

Radiological findings in cerebral venous thrombosis presenting as subarachnoid hemorrhage: a series of 22 cases

Monique Boukobza¹ · Isabelle Crassard² · Marie-Germaine Bousser² · Hugues Chabriat^{2,3}

Received: 26 August 2015 / Accepted: 9 September 2015 / Published online: 16 September 2015
© Springer-Verlag Berlin Heidelberg 2015

Abstract

Introduction The main objectives of the present study are to assess the incidence of cerebral venous thrombosis (CVT) presenting as isolated subarachnoid hemorrhage (SAH) and to determine the occurrence of cortical venous thrombosis (CoVT).

Methods Among 332 patients with CVT, investigated with the same CT and MR standardized protocol, 33 (10 %) presented with SAH, associated in 11 cases with hemorrhagic infarct or intracerebral hemorrhage. This study is based on 22 cases of CVT presenting as SAH in the absence of hemorrhagic brain lesion. Diagnosis of sinus thrombosis was established on T2* and magnetic resonance venography and that of CoVT on T2* sequence. Diagnostic of SAH was based on fluid-attenuated inversion recovery (FLAIR) sequence.

Results CVT involved lateral sinus in 18 patients, superior sagittal sinus in 16, and straight sinus in 1. Cortical veins were involved in all patients, in continuity with dural sinus thrombosis when present. SAH was circumscribed to few sulci in all cases and mainly localized at the convexity (21 cases). CoVT

implied different areas on the same side in four patients and was bilateral in seven. There was no perimesencephalic or basal cisterns hemorrhage. Cortical swelling was present in 12 cases, associated with localized edema. All patients except one had a favorable outcome.

Conclusion This report shows that the incidence of CVT presenting as isolated SAH is evaluated to 6.4 % and that SAH is, in all cases, in the vicinity of CoVT and when dural thrombosis is present in continuity with it.

Keywords Cerebral venous thrombosis · Cortical venous thrombosis (CoVT) · T2* · Subarachnoid hemorrhage · Cerebral convexity

Abbreviations

CVT Cerebral venous thrombosis
CoVT Cortical vein thrombosis
SAH Subarachnoid hemorrhage
SSS Superior sagittal sinus

Introduction

Cerebral venous thrombosis (CVT) has long been known as a cause of parenchymal hemorrhage. Subarachnoid hemorrhage (SAH), isolated or associated with parenchymal hemorrhages, is a rare presentation of CVT, only reported as isolated case reports or in small series. Up to now, 41 patients with complete radiologic data have been reported with CVT-related SAH out of which 32 presented isolated SAH and 26 have been evaluated by MRI examination [1–23]. Systematic studies investigating SAH occurring in CVT and using T2* sequence are missing, and the incidence of this

✉ Monique Boukobza
monique.boukobza@lrh.aphp.fr

¹ Department of Neuroradiology and Therapeutic Angiography, Assistance publique – University Hospitals Lariboisière-St-Louis-Fernand-Widal, APHP – Paris-Diderot University, France, 2, rue Ambroise Paré, 75010 Paris, France

² Department of Neurology, Assistance publique – University Hospitals Lariboisière-St-Louis-Fernand-Widal, APHP – Paris-Diderot University Paris, France, Paris, France

³ INSERM UMR 1161 and DHU NeuroVasc, Paris, France

association is not known. Therefore, the main objective of the present study was to evaluate the incidence and the radiological findings, using T2*, of SAH associated with CVT, with attention to the presence, location, and distribution of associated cortical venous thrombosis (CoVT).

This study is based on 22 patients seen in our institution from 2003 to 2013 who had CVT presenting as SAH without associated parenchymal hemorrhage.

Patients and methods

In our prospective registry of consecutive CVT patients, we retrospectively analyzed the data of all subjects (22 patients) with SAH (in the absence of parenchymal hemorrhage)-associated CVT between January 2003 and December 2013.

Non-contrast brain CT and MRI were obtained within 24 h of admission and at 3 months of follow-up.

Brain CT was performed on a CT 64-slice multi-detector row CT scan. CT imaging was obtained with 2.5-mm section thickness through the posterior fossa with 5-mm section thickness through the supratentorial hemispheres.

MRI was performed on a 1.5-T unit (GE Medical System) using a standardized protocol for brain examination, including sagittal T1, axial fluid-attenuated inversion recovery (FLAIR), coronal T2SE, T2*, and diffusion-weighted imaging (DWI) sequences, apparent diffusion coefficient (ADC) map and 2D time-of-flight (TOF) MR venography (MRV).

The diagnosis of both CVT and SAH was based on MRI using the following criteria: (1) for CVT, presence of intraluminal thrombosis in sinuses or veins on T1 (isointense or hyperintensity) or T2 (isointensity or hyperintensity) and on T2* (hypointensity), associated with a filling defect in venous structures on 2D TOF MRV; CoVT was recognized as a well-delineated, tubular, serpentine, or rounded hypointense signal on T2*, and (2) for SAH, presence of hyperintense subarachnoid signals on FLAIR sequence, without associated hemorrhagic parenchymal lesions on T2* sequence.

Clinical signs and etiological workup were recorded in detail for all patients.

Non-hemorrhagic intraparenchymal lesions were classified as cortical swelling when there was a sulcus effacement around the cortical SAH on FLAIR sequence, as localized parenchymal edema when there was an area of hypodensity on CT or of hyperintensity on FLAIR and DWI, and as cortical infarction when the DWI hypersignal was associated with ADC restriction [5].

Results

Among 332 patients with CVT, investigated during the same period in our institution with the same CT and MR standardized protocol, 33 (10 %) presented with SAH, associated in 11 cases with hemorrhagic infarct or intracerebral hemorrhage.

Thus, our study is based on the cohort of 22 patients (6.4 %) who had CVT presenting as SAH without associated parenchymal hemorrhage: 18 women and 4 men, mean age 39 years old (range 24–54).

CT and MR images were reviewed independently by a neuroradiologist (MB) and a neurologist (IC) experienced in CVT and blinded to the clinical and imaging findings. There was a complete agreement in the assessment of neuroradiological findings.

Baseline clinical, etiological, and radiological data are summarized in Table 1.

Headache was present in 21 patients (95 %), and in all, it was the initial symptom. Headache was of the thunderclap type in only three patients (Table 1). All patients received heparin (UFH or LMWH). One patient (no. 2) died of pulmonary embolism at day 9. The 21 surviving patients all had an excellent 3-month functional outcome.

SAH was visualized on non-enhanced CT in 7/22 cases. It was limited to a few sulci, appearing very slightly hyperdense in two cases (Fig. 1a). On FLAIR sequence, SAH was localized unilaterally and in most cases circumscribed to a few sulci at the convexities (21/22 cases) (Fig. 1b) (Table 1). In addition to convexity location, SAH was present in the frontal parasagittal sulci (Fig. 2) and in the distal sylvian fissure in respectively one case each.

Twelve patients had a localized cortical swelling adjacent to SAH, including five with additional localized subcortical edema.

Sinus thrombosis was present in 21/22 cases. Thrombosis affected both superior sagittal sinus (SSS) and lateral sinus (LS) in 12 patients.

CoVT was present in all patients. It appeared in all cases as a tubular, serpiginous strongly hypointense structure on T2*, hyperintense on T1 in five cases, and hyperintense on FLAIR and DWI in only one case. CoVT was visibly adjacent to the area of SAH in all cases (Figs. 1 and 2). When there was a sinus thrombosis, the cortical thrombus was always in continuity with it (Fig. 2).

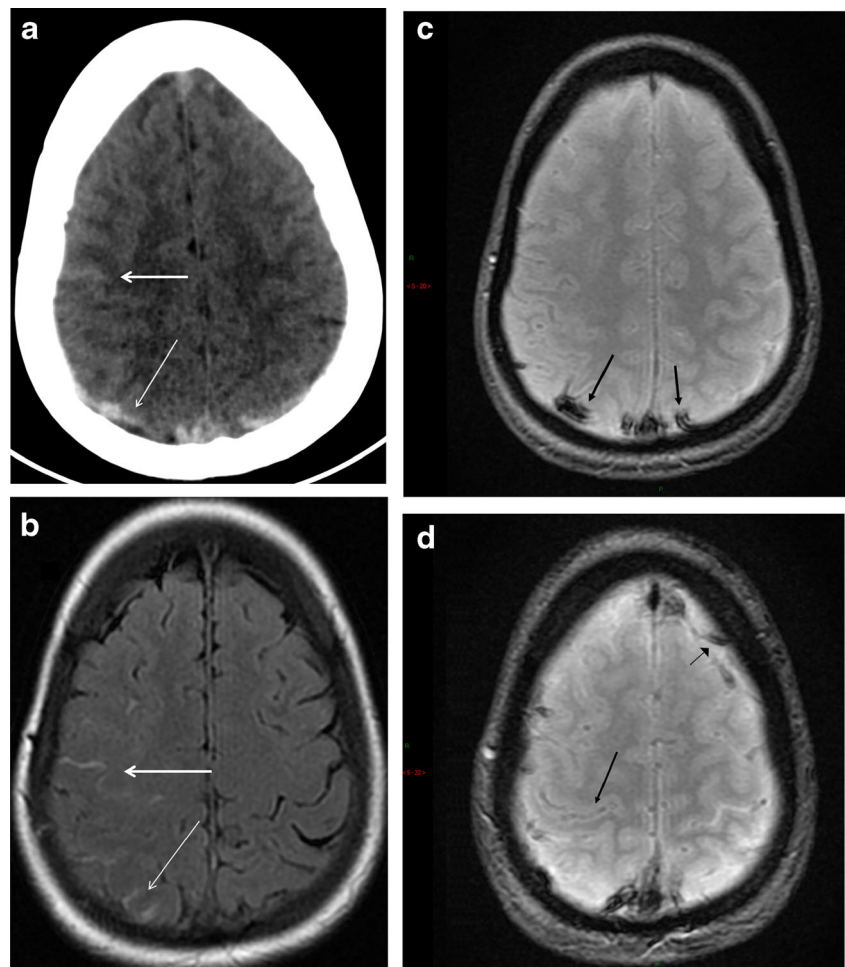
The vein most frequently involved was the vein of Labbé (14 patients), always in association with ipsilateral LS thrombosis. Frontal veins were involved in 11 patients, in association with SSS thrombosis in 10. There was frequently an association of several CoVT

Table 1 Clinical data and neuroradiological findings in 22 patients with CVT presenting as SAH

No.	Age/sex	Clinical presentation	Risk factors/causes	Sinus thrombosis	Cortical veins	SAH location	Localized cortical	
							Swelling	Edema
1	52/F	Diffuse headache, NV, generalized seizure	OC	SSS + right TS	Frontal (R)-parietal (Bil)	FTP (R) + parasagittal	Y	N
2	49/F	Thunderclap headache, then persistent headache. Visual aura, dysphasia, hemiparesis, coma	OC	Left LS	Labbé vein (L)	Temporal + Sylvian fissure (L)	N	N
3	27/F	Thunderclap headache, then persistent headache. Focal left seizure.	OC	SSS + right LS	Labbé vein (R) frontal bil	Frontal (R)	Y	Y
4	28/M	Gradual right hemiparesis, two generalized seizures	IBD diagnosed 3 months ago	Right LS	Labbé vein (R)	Temporal (R)	N	N
5	37/F	Gradual right hemiparesis, NV. Bilateral papilledema and altered consciousness	OC, post-abortion	SSS + right LS + SS	Labbé vein (R)-parietal (Bil)	Temporal (R)	Y	Y
6	35/M	Diffuse headache in context of viral meningitis. Left motor seizure with persistent paresis	Meningitis	SSS	Frontal (R)	Interhemispheric	N	N
7	49/F	Gradual left hemiparesis. Hypoesthesia left V	Essential thrombocytemia	Left LS	Labbé vein (L)	Temporal (L)	Y	N
8	32/F	Gradual diffuse headache, NV, left hemiparesis, altered consciousness	OC	SSS-right LS	Fronto-parietal (bil)	Frontal (R)	N	N
9	33/F	Orthostatic headache	OC, spontaneous CSF hypotension		Fronto-parietal (R)	Fronto-parietal (R)	Y	N
10	42/F	Thunderclap headache, after sneezing, then persistent headache. Generalized seizure	OC	SSS + left LS	Labbé vein (L)-frontal (L)	Frontal (R)	N	N
11	24/F	Gradual diffuse headache, NV. Generalized seizure. Hemiparesis, altered consciousness, bilateral papilledema	OC	SSS + left LS	Fronto-parietal (bil)	Frontal bil	Y	Y
12	54/F	Gradual diffuse headache. Bilateral papilledema	Essential thrombocytemia	Left LS + right TS	Labbé vein (L)	Temporal (L)	Y	N
13	44/F	Gradual left hemiparesis. Dysphasia, apraxia	OC	SSS + Left LS	Labbé vein (L)	Temporal (L)	Y	Y
14	43/F	Gradual left hemiparesis (nocturnal)	PAPS, OC	SSS	Frontal (L)	Frontal (L)	N	N
15	31/M	No headache. Left focal seizures	CVT 17 years before	SSS + right LS	Frontal (R)	Frontal (R)	Y	N
16	28/F	Right hemiparesis, then diffuse headache, TVO. Bilateral papilledema	OC	Right LS	Labbé vein (R)	Temporal (R)	Y	N
17	43/F	Right hemiparesis, then diffuse headache, NV. Diplopia, bilateral papilledema	OC, FV Leiden	SSS + right TS	Labbé vein (R)	Temporal (R)	N	N
18	43/F	Gradual diffuse headache. Alteration of consciousness	DVT and PE 8 years before	SSS + right TS	Labbé vein (R) + frontal bil	Frontal (bil)-temporo-occipital (R)	N	N
19	49/M	Right hemiparesis (nocturnal), then diffuse headache, NV. Bilateral papilledema	None	SSS + right TS	Labbé vein (R) + frontal bil	Temporal (R)	Y	Y
20	38/F	Occipital headache Right leg paresis	Post-partum, post peridural CSF hypotension	SSS	Parietal (R)	Parietal (R)	N	N
21	44/F	Gradual diffuse headache, NV	Behcet disease	SSS + right LS	Labbé vein (R)	Temporal (R)	Y	N
22	38/F	Gradual right hemiparesis	None	SSS + right LS	Labbé vein (R)	Temporal (R)	N	N

NV nausea and vomiting, OC oral contraceptives, IBD inflammatory bowel disease, PAPS primary antiphospholipid syndrome, DVT deep vein thrombosis, PE pulmonary embolism, TVO transient visual obscuration, SSS superior sagittal sinus, LS lateral sinus (=transverse + sigmoid sinus), TS transverse sinus, SS straight sinus, L left, R right, FTP fronto-temporo-parietal

Fig. 1 Case no. 3 non-enhanced CT scan (**a**, *short arrow*) and FLAIR images show cortical subarachnoid hemorrhage (SAH) at high right frontal convexity with cortical swelling (**b**, *short arrow*). FLAIR images show additional SAH hyperintensities at high parietal convexity (**b**, *long arrow*). SSS thrombosis and bilateral cortical vein thrombosis (CoVT) are seen on CT (**a**, *long arrow*). On T2*, CoVT appears as tubular hypointense structures converging to the SSS, adjacent to the areas of SAH (**c**). Additional thrombosis of left anterior frontal veins is depicted (**d**, *arrowhead*). Furthermore, an hypointense signal in the right fronto-parietal sulci, compared to the left one, is consistent with blood in the subarachnoid space (hemosiderin deposition) (**d**, *thin arrow*)



(Table 1) (Figs. 1 and 2). In the two cases presenting with bilateral frontal vein thrombosis, SAH was observed bilaterally in the frontal areas. In two other cases, limited SAH was observed in multiple areas. CoVT was isolated in one case, involving the frontal and parietal veins on the same side, and SAH was localized at the ipsilateral fronto-parietal convexity.

Discussion

We report a series of 22 patients with CVT presenting as isolated SAH, i.e., in the absence of brain hemorrhage. This is the largest series so far. The incidence of concomitant CVT and SAH, in the absence of cerebral bleeding, was of 6.4 % in our experience.

SAH was observed at the brain convexity in 21 patients, and it was always localized, involving only a few sulci. The diagnosis of SAH was overlooked on CT scan in 15 patients because of the small amount of

blood. The diagnosis was based on MRI [24–26] which is well established as superior to CT scan for presumptive diagnosis and localization of acute and subacute low-grade SAH. The typical pattern is the presence on FLAIR MRI of hyperintensities in subarachnoid spaces, sometimes associated with hypointensities at the same location on T2*.

In our series, all patients had CoVT and SAH was invariably located adjacent to the thrombosis. CVT is a well established but rare cause of SAH. The diagnosis of CVT in a patient with SAH is crucial, because of the need to treat CVT with heparin whereas, by contrast, heparin is contraindicated in all other causes of SAH. In patients presenting with isolated SAH, the diagnosis of CVT is easy when there is a sinus thrombosis as in 21 of our patients. By contrast, the diagnosis is extremely difficult when there is an isolated cortical vein thrombosis. Firstly, even in the absence of SAH, CoVT is seriously difficult to diagnose, because cortical veins are variable in number and location and because only

the largest veins are detectable on MRV or CT venography [24–26]. Secondly, when there is a SAH localized at the convexity, it is sometimes impossible on FLAIR sequence to differentiate it from a CoVT. T2* sequence is then crucial, showing CoVT as an hypointense tubular structure while SAH appears as a slight hemosiderin deposit [24–26].

Furthermore, as related in our series, SAH localized at the fronto-parietal areas, even bilaterally, and/or at the paragittal sulci should rise the possibility of thrombosis of the SSS and/or of the frontal and parietal veins, while SAH confined to the posterior temporal or temporo-occipital convexity or at the distal sylvian fissure suggests searching for signs of lateral sinus and Labbé vein thrombosis. Thus, the localization of convexal SAH appears to be a good indicator of the involved venous structure.

Localized non-traumatic convexity SAH has diverse causes that need to be ruled out, most commonly amyloid angiopathy and reversible cerebral vasoconstriction

syndrome (RCVS), but the pattern of clinical presentation is different, and other imaging signs are present such as micro-bleeds in amyloid angiopathy and segmental arterial vasoconstriction in RCVS [27–29]. Another important radiological clue in favor of CVT-related SAH is the location purely at the convexity, sparing the basal cisterns (2, 5–7, 10–12).

Different hypothesis have been suggested to explain the occurrence of SAH in patients with CVT. A first hypothesis is that sinus thrombosis increases the venous pressure, thereby causing the dilatation and rupture of the adjacent fragile, thin-walled cortical veins. This could lead to the localized hemorrhage entering the subarachnoid space [2, 11, 17, 30, 31]. Another hypothesis is that SAH could be due to the secondary rupture of parenchymal hemorrhagic lesions into the subarachnoid space [2, 4, 17]. This was however not the case in our patients since patients with brain hemorrhages were excluded. A third possibility is that the extension of sinus thrombosis into cortical or cerebellar veins causes a localized venous hyperpressure with subsequent dilatation and eventually rupture of these veins. Such a mechanism has been demonstrated in experimental studies [32–34]. Our findings are in favor of this last hypothesis, since there was in all cases a high concordance between the location of SAH and CoVT, which was in continuity with sinus thrombosis in 21 cases. Thrombosed cortical veins thus seem to be the cause of isolated SAH in patients with CVT.

Conclusion

SAH in the presence of CVT is uncommon, occurring in 6.4 % of CVT in our experience.

This report shows that in CVT presenting as isolated SAH, SAH is, in all cases, circumscribed and closely adjacent to the area of cortical vein thrombosis and when dural thrombosis is present in continuity with it. The presence of acute and limited SAH of the convexity, especially when basal cisterns are spared, should prompt dedicated vascular imaging, including the venous system along with T2* sequence.

Ethical standards and patient consent We declare that all human studies have been approved by the Ethics Committee of our Institution and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. We declare that all patients gave informed consent prior to inclusion in this study.

Conflict of interest We declare that we have no conflict of interest.

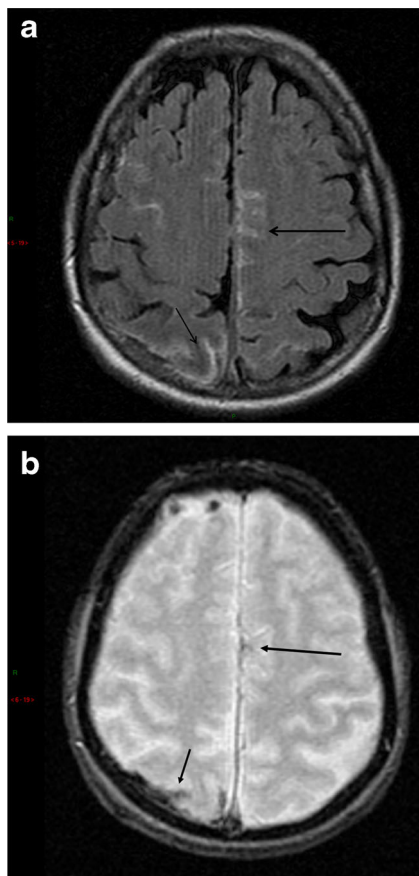


Fig. 2 Case no. 1. FLAIR images show two locations of SAH: in the fronto-parietal right sulci (*short arrows*) and in the left parasagittal sulci (*long arrow*) (a). T2*, in the same plane, show the cortical thrombosed vein adjacent to the area of SAH (*b, short arrow*) and hemosiderin deposition in the left parasagittal sulci (*b, long arrow*)

References

- Ciccone A, Citterio A, Santilli I, Sterzi R (2000) Subarachnoid haemorrhage treated with anticoagulants. *Lancet* 356:1818
- Sztajzel R, Coeytaux A, Dehdashati AR, Delavelle J, Sinnreich M (2001) Subarachnoid hemorrhage: a rare presentation of cerebral venous thrombosis. *Headache* 41:899–901
- Selim M, Fink J, Linfante I, Kumar S, Schlaug G, Caplan LR (2002) Diagnosis of cerebral venous thrombosis with echoplanar T2*-weighted magnetic resonance imaging. *Arch Neurol* 59:1021–1026
- Widjaja E, Romanowski CA, Sinanan AR, Hodgson TJ, Griffiths PD (2003) Thunderclap headache: presentation of intracranial sinus thrombosis? *Clin Radiol* 58:648–652
- Chang R, Friedman DP (2004) Isolated cortical venous thrombosis presenting as subarachnoid hemorrhage: a report of three cases. *AJNR Am J Neuroradiol* 25:1676–1679
- Oppenheim C, Domingo V, Gauvrit J-Y, Lamy C, Mackowiak-Cordoliani M-A, Pruvo J-P, Meder J-F (2005) Subarachnoid hemorrhage as the initial presentation of dural sinus thrombosis. *AJNR Am J Neuroradiol* 26:614–617
- Spitzer C, Mull M, Rohde V, Kosinski CM (2005) Non-traumatic cortical subarachnoid haemorrhage: diagnostic work-up and aetiological background. *Neuroradiology* 47:525–531
- Adaletti I, Sirikei A, Kara B, Kurugoglu S, Ozer H, Bayram M (2005) Cerebral venous sinus thrombosis presenting with excessive subarachnoid hemorrhage in a 14-year-old boy. *Emerg Radiol* 12:575–579
- Rice H, Tang YM (2006) Acute subarachnoid hemorrhage: a rare presentation of cerebral dural sinus thrombosis. *Austr Radiol* 50:241–245
- Lin JH, Kwan SY, Wu D (2006) Cerebral venous thrombosis initially presenting with acute subarachnoid hemorrhage. *J Chin Med Assoc* 69:282–285
- Pradhan S, Yadav R, Diwakar H, Phadke RV (2007) Subarachnoid hemorrhage following chronic dural venous sinus thrombosis. *Angiology* 58:498–501
- Wang YF, Fuh JL, Lirng JF, Chang FC, Wang SJ (2007) Spontaneous intracranial hypotension with isolated cortical vein thrombosis and subarachnoid haemorrhage. *Cephalalgia* 27:1413–1417
- Tang PH, Chai J, Chan YH, Chng SM, Lim CC (2008) Superior sagittal sinus thrombosis: subtle signs on neuroimaging. *Ann Acad Med Singapore* 37:397–401
- Jaiser SR, Raman A, Maddison P (2008) Cerebral venous sinus thrombosis as a rare cause of thunderclap headache and nonaneurysmal subarachnoid haemorrhage. *J Neurol* 255:448–449
- Lai NK, Hui JW, Wong GK, Yu SC, Sun DT, Poon WS (2008) Cerebral venous thrombosis presenting as subarachnoid haemorrhage. *Hong Kong Med J* 14:499–500
- Benabu Y, Mark L, Daniel S, Glikstein R (2009) Cerebral venous thrombosis presenting with subarachnoid hemorrhage. Case report and review. *Am J Emerg Med* 27:96–106
- Kato Y, Takeda H, Furuya D, Nagoya H, Deguchi I, Fukuoka T, Tanahashi N (2010) Subarachnoid hemorrhage as the initial presentation of cerebral venous thrombosis. *Intern Med* 49:467–470
- Morris JG, Fisher M, Carandang RA (2010) Cortical vein thrombosis as a mimic for isolated cortical subarachnoid hemorrhage and transient ischemic attack. *Case Rep Neurol* 2:63–68
- Panda S, Prashantha DK, Shankar SR, Nagaraja D (2010) Localized convexity subarachnoid haemorrhage—a sign of early cerebral venous sinus thrombosis. *Eur J Neurol* 17:1249–1258
- Oda S, Shimoda M, Hoshikawa K, Osada T, Yoshiyama M, Matsumae M (2011) Cortical subarachnoid hemorrhage caused by cerebral venous thrombosis. *Neurol Med Chir* 51:30–36
- Sayadnasiri M, Taheraghdam AA, Talebi M (2012) Cerebral venous thrombosis presenting as subarachnoid hemorrhage: report of two cases. *Clin Neurol Neurosurg* 114:1099–1101
- Yamamoto J, Kakeda S, Takahashi M, Idei M, Nakano Y, Soejima Y, Saito T, Akiba D, Shibata E, Korogi Y, Nishizawa S (2013) Severe subarachnoid hemorrhage associated with cerebral venous thrombosis in early pregnancy: a case report. *J Emerg Med* 45:849–855
- Sahin N, Solak A, Genc B, Bilgic N (2014) Cerebral venous thrombosis as a rare cause of subarachnoid hemorrhage: case report and literature review. *Clin Imaging* 38:373–379
- Idbaih A, Boukobza M, Crassard I, Porcher R, Bousser MG, Chabriat H (2006) MRI of clot in cerebral venous thrombosis: high diagnostic value of susceptibility-weighted images. *Stroke* 37:991–995
- Leach JL, Strub WM, Gaskill-Shibley MF (2007) Cerebral venous thrombus signal intensity and susceptibility effects on gradient recalled-echo MR imaging. *AJNR Am J Neuroradiol* 28:940–945
- Boukobza M, Crassard I, Bousser MG, Chabriat H (2009) MRI features of isolated cortical vein thrombosis: diagnosis and follow-up. *AJNR Am J Neuroradiol* 30:344–348
- Khurram A, Kleinig T, Leyden J (2014) Clinical associations and causes of convexity subarachnoid hemorrhage. *Stroke* 45:1151–1153
- Ducros A, Boukobza M, Porcher R, Sarov M, Valade D, Bousser MG (2007) The clinical and radiological spectrum of reversible cerebral vasoconstriction syndrome. A prospective series of 67 patients. *Brain* 130:3091–3101
- Katoh M, Yoshino M, Katsuyuki A, Aoki T, Imamura H, Kashiwazaki D, Takano K, Aida T (2007) A restricted subarachnoid haemorrhage in the cortical sulcus in cerebral amyloid angiopathy: could it be a warning sign? *Surg Neurol* 68:457–460
- Ameri A, Bousser MG (1992) Cerebral venous thrombosis. *Neurol Clin* 10:87–111
- Villringer A, Mehraein S, Einhüpl KM (1994) Pathophysiological aspects of cerebral sinus venous thrombosis (SVT). *J Neuroradiol* 21:72–80
- Nakase H, Heimann A, Kempfski O (1996) Local cerebral blood flow in a rat cortical vein occlusion model. *J Cereb Blood Flow Metab* 16:720–728
- Nakase H, Takeshima T, Sakaki T, Heimann A, Kempfski O (1998) Superior sagittal sinus thrombosis: a clinical and experimental study. *Skull Base Surg* 8:169–174
- Stracke CP, Spuentrup E, Katoh M, Günther RW, Spangenberg P (2006) New experimental model of sinus and cortical vein thrombosis in pigs for MR imaging studies. *Neuroradiology* 48:721–729