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Following Strangulation

To the Editor: Cerebral venous thrombosis (CVT) is a rare type of stroke, with an annual incidence of around 1/100,000.¹ Signs and symptoms are those associated with increased intracranial pressure due to impaired venous drainage and focal neurologic deficits resulting from venous ischemia or hemorrhage.^{2,3} The first group of symptoms includes headache (the most common symptom and present in around 90% of patients⁴) and papilledema. Possible focal neurologic signs of CVT include hemiparesis and aphasia. Epileptic seizures are also a common symptom of CVT, occurring in around 40% of patients.⁵

CVT is associated with a diverse range of etiologic factors. These factors include prothrombotic conditions like Factor V Leiden mutation, pregnancy, use of oral contraception, cancer, parameningeal infections (eg, sinusitis), and dehydration among others.^{3,4} Although venous stasis following strangulation has been hypothetically proposed as a possible cause of CVT, there has been no report to date of a patient in whom CVT occurred after strangulation.

Case report. A 75-year-old man was transferred to the psychiatric emergency department after an attempt to strangle himself with a belt. The patient suffered from previously uncharacterized dementia and depressive symptoms. His medical history included pulmonary embolism several years ago, atrial fibrillation, and chronic renal failure. His family history was unremarkable.

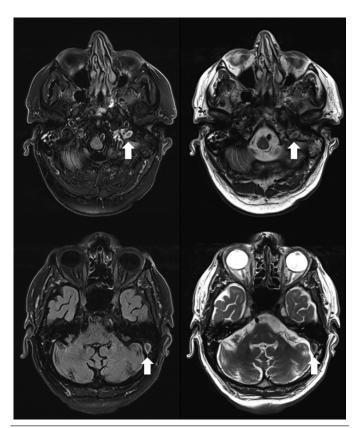
The patient had been treated for atrial fibrillation with enoxaparin subcutaneously in a dose adjusted to the renal function (70 mg/d). Anticoagulant treatment with rivaroxaban had been recently replaced with enoxaparin due to renal failure.

The patient's physical examination was remarkable for a strangulation mark on the left side of the neck. Neurologic examination revealed impaired cognitive function but no new neurologic deficit. The Mini-Mental Status Examination (MMSE)⁶ revealed a score of 14/30. Preexisting neurologic symptoms included dysarthria, mild facial asymmetry, and reduced coordination.

Laboratory workup confirmed renal failure (glomerular filtration rate 21 mL/min/1.73 m²). Duplex ultrasound provided no evidence of carotid dissection. Electroencephalogram results showed diffuse slowing. Two days later, cranial magnetic resonance imaging (MRI; Siemens TrioTim, 3 Tesla) for workup of dementia showed severe cerebral microangiopathy (Fazekas score of 3) indicating vascular dementia. As an incidental, unexpected finding, the MRI revealed a fresh thrombosis in the left transverse and sigmoid sinuses (Figure 1) without hemorrhage or edema in the adjacent brain tissue. A clinical and laboratory workup for causes of thrombosis was remarkable only for hyperhomocysteinemia (30.7 μ mol/L; normal range, < 9 μ mol/L).

The patient's anticoagulant treatment was switched to oral phenprocoumon. Eventually, the patient was transferred back to the nursing home after remission of depression.

Venous stasis due to compression of the jugular vein is the most plausible cause of CVT in our patient, while potentially Figure 1. Magnetic Resonance Imaging Showing New Cerebral Venous Sinus Thrombosis in Left Sigmoid Sinus (top row) and Left Transverse Sinus (bottom row) Marked by White Arrows^a



^aFluid-attenuated inversion recovery (FLAIR) images are shown in the left column; the right column contains T2-weighted images. MRI was performed using a Siemens TrioTim, 3 Tesla.

predisposing factors were hyperhomocysteinemia²⁻⁴ and recent change of the anticoagulant regimen.

Case reports have suggested that CVT may result from stasis due to venous dilation: decreased intracranial pressure (due to spontaneous intracranial hypotension^{7–9} or lumbar puncture¹⁰) may lead to dilation of cerebral veins and thus reduced blood flow velocity, contributing to the risk of CVT. Mechanical obstruction of venous drainage will also contribute to stasis and is a plausible etiologic factor. However, the evidence for this relationship is limited. In a large study⁴ of CVT, jugular catheter occlusion was reported in 0.8% of patients, and case reports^{11,12} have found CVT secondary to internal jugular vein thrombosis in the presence or absence of a central venous catheter.

The incidence of CVT after strangulation may be broadly underestimated especially in older patients, since clinical presentation may be atypical, with altered mental status as the prominent symptom.¹³ This case report demonstrates that CVT should be included in the differential diagnosis in patients who develop otherwise unexplained neurologic or neuropsychological symptoms after strangulation.

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Letter to the Editor It is illegal to post this copyrighted PDF on any website Association Stroke Council and the council on Epidemiology and opyrighted sinus and simultaneous pulmonary embolism. *Tress Med Sci.*

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Funding/support: None.

Patient consent: Consent was received from the patient to publish this report, and information has been de-identified to protect anonymity.

Published online: June 21, 2018.

Prim Care Companion CNS Disord 2018;20(3):17l02198

To cite: Clemm von Hohenberg C, Kerl U, Sartorius A, et al. Cerebral venous thrombosis following strangulation. *Prim Care Companion CNS Disord.* 2018;20(3):17102198.

To share: https://doi.org/10.4088/PCC.17l02198

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