

## Case Report

# Hemorrhage of a cavernous malformation associated with accidental electrocution: Case report and review of the literature

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## Abstract

**Background:** Cavernous malformations (CMs) are the second most common intracranial vascular lesions. They typically present after hemorrhage or as incidental findings. Several risk factors have been identified for hemorrhage, however, electrocution as a cause has not been described. We performed a literature review of electrocution associated with CM hemorrhage and of the mechanisms of pathological injury in the central nervous system (CNS) secondary to electrocution. We found no cases of hemorrhage of CMs associated with electrocution.

**Case Description:** A 19-year-old male electrician was accidentally electrocuted with 277 V of alternating current (AC) at a job site. He suffered no trauma or physical injuries and reported no immediate abnormal findings. He then experienced progressive nausea, emesis, and lethargy until he presented to the emergency department (ED) where it was discovered that he had a left thalamic/midbrain hemorrhage with hydrocephalus. His hydrocephalus was treated and he began to improve. Subsequent magnetic resonance imaging (MRI) of his head demonstrated characteristic features of a CM.

**Conclusions:** There are several proposed mechanisms in the literature by which electrocution may cause CNS damage. It is conceivable that given the pathology of CMs and the proposed mechanisms of electrical injury, these lesions may have an increased risk of hemorrhage as result of electrocution and we are reporting the first case of such an association.

**Key Words:** Cavernoma, cavernous malformation, electrocution, hemorrhage

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## INTRODUCTION

Cavernous malformations (CMs) or cavernomas are the second most common intracranial vascular lesions and typically present after hemorrhage or as incidental findings. They are composed of thin hyalinized vascular

channels without intervening parenchymal tissue. CMs differ from other vascular malformations as they are angiographically occult due to the fact that they do not shunt blood. With the routine use of magnetic resonance imaging (MRI), at least 40% are found incidentally.<sup>[3]</sup> Autopsy and MRI studies now approximate the incidence

to be 0.4-0.8%.<sup>[15]</sup> They have characteristic MRI findings and are frequently described as having a “popcorn-like” appearance on MRI due to the multiple stages of bleeding within them. Several risk factors for hemorrhage such as lesion size, location, and history of a prior bleed have been identified. However, electrocution has not been explored as a risk factor for hemorrhage.

We performed a literature review of electrocution associated with CM hemorrhage and of the mechanisms of pathological injury in the central nervous system (CNS) secondary to electrocution. We found no cases of hemorrhage of CMs associated with electrocution.

## CASE DESCRIPTION

A 19-year-old male electrician was working at a commercial job site when his right hand came in contact with 277 V of alternating current (AC). He was in contact with the high voltage wire for only a few seconds before it knocked him off his ladder landing on his feet. He did not suffer any trauma and had no obvious physical injuries. The patient reported no abnormal findings, until the next day when he began to feel nauseous and tired. He continued to decline over the next 4 days with progressively increasing nausea, emesis, and lethargy until a friend brought him to the emergency department (ED) because he was somnolent. A computed tomography (CT) examination of his head revealed a left thalamic/midbrain “overt” hemorrhage with hydrocephalus [Figure 1]. An external ventricular drain was placed and he clinically began to improve. However, he was unable to tolerate weaning of his drain and subsequently had a right frontal ventriculoperitoneal programmable shunt placed. MRI of his head demonstrated features typically seen with hemorrhage involving a CM [Figure 2a and b]. Conventional cerebral angiography revealed no abnormal findings. Unfortunately, despite improvement



**Figure 1: Axial CT image without intravenous contrast shows a nodular, high attenuation, intraaxial hematoma in the left thalamus and left cerebral peduncle with localized mass effect**

on MRI [Figure 2c], he has persistent right-sided hyperthesias, dysesthesias, and allodynia consistent with thalamic pain syndrome, also known as Dejerine – Roussy syndrome. Approximately 1 year after his original hemorrhage, he suffered an episode of transient right-sided weakness and worsening of his thalamic pain syndrome. Given his symptoms and CT showing a new slight hyperdensity in the area of his prior bleed the authors suspected he had another small bleed or “ooze” from his suspected cavernoma [Figure 3a and b]. Given this rehemorrhage and critical location it was deemed best to treat this probable CM with stereotactic radiosurgery. Fortunately, this patient has not had any further hemorrhages and has remained relatively stable.

## DISCUSSION

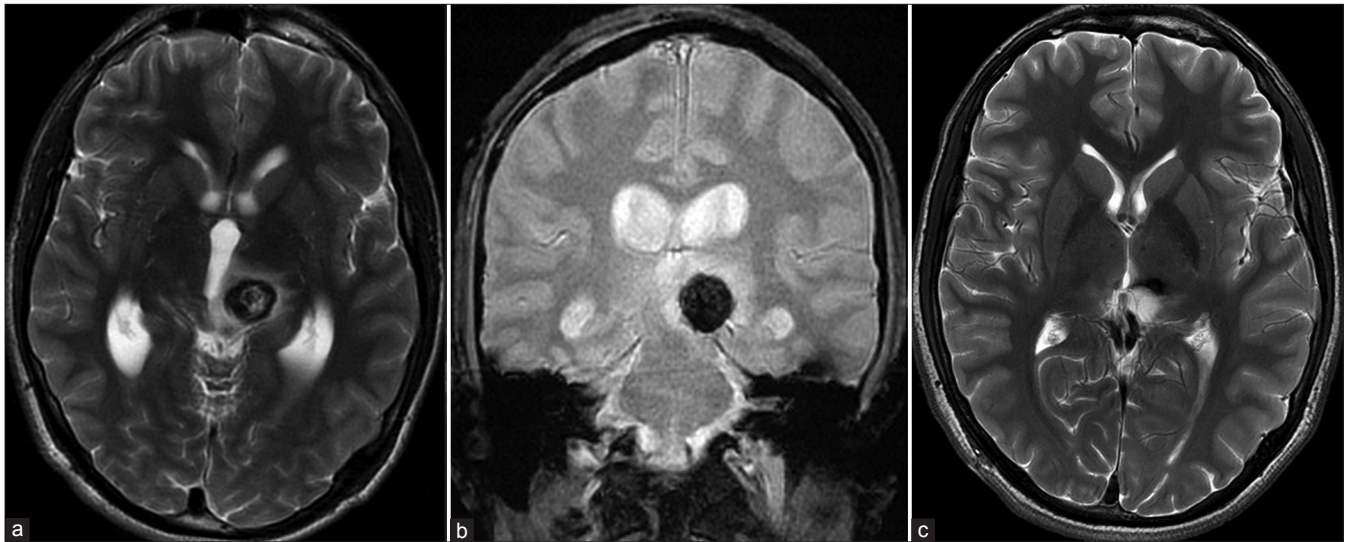
### CM natural history

CMs can occur as sporadic or in familial forms and account for approximately 10-15% of all vascular malformations.<sup>[3]</sup> Familial forms account for 6-50% of CMs and more than 50% of them have multiple CMs while only 2-20% of sporadic cases have multiple CMs.<sup>[3,15]</sup>

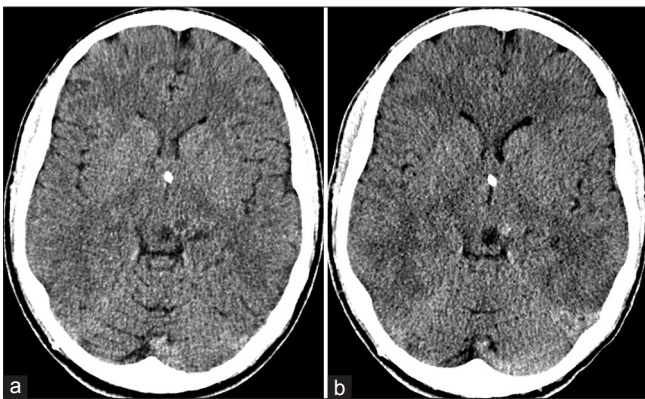
CMs in adults typically present in the fourth and fifth decades of life, whereas in children, there is a bimodal presentation with peaks in children aged 0-2 and 13-16 years.<sup>[6,8]</sup> Presentation is widely varied and is largely dependent upon lesion location. Presentations include headache, seizure, focal neurological deficit, or even death. Many, however, are asymptomatic. Supratentorial lesions most commonly present with seizure while infratentorial lesions typically present with a focal deficit, including hemiparesis, hemisensory deficits, and cranial nerve palsy. Basal ganglia or thalamic lesions commonly present with sensorimotor deficits or less commonly movement disorders or hydrocephalus. Brainstem CMs can present with cardiovascular or respiratory difficulties or even hiccups. Any of the above presentations may present without acute hemorrhage.

Determining rates for hemorrhage is complicated by studies defining hemorrhage differently. Some require a new neurological event with positive imaging findings, while others require only the imaging to be positive. Washing *et al.*<sup>[15]</sup> recently reviewed the natural history of CMs and found that a majority of natural history studies report symptomatic hemorrhage rates ranging from 0.7% to 6% per patient-year. They also reported symptomatic per lesion-year hemorrhage rates ranging from 0.1% to 2.7%.

Aiba *et al.*<sup>[1]</sup> were the first to report that symptomatic hemorrhage negatively impacts the natural history of CMs. They also found that symptomatic hemorrhage rates for those presenting with seizure or incidentally were extremely low, 0.39% and 0% per year per patient,



**Figure 2:** (a) Axial T2-weighted MR image shows the lesion to have a slightly irregular rim of low signal surrounding a central zone with mixed high and low signal. A small poorly defined zone of high signal consistent with edema is seen in the adjacent brain tissue, (b) Coronal gradient echo MR image at the same time as Figure 2a shows a low signal at the site of the hematoma, (c) Axial T2-weighted image 2 months later shows evolutionary changes of the intraaxial hematoma with low signal hemosiderin anterior to a high signal cystic-appearing zone. There is decreased mass effect associated with the lesion



**Figure 3:** (a) Axial CT image without intravenous contrast at the same time as Figure 2c shows a small zone of low attenuation consistent with resolved hematoma and remaining cystic-appearing zone, (b) Axial CT image without intravenous contrast one year after the initial hemorrhage shows a small zone with slightly high attenuation consistent with localized rehemorrhage located anterior to the cystic-appearing zone

respectively. However, those initially presenting with hemorrhage had a much high rehemorrhage rate, 22.9% per year per lesion. Kondziolka *et al.*<sup>[5]</sup> reported similar findings, demonstrating a 0.6% hemorrhage rate per year in patients without a history of hemorrhage and a 4.5% bleed rate per year in patients with prior hemorrhage. Furthermore, the increased risk of rebleeding rating is time limited and is often referred to as “temporal clustering”. This is supported by Barker *et al.*,<sup>[2]</sup> who found that the increased risk of rebleeding was highest in the first 28 months (2.1% per month) then rapidly declined after that and stabilized at 0.8% per month.

Deep and infratentorial CMs also have increased

recurrent symptomatic rehemorrhage rates, while brainstem CMs have particularly poor natural histories with both increased primary and recurrent symptomatic hemorrhage rates. Furthermore, brainstem CMs have an increased risk of major neurological deficit associated with hemorrhage. Recent literature supports treating such CMs (deep and critical locations, e.g., thalamus and brainstem) with stereotactic radiosurgery (SRS) given the extremely high risk associated with surgical resection and their reported higher incidence of rehemorrhage and devastating neurological effects.<sup>[7,9]</sup> This remains a controversial subject as there are several papers published in support of and against treating CMs with radiosurgery. The controversy exists for many reasons including, radiosurgical technique, heterogeneity of the patient population, location and size of the CM, difficulty demonstrating radiographic improvement, ill-defined natural history.<sup>[11]</sup>

Many studies have also found increase hemorrhage rates in women, though not all studies report this increased risk.<sup>[15]</sup>

CMs may also significantly vary over time with respect to size. They can both increase and decrease in size over time. Increasing size, however, has not been correlated with increase risk of hemorrhage.<sup>[15]</sup>

### Literature review

The authors performed a search for all English-language publications listed in MEDLINE for: (1) any CNS injury or neurological sequelae from electrocution or electrical injury, (2) any association between (CM, cavernoma, cavernous hemangioma) or cerebrovascular malformations and hemorrhage, bleeding, or rupture, and (3) pathological mechanisms for electrical injury in

the nervous system.

Electrical injury is relatively common, though severity depends on voltage, current, skin resistance, and pathway traveling through the body. Voltages as low as 25 V may be dangerous, and fatal cases have occurred with voltages as low as 46 V. AC is twice as dangerous with frequencies between 40 and 150 Hz.<sup>[12]</sup> Severity of injury may also be increased in hypothyroidism, old age, arterial disease, or even fatigue. The nature of how the current travels through the body is hard to predict though studies have shown that it passes through tissues as if they were a “structureless gel”.<sup>[12]</sup>

The clinical effects resulting from electrical injury can be divided into immediate and late manifestations. Immediate complications include loss of consciousness, amnesia, motor and sensory disturbances, and even cardiac or respiratory arrest. Late manifestations may be either focal or nonfocal. Focal presentations include radiculopathies, neuropathies, hemiplegia, paresthesias, hyperesthesia, aphasia, muscle atrophy, and transverse myelitis.<sup>[4]</sup> These are highly dependent to the path of the electrical current, however, nonfocal symptoms often occur when the brain is outside the pathway of electrical current. Nonfocal symptoms include personality or behavioral disturbances, memory difficulties, confusion, and headache. Imaging of the brain may be normal in these circumstances, however, intracranial hemorrhages, hematomas, and arterial and venous thrombosis have all been reported.<sup>[12]</sup> Furthermore, presentations of intracranial hemorrhage have been reported as presenting in both immediate and delayed fashion and when not associated with any head trauma.

Four mechanisms are theorized to cause the various histopathologic features of electrical injury. The first is the electrostatic theory, which proposes that electrical charges build up in the body if a victim is not grounded.<sup>[10,12]</sup> These charges build and like charges oppose one another and result in a sudden expansile force of tissues yielding waves of decompression under the skin. This is supported by autopsy findings of tissue rupture after electrocution. The second theory is that vasoconstriction cause acute manifestations while later intimal injury and thrombosis may present as delayed symptoms.<sup>[10,12]</sup> This is referred to the vascular theory and is supported by pial vessel constriction found in animal studies of electrical injury. The third theory suggests that thermal injury occurs in tissue and result in pathological findings.<sup>[10,12,13]</sup> In fact, 5 hours after lethal electrocution temperatures of up to 145°F have been reported. Finally, the mechanical theory postulates that injury occurs because of violent jarring of tissue from electrical current.<sup>[10,12]</sup> As no single theory can account for all of the histopathologic findings, it is likely that a combination of these mechanisms account for the various findings with electrical injury.

The authors found no publications, describing any

relationship between cerebrovascular malformations, including CMs, and electrical injury or electrocution. One paper by Stewart and Long<sup>[14]</sup> did recognize a concern for potential hemorrhage in cerebrovascular malformations. They expressed caution when using electroconvulsive therapy (ECT) and considered cerebrovascular malformations a relative contraindication. However, there have been a few reports of patients with CMs being treated with ECT and these are performed under strict control of vital signs with the use of medication.<sup>[14]</sup> Furthermore, the ECT settings are reduced from the standard treatment. Additionally it must be recognized that ECT is direct current (DC) rather than AC, which has less potential risk of injury. Even with these few treatments under idealized settings, the authors state that caution should be exercised when using ECT on patients with CMs.

Though no causality between electrocution and CM rupture can be proven at this time, we would propose that given the history of this patient and what is known regarding electrical injuries, an association between electrocution and CM rupture is highly plausible. Any of the four electrical injury theories could explain the rupture of a CM, however, the vascular theory appears most likely. Though CMs are separate from the circulation, they would still likely suffer the same effects as normal blood vessels. In fact, given their abnormal morphology and propensity to spontaneously hemorrhage, CMs would likely be more susceptible to hemorrhage secondary to electrocution. Fortunately, some in the ECT community have acknowledged the potential association between electrocution and hemorrhage of CMs. Other than ECT, there have been no publications or recommendations regarding the risk of hemorrhage of CMs and electrocution.

## CONCLUSION

The presented case is the first in the literature to describe a potential association between electrocution and CM hemorrhage. Further studies and case reports are needed to validate this hypothesis in order to make firm recommendations regarding risk of CM hemorrhage and electrocution. In the meantime it may be advisable to caution patients of the potential increased risk of hemorrhage in CMs secondary to electrocution.

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