Epidemiology, diagnosis and treatment of cerebral venous thrombosis
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Small juxtacortical hemorrhages: a characteristic sign of cerebral venous thrombosis

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Submitted
Abstract

Background: Intracerebral haemorrhages are common in patients with cerebral venous thrombosis (CVT). We examined whether small juxtacortical haemorrhages (JCH) are characteristic for CVT and studied their radiological and pathological properties.

Methods: We identified all patients with CVT and an intracerebral haemorrhage at baseline admitted to our hospital between 2000 and 2010 (prospectively from July 2006). A JCH was defined as a small haemorrhage (diameter < 20 mm), located in the white matter just below the cortex. To determine the specificity of JCHs for CVT, we examined the frequency of JCHs in a control group of patients of similar age with an intracerebral haemorrhage not related to CVT. All imaging results were re-evaluated by two neuroradiologists.

Results: Of 114 patients with CVT, 53 had an intracerebral haemorrhage. One or more JCHs were present in 14/53 (26%) of them. The remaining 39 had other kinds of haemorrhages. Papilloedema was more common among patients with a JCH compared to patients with other types of haemorrhages (44% vs. 9%, p=0.01). Other clinical characteristics and outcome did not differ significantly. All patients with a JCH except one (93%) had thrombosis of the superior sagittal sinus, compared to 49% of patients with CVT and other kinds of haemorrhages (p=0.004). Thrombosis of the other cerebral sinuses did not differ significantly between the groups. Re-analysis of the cerebral imaging showed that JCHs are confined to the juxtacortical white matter, near the U-fibres, and that they follow the curvature of the cortex. This feature results in a cashew-nut or round appearance, depending on the location of the JCH. These findings were confirmed by histopathologic analysis of the brain of patient with CVT and JCHs. Among the 196 control patients (spontaneous intracerebral haemorrhage, not caused by CVT) only three patients had a JCH. One of these three controls appeared on re-examination of all imaging results to have had CVT, which had been missed at first presentation.

Discussion: The location and the shape of JCHs and their association with thrombosis of the superior sagittal sinus suggest that these haemorrhages probably originate in the arcuate segment of the subcortical vein of the
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superficial venous system. We conclude that small juxtacortical haemorrhages are a characteristic feature of cerebral venous thrombosis and rarely encountered in other conditions.
Introduction

Cerebral venous thrombosis (CVT) is a particular type of stroke with an annual incidence of approximately 1.3 per 100,000. Its clinical manifestations are highly variable, and range from chronic headache to a severe encephalopathy with seizures and coma. Localized cerebral edema and haemorrhages frequently occur as a consequence of venous thrombotic occlusions. Large haemorrhagic infarcts can cause death by cerebral herniation. Because of the variable presentation and the rarity of the disease, the diagnosis is difficult and may be missed at the first consultation. A diagnostic delay can be detrimental because progression of the thrombosis may cause further clinical deterioration if adequate treatment with heparin is delayed. Timely recognition of CVT is therefore important.

Most patients with symptoms of CVT - often patients in their thirties or forties with recent headache, seizures, or neurologic deficits - are initially examined in the emergency room. A non contrast-enhanced CT-scan (NCCT) of the brain is usually the first radiological examination. A common CT finding in patients with CVT is an intracerebral haemorrhage, present in approximately 40% of patients. The morphology of these haemorrhages varies, ranging from localized subarachnoid haemorrhage and small juxtacortical lesions to large, space-occupying haemorrhagic infarcts. Since diagnosing CVT can be difficult, recognition of abnormalities on cranial imaging that suggest CVT is important. In our experience, small juxtacortical haemorrhages (JCH) occur frequently in patients with CVT, but rarely in other conditions. We examined whether these JCHs are indeed characteristic for CVT and if they are associated with a distinctive clinical profile. We also examined the pathological properties and propose a pathophysiological mechanism for the development of these haemorrhages.

Methods

Patient identification and selection
Our hospital is a tertiary referral centre for CVT cases in the Netherlands. The study cohort consisted of consecutive patients with CVT and an intracerebral haemorrhagic lesion on baseline CT imaging, treated between January 1st 2000
and January 1st 2011. We recorded all CVT patients admitted after July 2006 in a prospective database. Patients treated between January 1st 2000 and June 30th 2006 were identified retrospectively using two different search methods. First, we identified cases with the international classification of diseases, 9th revision (ICD-9) coding system. We compiled a list of every patient who received any of the following codes in the specified time period: 437.6 (non-pyogenic thrombosis of intracranial venous sinus), 325 (phlebitis and thrombophlebitis of intracranial venous sinuses), 671.5 (other phlebitis and thrombosis of pregnancy and puerperium), and 437.8 (other cerebrovascular disease). Second, we used the Dutch financial coding system for hospital care (DBC) to search for patients. This system assigns a code to each patient which corresponds to the diagnosis. The appropriate code for CVT is 1199 “cerebrovascular diseases, not otherwise specified”. We hand-searched the medical records and cerebral imaging of all patients identified by either search method to confirm or refute the diagnosis of CVT. For confirmation of the diagnosis CVT we required thrombosis of at least one cerebral sinus on one of the following: CT-venography, MRI with MR-venography, conventional cerebral angiography or autopsy, according to international standards. Children under the age of 12 were excluded.

To determine the specificity of JCHs for the diagnosis CVT, we composed a control group of consecutive adult patients with an intracerebral haemorrhage that was not due to CVT, admitted in the same time period as the CVT cases. These patients were identified with the ICD-9 code 431 (Intracerebral haemorrhage). Since CVT is very rare in elderly patients, we regarded patients above the age of 60 inappropriate as controls and excluded them. Patients with traumatic haemorrhages, aneurysmatic subarachnoid haemorrhages or isolated intraventricular haemorrhages were excluded. Because we have never encountered a JCH located in the cerebellum or brainstem, haemorrhages restricted to these locations were also excluded from the control group.

Clinical data
In the prospective database, information on demographics, date of symptom onset and admission, baseline clinical manifestations, and risk factor profile is recorded on a case record form for each patient. For the retrospective cohort, this information was extracted from the medical records. We classified clinical outcome according to the modified Rankin Scale (mRS), a 7 point scale which
ranges from 0 (complete recovery) to 6 (dead). We defined good outcome as a mRS score of 0 or 1 at last follow-up. Unless the mRS had already been recorded at an earlier stage, patients from the retrospective period were contacted by telephone to determine the score on the mRS with a structured interview.10

Evaluation of neuroimaging
All imaging results were re-examined by two board certified neuroradiologist (RvdB or CBM). The baseline NCCT was assessed for the presence and type of intracerebral haemorrhagic lesions. A JCH was defined as a small haemorrhage (largest diameter < 20 mm), located just below the cortex and exclusively in the white matter (see figure 1A for example). All intracerebral haemorrhages caused by CVT that did not fulfil these criteria were scored as “other intracerebral haemorrhage". If a single patient had both a JCH and another type of intracerebral haemorrhage, it was classified as “other intracerebral haemorrhage". Patients without haemorrhagic lesions, and those with only subdural or subarachnoid haemorrhages were excluded. We scored the total number of JCHs, the widest diameter, and the location of the thrombosis. The baseline NCCT scans of the control patients (intracerebral haemorrhage, but no CVT) were scored for the presence of a JCH.

Histopathologic analysis
Autopsy was performed on a patient with CVT who had a JCH and who died of central transtentorial herniation. Fixed coronal brain sections were embedded in paraffin, and whole-mount 6 μm thick sections were made. Slices were stained for hematoxylin-eosin, Klüver-Barrera and Elastic van Gieson.

Statistical analysis
We analysed categorical data with the X2 test and continuous data with a Mann–Whitney test. All data were analyzed with SPSS, version 20.

Results

Patient selection and baseline clinical data
Between July 2006 and December 2010, 70 patients with CVT were included in the prospective registry and 44 cases admitted between 2000 and 2006
were identified retrospectively. From these 114 patients, 61 were excluded because they had no haemorrhagic lesion at baseline (n=47), or were younger than 12 (n=14). Thus, 53 patients with CVT and cerebral haemorrhages were retained in the study. Fourteen (26%) of them fulfilled the criteria for a JCH and the remaining 39 had other types of haemorrhages. There were no significant differences in age, duration of symptoms to diagnosis, and sex ratio between patients with a JCH and those with other types of intracerebral haemorrhage (table 1). Papilloedema was significantly more common among patients with a JCH (44% vs. 9%, p=0.01). Other baseline clinical characteristics or risk factors did not differ significantly.

Table 1 Baseline characteristics

<table>
<thead>
<tr>
<th></th>
<th>JCH n=14</th>
<th>Other ICH n=39</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Baseline clinical symptoms</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (median, IQR)</td>
<td>35 (25-47)</td>
<td>40 (35-51)</td>
<td>NS</td>
</tr>
<tr>
<td>Duration symptom onset – diagnosis (days, IQR)</td>
<td>3 (1-7)</td>
<td>3 (1-4)</td>
<td>NS</td>
</tr>
<tr>
<td>Sex (% females)</td>
<td>79%</td>
<td>77%</td>
<td>NS</td>
</tr>
<tr>
<td>Glasgow Coma Scale (IQR)</td>
<td>11 (5-15)</td>
<td>13 (8-15)</td>
<td>NS</td>
</tr>
<tr>
<td>Headache</td>
<td>93%</td>
<td>79%</td>
<td>NS</td>
</tr>
<tr>
<td>Focal neurological deficit</td>
<td>80%</td>
<td>51%</td>
<td>NS</td>
</tr>
<tr>
<td>Papilledema</td>
<td>44%</td>
<td>9%</td>
<td>0.01</td>
</tr>
<tr>
<td>Seizure(s)</td>
<td>31%</td>
<td>39%</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Risk factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oral contraceptives*</td>
<td>100%</td>
<td>74%</td>
<td>NS</td>
</tr>
<tr>
<td>Pregnancy or puerperium*</td>
<td>9%</td>
<td>10%</td>
<td>NS</td>
</tr>
<tr>
<td>Genetic thrombophilia</td>
<td>30%</td>
<td>30%</td>
<td>NS</td>
</tr>
<tr>
<td>Inflammatory bowel disease</td>
<td>0%</td>
<td>8%</td>
<td>NS</td>
</tr>
<tr>
<td>Previous thrombosis</td>
<td>7%</td>
<td>13%</td>
<td>NS</td>
</tr>
<tr>
<td>Malignancy</td>
<td>0%</td>
<td>13%</td>
<td>NS</td>
</tr>
</tbody>
</table>

JCH = juxtacortical hemorrhage, ICH = intracerebral hemorrhage, IQR = interquartile range, NS = not significant. *Percentage of women.

Cerebral imaging

All patients except one with a JCH had a thrombosis of the superior sagittal sinus, compared to 49% of patients with other kinds of intracerebral haemorrhage (p=0.004, table 2). Distribution of thrombosis of the other sinuses did not differ significantly between the groups, although thrombosis of the right lateral sinus was more common among patients with a JCH (p=0.06). The majority of patients with a JCH had multiple haemorrhages, with a median number
of two JCHs per patient (IQR 1-5). One patient had 19 different JCHs across both hemispheres (figure 1B). In four of the 14 patients (29%) JCHs occurred bilaterally and most JCHs were located in the frontal and parietal lobes. The median diameter of the JCHs was 9 mm (IQR 7-12 mm). Figure 1 depicts several examples of JCHs on axial NCCT images, showing that these haemorrhages are confined to the juxtacortical white matter and that they follow the curvature of the cortex. A juxtacortical location near the bottom of a sulcus results in a characteristic concave shape, resembling a cashew nut (see examples in figure 1A, C and D). If the haemorrhage is situated in a gyrus, it usually appears more round or elongated (figure 1B). Unlike larger haemorrhagic venous infarcts, JCHs generally have little or no surrounding oedema and do not cause cerebral displacement.

Table 2 Radiological findings

<table>
<thead>
<tr>
<th>Location thrombosis</th>
<th>JCH n=14</th>
<th>Other ICH n=39</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior sagittal sinus</td>
<td>93%</td>
<td>49%</td>
<td>0.004</td>
</tr>
<tr>
<td>Left lateral sinus*</td>
<td>43%</td>
<td>39%</td>
<td>NS</td>
</tr>
<tr>
<td>Right lateral sinus*</td>
<td>64%</td>
<td>33%</td>
<td>0.06</td>
</tr>
<tr>
<td>Straight sinus</td>
<td>43%</td>
<td>21%</td>
<td>NS</td>
</tr>
<tr>
<td>Deep venous system$</td>
<td>21%</td>
<td>15%</td>
<td>NS</td>
</tr>
<tr>
<td>Significant mass effect</td>
<td>0%</td>
<td>62%</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Characteristics JCH

| Number of JCH per patient (median, IQR) | 2 (1-5) | — | NA |
| Multiple JCH (%) | 64% | — | NA |
| Bilateral JCH (%) | 29% | — | NA |
| Diameter JCH (median mm, IQR) | 9 (7-12) | — | NA |

JCH = juxtacortical hemorrhage, ICH = intracerebral hemorrhage, IQR = interquartile range, NS = not significant, NA = not applicable. *Includes sigmoid and transverse sinus. $thrombosis of one of the following veins: internal cerebral veins, great cerebral vein of Galen, or basal vein of Rosenthal.

Findings at autopsy

Autopsy was performed on a 32 year old woman who was initially admitted with headache and vomiting due to a thrombosis of the superior sagittal sinus. After
2 days she clinically deteriorated and became comatose, and was transferred to our hospital for endovascular treatment. Despite a technically successful procedure, the patient died due to progressive generalized cerebral oedema and central transtentorial herniation.

The brain was oedematous, with flattened gyri and localized subarachnoid haemorrhages. The brain surface showed congested veins mainly over the right parietal lobe. The superior sagittal sinus contained several smaller thrombi.
attached to the vessel wall, but the sinus was not completely blocked. The cut surface of the brain after routine fixation showed a cashew nut shaped haemorrhage in the region of the U-fibres (figure 2). Microscopic examination did not reveal a blood vessel or material from a vessel wall in the JCH. In addition several isolated small cortical haemorrhages were present. Throughout the brain, but mainly in the parietal occipital area, several venous thrombi were found. In the area with the JCH shown in figure 2 (see page 66), the superficial cortical vein was partially occluded by a thrombus over a longer distance.

**Treatment and follow-up**

Nearly all patients with CVT were treated with anticoagulation, in agreement with international guidelines (table 3). In addition, a significant minority underwent endovascular thrombolysis or decompressive hemicraniectomy, indicating severity of the condition in patients with CVT and a cerebral haemorrhage. Data on clinical outcome at follow-up were available for all but one patient. Nine patients were contacted by telephone to determine clinical outcome. The median duration of follow-up did not differ between the groups. At last follow-up, 64% of patients with a JCH had a good clinical outcome, compared to 45% of patients with another type of intracerebral haemorrhage (p>0.1). Mortality did not differ significantly between the groups.

<table>
<thead>
<tr>
<th></th>
<th>JCH n=14</th>
<th>Other ICH n=39</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Duration of admission (days, IQR)</strong></td>
<td>14 (5-34)</td>
<td>9 (4-20)</td>
<td>NS</td>
</tr>
<tr>
<td>Anticoagulation</td>
<td>93%</td>
<td>87%</td>
<td>NS</td>
</tr>
<tr>
<td>Endovascular thrombolysis</td>
<td>43%</td>
<td>23%</td>
<td>NS</td>
</tr>
<tr>
<td>Decompressive hemicraniectomy</td>
<td>7%</td>
<td>28%</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Clinical outcome</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration of follow-up (months, IQR)</td>
<td>13 (4-68)</td>
<td>9 (5-60)</td>
<td>NS</td>
</tr>
<tr>
<td>mRS 0-1</td>
<td>64%</td>
<td>45%</td>
<td>NS</td>
</tr>
<tr>
<td>Mortality</td>
<td>14%</td>
<td>26%</td>
<td>NS</td>
</tr>
</tbody>
</table>

JCH = juxtacortical hemorrhage, ICH = intracerebral hemorrhage, IQR = interquartile range, NS = not significant.

**Specificity of juxtacortical haemorrhages for cerebral venous thrombosis**

The control group of patients of similar age with an intracerebral haemorrhage not related to CVT consisted of 196 patients. Sixty-six patients (34%) had a lobar
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hematoma and in the remaining patients the hematoma was located in the basal ganglia or deep white matter. Sixty-three patients (32%) had extension of the haemorrhage into the ventricles. Three of the 196 control patients (2%) had a haemorrhage which fulfilled the criteria for a JCH as defined above. Therefore, the specificity of a JCH for CVT is 1-(3/196) = 0.98 (95% CI 0.95 – 1.0). The sensitivity is of course low: 0.26 (95% CI 0.16 – 0.41).

Representative cerebral images of the 3 control patients are shown in figure 3. The first patient was a 58-year old woman with an acute left sided hemiparesis due to an intracerebral haemorrhage in the right hemisphere (figure 3A). Conventional angiography showed a small arteriovenous malformation, which was treated with embolization. The second patient was a 58-year old man with a history of hypertension who had suffered from an episode of amnesia, initially interpreted as a transient global amnesia. CT scan of the brain showed a small right-sided JCH (figure 3B). Despite ancillary investigations, an underlying cause of the haemorrhage was not identified. The third patient, a 49-year old woman with a history of breast cancer, developed acute weakness of the left arm and leg. CT scan of the brain showed a JCH in the right hemisphere and the patient was diagnosed with a haemorrhagic stroke (figure 3C). Follow-up MRI was performed 2 weeks later to rule out metastatic disease, which was not identified. Re-evaluation of these images during the current study, however, clearly showed a hyperintense signal in the superior sagittal sinus both on T1 and T2 weighted images, indicating cerebral venous thrombosis was the cause of the haemorrhage (figure 3D).

Discussion

This is the first report on juxtacortical haemorrhages in cerebral venous thrombosis. Our data indicate that small juxtacortical haemorrhages are a characteristic feature of cerebral venous thrombosis and that these haemorrhages are rarely encountered in other conditions.

An unexpected result of our study is that JCHs are significantly associated with thrombosis of the superior sagittal sinus. This characteristic may provide an insight into the origin of these haemorrhages. The venous drainage of the brain
Three patients from the control group (i.e. without CVT) had a hemorrhage indistinguishable from a JCH. A: axial NCCT of a 58 year old woman with a right sided intracerebral hemorrhage due to an arteriovenous malformation. B: axial NCCT of a 58 year old man with a right-sided hemorrhage of unknown origin. C: axial NCCT of a 49 year old woman with a JCH in the right hemisphere initially interpreted as a hemorrhagic stroke. D: axial T1 weighted MRI of the same patient as in C, which shows a hyperintense signal in the superior sagittal sinus (arrow), indicating CVT was the cause of the hemorrhage.

An unexpected result of our study is that JCHs are significantly associated with thrombosis of the superior sagittal sinus. This characteristic may provide an insight into the origin of these haemorrhages. The venous drainage of the brain is anatomically divided into a deep and a superficial system.\textsuperscript{11,12} The superficial system drains blood from the cortex and the outer 1 or 2 centimetres of white matter (figure 4). Blood is collected in subcortical and intracortical veins and subsequently transported through pial veins towards cortical veins. Cortical veins mainly drain into the superior sagittal sinus. The area of drainage of the
Small juxtacortical hemorrhages in CVT

Subcortical veins coincide with the location of the JCHs. These subcortical veins are subdivided into 3 segments, one of which is the arcuate segment. These arcuate veins run parallel to the arcuate fibres (U-fibres) in the white matter and thus follow the curvature of the cortex, similar to the JCHs. Both the location and the shape of JCHs coincide with these arcuate segments of the subcortical veins. Therefore, it seems likely that this is the anatomical origin of JCHs in CVT.

To explain the pathophysiology of JCHs we need to know why thrombosis of the superior sagittal sinus causes haemorrhages to occur at this specific location. Thrombosis of the superior sagittal sinus causes an outflow restriction of the superficial venous drainage of the brain, which leads to an increase in pressure in the more proximally located veins. One possibility could be that the arcuate segments of the subcortical veins differ anatomically from other parts of the superficial drainage system, which may make them more susceptible to rupture in response to increased pressure. We did not find any data which support this hypothesis in the literature on the anatomy of the cerebral venous system. An alternative explanation is that the veins in the cortex are more resistant to rupture because the cortex itself has a more dense structure, which may provide resistance against the increased pressure inside the veins. In contrast, the white matter may have a looser structure, making veins in the white matter the weakest link in the superficial venous system, and hence the most susceptible to rupture. A third possibility is that the actual site of the haemorrhage is the anastomotic medullary veins that connect the superficial and deep venous systems. The existence of these anastomotic veins has been shown in several studies. Indeed, if anastomotic veins did not exist, thrombosis of the superior sagittal sinus with occlusion of cortical veins would cause a complete arrest of the blood flow in the cortex, which would cause more extensive neurological deficits than is generally seen in patients with CVT. Restriction in outflow through the superficial system results in recruitment of the anastomotic veins, and hence to an increased pressure in these veins. A paucity of anastomotic veins has been associated with a larger increase in diameter in response to a local increase in pressure. Data from a rat model of CVT suggest that the extent of the venous collateral circulation is an important factor in determining whether occlusion of the superior sagittal sinus leads to parenchymal damage. Hence, one could hypothesize that the smaller the number of anastomotic veins, the more likely they are to rupture as a result of thrombosis of the superior sagittal sinus.
Figure 2 Neuropathological findings

Macroscopic coronal section of the brain showing a concave shaped JCH, located in the left hemisphere (arrow). Small cortical petechial hemorrhages are also present (arrowhead).

In our cohort of patients with CVT, a JCH at baseline was not uncommon (14 out of the original 114 patients, 12%). It is, however, unlikely that this percentage is representative for CVT patients in general. As a tertiary referral hospital, we receive patients in a severe clinical condition, who more often have an intracerebral haemorrhage. The sensitivity of a JCH is anyhow too low to exclude the diagnosis of CVT. More interesting is its high specificity (98%) in non-traumatic, non-aneurysmatic cerebral parenchymal haemorrhage in patients under the age of 60. Of the 196 control cases with an intracerebral haemorrhage but no CVT, only three had a JCH. The fact that, on reassessment, one of these three patients was found to have CVT further strengthens our conclusion: if a small JCH is found on a standard NCCT scan in a patient under the age of 60, CVT is by far the most likely diagnosis.
The baseline clinical condition of our patients with a JCH was similar to those with other types of intracerebral haemorrhage caused by CVT. This is surprising since, by definition, the volume of the haemorrhagic lesions was much larger in the latter group. In addition, larger haemorrhagic infarcts in CVT are usually accompanied by significant vasogenic oedema which often results in brain tissue displacement. Thus, one would expect patients with a JCH to be in a better clinical condition compared to those with larger haemorrhages. The fact that this is not the case is consistent with the hypothesis that the presence of JCHs
signifies poor collateral venous pathways. The higher frequency of papilledema in CVT patients with a JCH in our study also points in this direction. Indeed, some studies have suggested a pivotal role of the failure of venous collaterals and the development of severe neurological deficits.²⁰⁻²²

In conclusion, we have shown that small juxtacortical haemorrhages are characteristic for cerebral venous thrombosis and associated with thrombosis of the superior sagittal sinus. Physicians should be aware that the finding of a juxtacortical haemorrhage indicates that cerebral venous thrombosis is the most likely diagnosis.
References


