaging predictors of clinical deterioration in patients with CVT. We hypothesised that presence of venous infarcts, hyperintensity on diffusion-weighted imaging (DWI), parenchymal haemorrhage, vasogenic oedema, midline shift and thrombosis location are predictive of clinical deterioration in patients with CVT.

2. Patients and methods

2.1. Identification of patients

Ethics Committee approval was obtained from the Royal Melbourne Hospital Human Research Ethics Committee (HREC Project Number QA2010060) prior to the commencement of the study.

The medical records of patients presenting with CVT at Royal Melbourne Hospital between 1997 and 2010 were reviewed retrospectively. A neuroradiology database at the hospital was also used to identify patients with CVT detected on imaging. Patients were included with a confirmed diagnosis of CVT using digital subtraction angiography (DSA), CT venography or MRI/magnetic resonance venography (MRV). For confirmation of the CVT diagnosis, neuroimaging scans were examined independently by three experienced neuroimaging specialists (P.J.M, R.J.D. and B.Y.).

2.2. Data collection

Data were collected on all patients from the day of CVT diagnosis to the day of discharge. Medical records of all patients were

* Corresponding author. Tel.: +61 3 93492477; fax: +61 3 93494489.
E-mail address: bernard.yan@mh.org.au (B. Yan).

0967-5868/$ - see front matter © 2012 Elsevier Ltd. All rights reserved.
http://dx.doi.org/10.1016/j.jocn.2012.02.005

examined for baseline characteristics, initial clinical presentation, treatment and any clinical deterioration. In terms of motor function, patients with a motor power of 0, 1 and 2 were classified as having severe impairment; those with a motor power of 3 and 4 with moderate impairment; while patients with a motor power of 5 had no motor impairment. Clinical deterioration was defined as any new or worsening of neurological symptoms from the day of CVT diagnosis to the day of discharge. These included new onset or worsening of visual disturbances, symptoms secondary to increased intracranial pressure, a decline in motor function, a decrease in Glasgow Coma Scale scores, or new symptoms of sensory changes, dysphasia, neglect or ataxia. Neuroimaging results (DSA, CT venography and MRI/MRV) were reviewed for clot location. Apart from the absence of flow in the thrombosed vessel on MRV, diagnostic criteria for thrombosis location also included hyperintensity within the thrombosed vessel on T1-fat saturation sequence on MRI. The presence of hyperintense lesions on DWI, sulcal effacement, hydrocephalus, mass effect, midline shift, intracranial haemorrhage, venous infarcts and oedema were assessed with MRI and recorded. Venous infarcts were diagnosed with a positive DWI, with hyperintensity on T2-weighted MRI. Venous infarcts were differentiated from arterial infarcts based on the pattern of DWI hyperintensity, as venous infarcts do not conform to a typical arterial territory.

2.3. Statistical analysis

Chi-square test or Fisher's exact test (with Bonferroni correction) was used to test the null hypothesis that the imaging variables were not predictive of clinical deterioration. The following imaging variables were analysed: hyperintensity on DWI, parenchymal haemorrhage, vasogenic oedema, midline shift, venous infarcts and thrombosis location. Statistical analysis was performed with Statistical Package for the Social Sciences version 18.0 (Chicago, IL, USA).

3. Results

A total of 106 patients were included in the study. There was a 3:1 female to male ratio (77 women) with a mean age of 43 years (range 19–79 years).

3.1. Clinical presentation

The initial clinical presentation of patients with CVT is described in Table 1. There were incomplete data on the overall clinical presentation of one patient who presented with headache, and there was no documentation of motor function in seven patients. The clinical presentation of CVT was variable, but the most common symptom was headache, in 76 patients (72%). This was followed by seizures in 30 patients (29%) and severe motor impairment in 20 patients (20%). Some patients had more severe symptoms such as impaired level of consciousness and symptoms secondary to increased intracranial pressure (11% each). Patients also presented with visual problems; six patients (6%) experienced visual loss while 18 patients (17%) had papilloedema. In terms of the patients who presented with headache and papilloedema, there were no significant differences between the group with clinical deterioration and the group without.

3.2. Neuroimaging

The diagnosis of CVT was established using MRI/MRV in 101 patients (95%), CT venography in three (3%) and DSA in two (2%). The transverse sinus was the most common site of thrombosis (Table 2) with 85 patients (80%), followed by superior sagittal sinus with 55 (52%). Thirteen patients (12%) had deep venous sinus involvement.

Of those with MRI results (Table 2), 40 patients (40%) had intracranial haemorrhage while 33 (33%) had venous infarcts (Figs. 1 and 2). DWI scans were performed in 84 patients; and of those, hyperintense lesions on DWI were identified in 47 (56%).

Table 1
Demographic details of lot patients with cerebral venous thrombosis and their clinical presentation

	No. of patients	%
Mean age = 42.75 years (range 19–79 years)		
Female	77	72.6
Headache	76	71.7
Motor function		
No impairment	53	53.5
Moderate impairment	26	26.3
Severe impairment	20	20.2
Increased intracranial pressure	12	11.4
Seizure	30	28.6
Visual loss	6	5.7
Papilloedema	18	17.1
Diplopia	11	10.5
Palsy of cranial nerve VI	6	5.7
Aphasia	18	17.1
Sensory deficit	11	10.5
Hemiparesis	16	15.2
Ataxia	6	5.7
Glasgow Coma Scale score		
14–15	85	81.0
9–13	8	7.6
<9	12	11.4

Table 2
Neuroimaging findings of 106 patients with cerebral venous thrombosis

	No. of patients	(%)
Thrombosis location		
Superior sagittal sinus	55	51.9
Transverse sinus	85	80.2
Sigmoid sinus	50	47.2
Cortical veins	19	17.9
Deep venous system	13	12.3
Straight sinus	11	10.4
Inferior sagittal sinus	3	2.8
Others (vein of Galen)	4	3.8
MRI findings		
DWI hyperintensity	47	56.0
Oedema	40	39.6
Vasogenic oedema	27	26.7
Cytotoxic oedema	13	12.9
Venous infarcts	33	32.7
Intracranial haemorrhage	40	39.6
Parenchymal haemorrhage	37	36.6
Subarachnoid haemorrhage	5	5.0
Sulcal effacement	12	11.9
Hydrocephalus	3	3.0
Mass effect	23	22.8
Midline shift	12	11.9

Patients may have had more than one occluded sinus/vein.
DWI = diffusion-weighted imaging.

3.3. Treatment

Data on treatment were recorded for 99 patients and there were incomplete data for seven patients (Table 3). There was no significant difference in treatment between the group with clinical deterioration and the group without. A total of 91 patients (92%) were treated with anticoagulation; among them, eight patients (8%) underwent dural thrombolysis. For the group of patients receiving