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Case Report

Cerebral venous sinus thrombosis complicated by subdural hematomas: Case series and literature review

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Abstract

Background: Cerebral venous sinus thrombosis (CVST) can cause elevated intracranial pressure, hemorrhagic venous infarct, and cortical subarachnoid hemorrhage. We present a case series and literature review to illustrate that CVST can also present with subdural hematoma (SDH).

Case Description: Chart review was completed on a retrospective case series of CVST with spontaneous SDH. We also conducted a literature search. Over a 6 year interval, three patients with CVST and SDH were admitted to the neurointensive care unit. A 38-year-old woman had both SDH and a hemorrhagic venous infarct associated with a transverse sinus thrombosis. She was managed conservatively with long-term anticoagulation. A 68-year-old woman presented with an acute SDH requiring craniotomy and a thrombosed cortical vein was noted intraoperatively. Computed tomography venography showed thrombosis of the superior sagittal sinus. She had polycythemia vera with the V617 Jak2 gene mutation and was managed with aspirin and hydroxyurea. A 60-year-old male had recurrence of a spontaneous convexity SDH requiring reoperation. Neuroimaging identified ipsilateral transverse sinus thrombosis with retrograde flow into the opposite sinus. Manometry demonstrated elevated venous pressures and these normalized after thrombectomy. Angiography performed after endovascular treatment demonstrated a normal venous drainage pattern. There have been limited reports of SDH complicating CVST in the literature.

Conclusion: This case series and literature review demonstrates that CVST can also present with spontaneous SDH with or without associated venous infarctions. Treatment must be individualized. This is the first published description of endovascular thrombectomy for recurrent symptomatic SDH due to CVST.

Key Words: Cerebral sinus thrombosis, cerebral venous thrombosis, embolectomy, polycythemia vera, subdural hematoma, stroke

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INTRODUCTION

Cerebral venous sinus thrombosis (CVST) is an uncommon cause of stroke.[20] The most common clinical presentations reported in large case registries are intracranial hypertension and hemorrhagic cerebral venous infarctions.[5,14,20] The intracranial hypertension is caused by impaired venous drainage. The hemorrhagic cerebral venous infarctions are attributed to reduced cerebral blood flow due to venous outflow obstruction with associated edema and hemorrhage. CVST can also present with isolated cortical subarachnoid hemorrhage (SAH). Most patients with CVST are under the age of 50 years. There are extensive lists of associated conditions that predispose patients to CVST.[20] Systemic conditions that increase the risk of CVST include genetic conditions, pregnancy, hormonal therapies, malignancy, polycythemia vera and other myeloproliferative disorders, dehydration, or trauma. Mechanical factors, which reduce blood flow in the cerebral sinuses and promote thrombosis, include adjacent infections (typically mastoiditis), neoplastic invasion of the sinus, trauma, and neurosurgical procedures.

The increasing availability of magnetic resonance imaging (MRI), MR venography and computed tomography (CT) venography has improved our ability to detect other clinical manifestations of CVST including subdural hematoma (SDH)[3,13,25,27] and cortical SAH.[2,7,16,18,19,21] It is interesting that none of the 624 patients enrolled in the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT) were reported to have SDH or cortical SAH. As most participants in the ISCVT were neurologists, this may have restricted enrollment of CVST presenting with these other forms of intracranial bleeding. In our tertiary care neurointensive care unit, patients receive treatment under a multi-disciplinary team, which includes neurocritical care, neurosurgery, and interventional neuroradiology. We reviewed our inpatient neurointensive care registry for CVST complicated by SDH and also completed a literature review.

MATERIALS AND METHODS

Under institutional review board (IRB) approval, a prospective inpatient neurosurgery database is maintained that records patients admitted to the neurointensive care unit since 2006. We reviewed this database for CVST and SDH from 2006 to 2012 and identified three patients. We then completed retrospective chart review for patients diagnosed with CVST and SDH. Using PubMed, a literature search was completed for CVST and SDH.

Case summaries

Case 1

A 38-year-old woman on oral contraceptives presented with headache and difficulty speaking. She had no other significant past medical history. A left frontal SDH and left temporal venous infarct with a left transverse sinus thrombosis was demonstrated on CT and MRI [Figure 1]. A hypercoagulable work-up did not reveal any additional thrombophilias. She was initially managed with systemic anticoagulation using intravenous heparin. After serial neurologic examinations and repeat CT, head imaging demonstrated no further bleeding; she was transitioned to oral warfarin and discharged home. She had excellent neurologic recovery (modified Rankin score 0) at 3 months follow up visit.

Case 2

A 68-year-old woman with polycythemia vera experienced a global, nonpositional headache for one day. She subsequently became comatose. On emergent evaluation she exhibited decorticate posturing. Her Glasgow coma score was 6. There was no preceding history of trauma, infection, or dehydration. She was not on anticoagulants or antiplatelet agents. Her polycythemia vera had been managed with therapeutic phlebotomy and hydroxyurea (500 mg daily). She had no prior history of thromboembolic events. Noncontrasted head CT showed extensive left 20 mm acute SDH with 16 mm of midline shift [Figure 2a]. She underwent emergent craniotomy and hematoma evacuation. On careful inspection of the exposed brain, a prominent and engorged cortical vein observed. No other vascular abnormalities or potential bleeding sources were identified. CT venography and MR venography detected partial superior sagittal sinus thrombosis, which leads to the empty delta sign (the intraluminal thrombus prevents central filling of the sinus; Figure 2b). She was managed conservatively with hydroxyurea, hydration, and aspirin. Laboratory analyses were notable for normal coagulation panel,

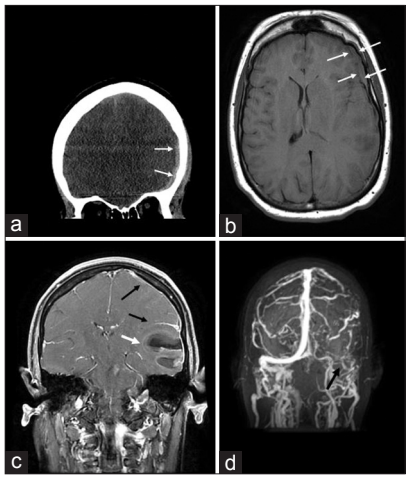

Figure 1: Case 1. A 38-year-old woman on oral contraceptives presented with headache and difficulty speaking. She had no other significant past medical history. A left frontal subdural hematoma (white arrows, a, b; black arrow, c) and left temporal venous infarct (white arrow, c) with a left transverse sinus thrombosis (black arrow, d) was demonstrated on CT (a) and MR imaging (b, c, d). A hypercoagulable work-up did not reveal any additional thrombophilias

normal platelet function assays, hematocrit of 45.9, negative thrombophilia evaluation (protein S, protein C, antithrombin III, anticardiolipin antibody, homocysteine, lupus anticoagulant, beta-2 glycoprotein antibodies, Factor V Leiden mutation, prothrombin gene mutation). Analysis of DNA extracted from her blood revealed the presence of the V617F mutation within the JAK2 gene. Her 3-month outcome was poor (modified Rankin score 4).

Case 3

A 60-year-old male experienced one month of escalating bifrontal headaches refractory to analgesics and antibiotics prescribed for presumed sinusitis. The headaches increased during sneezing, bending over, or bedrest and improved when he was sitting or standing.

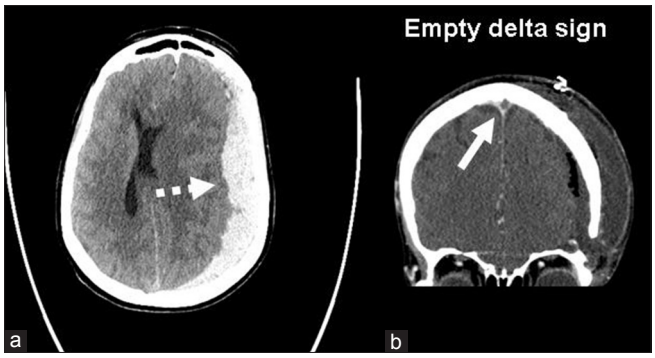

Figure 2: Case 2. A 68-year-old woman with polycythemia vera experienced a severe headache and then deteriorated to coma. At the time of craniotomy, the neurosurgeon observed a thrombosed cortical vein following evacuation of the subdural hematoma. A coronal CT head (a) demonstrates a left acute subdural hematoma (dashed arrow). CT venogram (b) is notable for the empty delta sign (solid arrow) due to intraluminal thrombus preventing normal opacification of the sagittal sinus. Genetic testing demonstrated the V617F mutation within the JAK2 gene

There was no history of trauma. His prior medical history was notable for hypertension and hyperlipidemia. There was no family history of coagulopathy or thrombophilia. He was married, employed, used 0.5 packs of tobacco (7.5 packs/years), consumed alcohol socially, and did not use any illicit drugs. His vital signs were notable for sinus bradycardia with a heart rate of 39 and elevated BP of 184/86 mmHg. He had no other significant findings on physical examination; detailed neurologic examination was unremarkable. Routine admission laboratory studies were within normal limits. Coagulation studies including platelet function assays were normal.

Noncontrasted head CT demonstrated a 19 mm left isodense SDH with 10 mm of midline shift [Figure 3a]. The collection was more prominent in the frontal region. A CT angiogram was obtained prior to surgery, and no source of bleeding was identified. He underwent a mini-craniotomy; and repeat CT imaging after the procedure [Figure 3b] demonstrated excellent hematoma evacuation and brain reexpansion.

A scheduled follow up head CT obtained on postoperative day 27 was notable for recurrent SDH with a frontal predilection [Figure 3c]. The appearance and location were similar to the initial presentation. He was readmitted to the hospital and underwent SDH drainage via burr hole. On subsequent imaging with MRI, MR angiography, and MR venography, a filling defect was visible in the distal left transverse sinus and no flow was observed in the distal left sigmoid sinus or internal jugular vein. These findings were confirmed on CT venography.

Patient underwent cerebral angiography on postoperative day 7 after SDH evacuation [Figure 4]. Left carotid injection was notable for a prominent left vein of

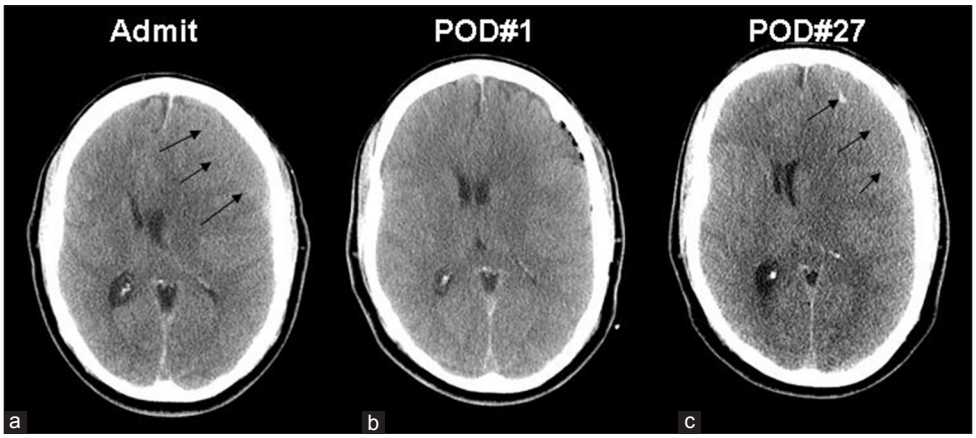

Figure 3: Case 3. A 60-year-old male presented with one month of escalating headaches and no history of trauma or use of antiplatelets or anticoagulants. CT head demonstrated a left isodense subdural hematoma (a). This was evacuated surgically with mini-craniotomy. The initial imaging on postoperative day one (POD#1, b) showed excellent radiographic results. Repeated imaging on postoperative day 27 (POD#27, , c) showed recurrence of the left isodense subdural hematoma

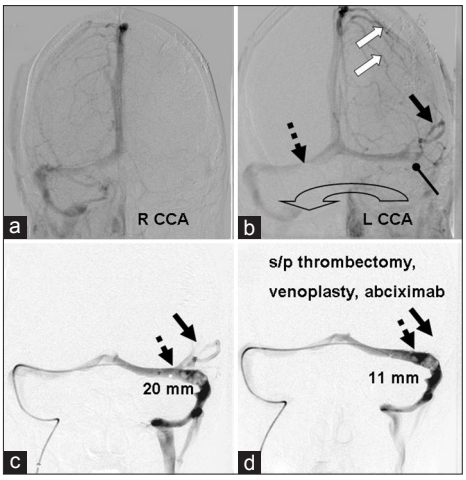

Figure 4: Case 3. Catheter angiography (a and b) and venography (c and d) were performed. Right common carotid artery injection shows normal venous draining pattern (a). Left common carotid injection (b) shows stagnate flow in the vein of L abbe (black arrow), retrograde drainage via the right transverse sinus (dashed and open arrows), and a filling defect (black circle). Selective venography of the left transverse sinus (c) demonstrates back-filling of the vein of Labbe (black arrow) and elevated pressures. Following endovascular treatment (d), normal venous drainage and pressures are restored

Labbe with delayed wash-out, a filling defect in the left transverse sinus, and retrograde flow in the left transverse sinus with subsequent drainage into the right transverse sinus. Transvenous catheterization confirmed retrograde drainage of the left transverse sinus and venous stasis in the left vein of Labbe. Manometric readings in the left sigmoid sinus and both transverse sinuses were elevated (18-25 mmHg range). Thrombectomy with the Penumbra device and venoplasty with 7 × 20 mm balloon were performed; 10 mg of systemic abciximab was administered. Follow up venography demonstrated improved flow in the left vein of Labbe, restored normal direction of flow in the sinuses with normalized pressure (11 mmHg) in the left transverse sinus. Daily aspirin (325 mg) was initiated on the day of this intervention; patient was discharged home without neurologic deficits. A follow-up CT venogram at 6 weeks demonstrated patent venous sinuses and no recurrence of his SDH. His 3 month clinical outcome was excellent (modified Rankin score 0).

DISCUSSION

Diagnosis of CVST is challenging given the variability in presenting symptoms and signs. It is widely accepted that CVST may present with elevated intracranial pressure (ICP) and hemorrhagic venous infarcts.[20] There is growing evidence that it can cause SDH [Table 1] and focal cortical SAH as well.[2,7,16,18,19,21] Our case series provides additional support for the association of CVST and SDH.

A literature review was completed, and we identified seven case reports [Table 1]. The earliest report of SDH complicating CVST was published by Bucy *et al.* in 1942.[1] They describe a young male with history of recurrent venous thromboses who developed seizures. Ventriculogram demonstrated a left brain mass and craniotomy identified an acute SDH with associated cortical vein thrombosis. These authors attributed the obstructed venous outflow due to venous thrombosis as the precipitating factor for the SDH. Many of the patients with SDH complicating CVST had underlying hypercoagulable states. Whether the SDH was caused by the CVST can be contested in a few of these reports. The case report by Matsuda *et al.*[4] diagnosed SDH in a delayed fashion after the patient was initially treated for CVST using systemic thrombolytics. Therefore it is more likely that the SDH was a complication of the treatment with thrombolytics. The case report by Takamura *et al.*[28] is suspicious for a dural arteriovenous fistula (AVF) arising secondary to chronic CVST. They state that the angiogram demonstrating "complete occlusion of the anterior segment of the superior sagittal sinus and the right cavernous sinus, and segmental occlusion of the left transverse sinus" and "… multiple dilated veins as small enhanced lesions adjacent to the dura mater." Therefore, the bleeding source for the SDH could be due to a dural AVF rather than the CVST. The case by Singh *et al.*[25] had initial radiographic findings demonstrating bilateral hygromas in a young woman suffering from headaches. She later developed CVST and SDH. This sequence of events suggests an alternative explanation. She may have initially developed spontaneous intracranial hypotension causing headaches and bilateral hygromas with subsequent formation of SDHs. It well established that both CVST and SDH can arise as a secondary complication of intracranial hypotension.[6,8,12,15,22-25,29,30]

Many patients with SDH complicating CVST have underlying thrombophilias. Once the need for neurosurgical intervention has been clarified, some patients may require cautious treatment with anticoagulation. The presence of thrombophilias is generally established based on personal and family history and selective laboratory testing. The first patient that we presented had increased risk of venous thrombosis due to oral contraceptive agents. Our second patient was at risk of cerebral venous thrombosis due to polycythemia vera and her case shares many features with the prior report of a patient with polycythemia vera complicated by CVST and SDH.[26] Also a higher venous thrombosis risk has been reported in patients with myeloproliferative disorders and the V617F JAK 2 mutation and this mutation was present in our patient as well.[9,11] We were unable to identify a risk factor for our third patient. An evaluation for risk factors for cerebral venous thrombosis should include screening for thrombophilias including: Medications such as anabolic or sex hormones and L-asparaginase, deficiencies in protein C, protein S, antithrombin III; genetic mutations in the prothrombin gene (G20210A

Table 1: Literature review of cerebral venous sinus thrombosis and subdural hematomas

Reference	Age (years)/sex	Description	Treatments	Comments
Bucy *et al*., 1942	37 M	Seizures, headache, papilledema, ventriculogram showing a left mass, acute left subdural hematoma evacuated, cortical venous thrombosis directly visualized	Craniotomy, subdural hematoma evacuation, subtemporal decompression	Prior history of multiple and recurrent deep venous thrombosis; the proposed mechanism for the SDH was due to cerebral venous bleeding due to obstructed venous return
Matsuda 1982	33 F	CVST of sagittal sinus (SS) treated first with thrombolytics; then mixed density convexity SDH developed	Intravenous thrombolytics; burr hole for subsequent development of SDH	Preceding use of thrombolytics increases the likelihood that this is an iatrogenic subdural hematoma
Takamura 1996	35 M	Chronic subdural hematoma on CT, catheter angiogram with CVST of SS, right cavernous sinus, left transverse sinus, multiple torturous collateral vessels	Recurrent SDH after burr hole; craniotomy and electrocautery of dura and outer SDH membrane	The authors' description of angiogram suggests a dural arteriovenous fistula, which can develop following CVST
Chu 2001	40 F	Papilledema, bilateral VI n deficits; MRI and MRV with L SDH, CVST of L Sylvian vein, SS, L transverse sinus	Cessation of oxymetholone	Synthetic androgen, oxymetholone given for aplastic anemia; no anticoagulation due to chronic thrombocytopenia
Singh 2005	39 F	Initially bifrontal hygromas and HA; subsequently developed bilateral chronic SDH and straight sinus thrombosis	Systemic anticoagulation	Some elements suggest intracranial hypotension was the initial condition with secondary CVST and secondary SDH
Sirin 2010	77 M	Cranial and spinal SDH, hemorrhagic venous infarct by MRI, and CVST of SS, transverse sinus, sigmoid sinus on catheter angiogram	Phlebotomy, hydroxyurea; delayed institution of anticoagulation due to relapse	Polycythemia vera, JAK2 mutation+
Takahashi 2012	55 M	Acute onset HA while weight lifting. Acute SDH and CVST of SS by MRI and catheter angiography	Anticoagulation	Prominent Sylvian vein providing collateral venous drainage

CVST: Cerebral venous sinus thrombosis, CT: Computed tomography, SDH: Subdural hematoma, MRI: Magnetic resonance imaging, SS: Sagittal sinus, HA: Headache

mutation) and Factor V gene (Leiden mutation); anticardiolipin antibodies; antibeta-2-glycoprotein I antibodies; antiphosphatidylserine antibodies; lupus anticoagulant; homocyteinemia related to mutations in the methyltetrahydrofolate reductase gene (C677T and A1298C mutations); and hemoglobinopathies such as sickle cell disease. When thrombophilias are diagnosed, then we recommend consultation with hematologists to coordinate timing and duration for anticoagulation. Cerebral venous thrombosis may arise from other clinical factors such as pregnancy, puerperium, malignancies, dehydration, tumor invasion, local infections such as mastoiditis, and trauma. About one-third of patients with CVST do not have a thrombophilia or contributing condition identified.[20]

Our manometric measurements before and after endovascular treatment contribute novel information to the pathophysiology of SDH complicating CVST. The hemodynamic consequences of CVST found in our third patient included: Flow reversal in the transverse sinus, venous stasis, delayed drainage of the vein of Labbe, and elevated venous pressures. While the precise source of subdural bleeding was not visible at the time of surgery, we attribute the SDH venous bleeding caused by cortical venous hypertension and associated venous engorgement. The initial evacuation of the hematoma allowed for short-term radiographic improvement but may have reaccumulated to provide a tamponading effect on the bleeding source. Once the CVST was identified and treated using a novel endovascular approach, he had a satisfactory recovery and no additional recurrences of his SDH. Our proposed mechanism of SDH arising from CVST (venous engorgement and hypertension) is analogous to the mechanism of SDH arising from dural AVFs.[10,17] Venous hypertension develops initially and then venous bleeding occurs. Bleeding stops as the SDH accumulates and tamponades the site of venous bleeding. This is the first published report of endovascular treatment of CVST for recurrent SDH to our knowledge.

While the cornerstone of treatment for CVST complicated by elevated ICP or venous infarction is anticoagulation,[20] the management of CVST complicated by SDH or SAH is not well established and therefore controversial. The rationale behind anticoagulation for CVST is to facilitate recanalization and to prevent further venous thrombosis. The use of anticoagulation for patients with hemorrhagic infarct and CVST has gained acceptance.[20] In patients with small SDHs (with or without associated intracerebral hemorrhage), there are no clear guidelines for patient management. There

are two prior case reports of medically managed SDHs that were placed on systemic anticoagulation [Table 1], and we also successfully managed our first patient with systemic anticoagulation. Patients should be hospitalized at a facility with neurosurgical support with frequent neurologic, laboratory, and radiographic monitoring, when systemic anticoagulation is initiated.

In contrast, we recommend against early initiation of full anticoagulation (e.g., heparin and heparinoids) for patients who require neurosurgical intervention for intracranial bleeding complicating CVST. Our second and third patients were symptomatic from the SDH and required surgical intervention. We did not anticoagulate these surgical patients but did provide hydration and antiplatelet agents. The recurrence of the SDH in patient 3 illustrates the risk of rebleeding if the underlying CVST is not recognized and treated. At our center, we reserve full anticoagulation after craniotomy for a minimum of 2 weeks in uncomplicated cases and often extend this interval based on case-specific details such as risks of delayed neurologic decline, underlying neuropathology, or need for additional surgeries. When anticoagulation is recommended following intracranial procedures, we avoid bridging therapy (heparin, heparinoids) and start warfarin unless there are other compelling features such as pulmonary embolus with hemodynamic compromise. While we did start patients 2 and 3 on aspirin, the benefit of antiplatelet agents on venous thrombosis is negligible. We did not identify any reported cases of CVST and surgically managed SDH treated with systemic anticoagulation [Table 1].

It is important to recognize that SDH and CVST may both arise as a rare complication of intracranial hypotension. It is well known that intracranial hypotension can cause SDH due to sagging brain structures and venous engorgement.[6,8,12,15,22-25,29,30] The secondary cerebral venous engorgement due to the low ICP[22] and associated stasis[30] can lead to secondary CVST. Orthostatic headaches should increase concern for intracranial hypotension. The pattern of headache will change to a more constant pattern when secondary complications of intracranial hypotension arise such as SDH and at times CVST.[8,12] Clinicians should also seek out information that would increase suspicion for intracranial hypotension such as recent lumbar punctures, lumbar drains, spine surgery, trauma, and epidural anesthesia. Our three patients had neither a clinical history nor radiographic findings concerning for intracranial hypotension. When SDH arise from intracranial hypotension, the cornerstone of treatment should address the cerebrospinal fluid (CSF) leak, typically through epidural blood patches.[23] Whether surgical evacuation of the SDH is also required must be determined based on the clinical circumstances and can be hazardous.[4]

In conclusion, CVST may present with spontaneous SDH with or without associated hemorrhagic infarctions. With the rising clinical access to noninvasive MR venography and CT venography, we anticipate that the diagnosis of CVST complicated by SDH and cortical SAH will increase. Management of patients with SDH complicating CVST is complex due to contraindications for anticoagulation in patients with symptomatic SDH. Surgical intervention for SDH with mass effect appears to be well tolerated. Endovascular thrombectomy of the CVST for patients with recurrent SDH is an emerging treatment option to treat the underlying venous hypertension and avoiding the bleeding risks of systemic anticoagulation. Clinicians must also remain attentive to the possibility that the SDH and CVST arise as a delayed complication of intracranial hypotension, as the treatment approach must incorporate treatment for a CSF leak.

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