Vertebrobasilar Artery Dissection

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Citation

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Abstract

Object: Isolated basilar artery dissection, is an uncommon clinical event that may present as stroke. It has rarely been reported as gross and microscopic autopsy findings.

Methods: We describe a 46-year old man found unconscious and in the hospital a computerized tomography scan of the head showed cerebellar and brainstem infarct. He became comatose and died after a brief hospitalization without intervention. The basilar artery showed an acute tear of the intima and media with associated acute thrombosis.
Basilar artery dissection alone presents clinically as stroke due to either thrombosis or subarachnoid hemorrhage (SAH). The presence of thrombosis or SAH depends on the plane of dissection. Classically, angiographic studies are used to confirm the diagnosis. The mortality rate for this disorder is expectantly high. Treatment is typically supportive.

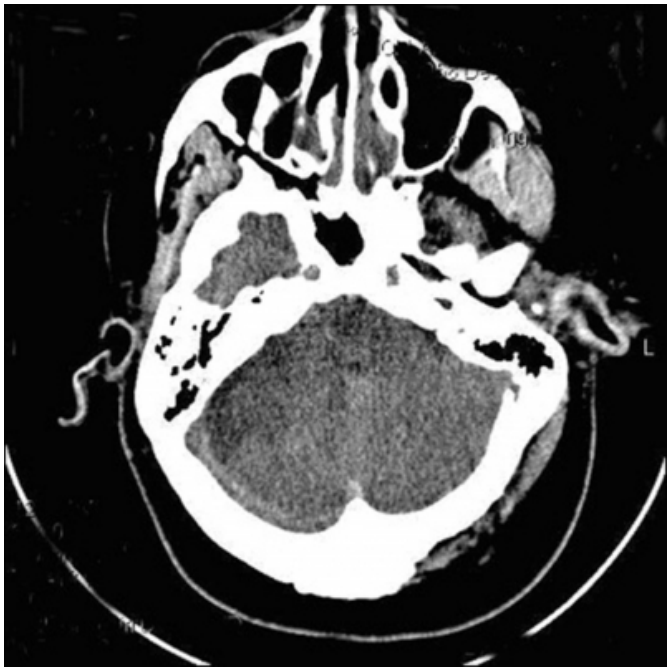
Conclusion: We report the clinical, gross, and microscopic findings of basilar artery dissection infarction of the cerebellum and brainstem.

CASE REPORT

A 46-year-old white man was found unconscious in the field, was resuscitated and intubated on site. On admission to the hospital, the patient was found to be hypotensive (52/31 mmHg), with a pulse of 92 beats per minute, oxygen saturation of 100 % and Glasgow Coma Scale of 3. Past history includes diabetes, hypercholesterolemia and smoking. The patient was given intravenous fluid and dopamine. Physical examination revealed loss of corneal and oculovestibular reflexes. CT scan of the head showed evolving subtotal cerebellar infarct and associated brain stem and midbrain infarct with edema (Figure 1).

Figure 1

Figure 1: Computerized tomographic scan of head depicting evolving posterior fossa subtotal cerebellar infarct and associated brain stem and mid brain infarct with edema.



Hydrocephalus with ventriculomegaly was also seen. The brain flow studies showed absent flow in the anterior and left

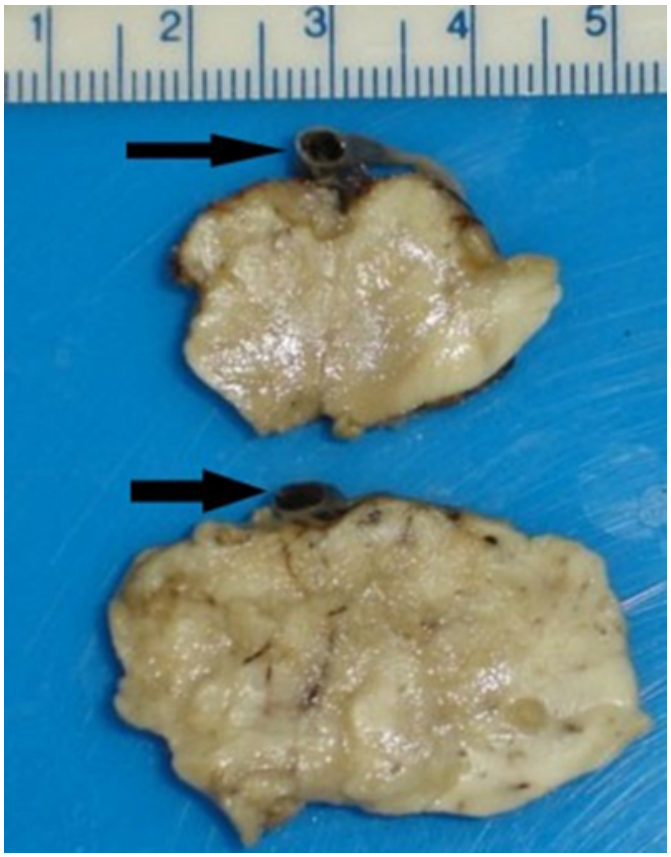
middle cerebral arteries with low cerebral perfusion without evidence of brain death. Electroencephalogram showed continuous beta range frequency (12-15 Hz) on a poorly reactive background intermixed with slower frequency in 7-8.5 Hz range and no significant focality. The patient became comatose, with no brain stem reflexes and was pronounced dead.

GROSS AND MICROSCOPIC FINDINGS

The gross examination of the brain (1,410 grams), revealed an 8.0 cm area of infarct involving the anteroinferior cerebellar surface, cerebellar vermis and brainstem. Complete basilar artery thrombosis was seen (Figure 2).

Figure 2

Figure 2: Cross section of brainstem including pons with complete basilar artery thrombotic occlusion (arrows)



The ventricles appeared slightly enlarged. Cross sections throughout the pons, medulla and cerebellum showed diffuse area of soft tan white infarcted tissue.

Microscopic examination of the cerebellum and brainstem showed ischemic necrosis. Acute basilar artery thrombosis with 100% occlusion was seen. The basilar artery showed tear of the intima and media confirmed by elastic stain consistent with basilar artery dissection (Figure 3 and 4).

Figure 3

Figure 3: Basilar artery dissection in the plane between the intima and media (hematoxylin and eosin, original magnification x 400).

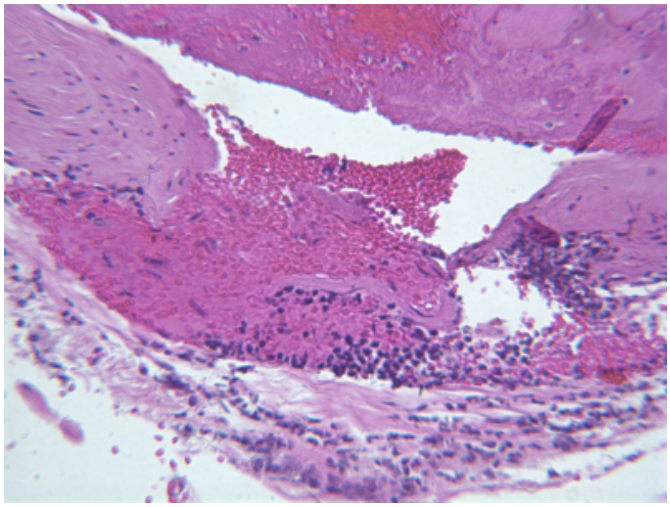
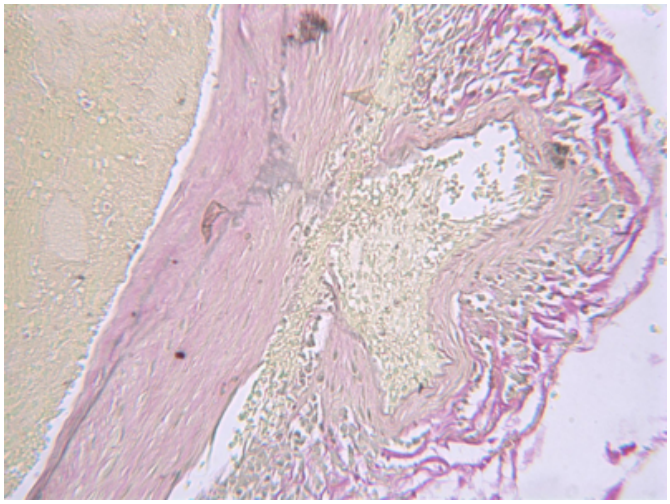


Figure 4

Figure 4: Elastic staining, original magnification x 400.



DISCUSSION

Basilar artery dissection, a rare type of vertebrobasilar dissection can cause thrombosis and SAH leading to stroke. In general, patients with intracranial vertebrobasilar dissection can be divided into two distinct groups on the basis of pathologic features, which affects clinical presentation. If the plane of dissection occurs between the internal elastic lamina and media, an expanding intramural hematoma can lead to stenosis or occlusion of the vessel lumen or perforating arteries arising from the affected segment, resulting in brainstem ischemia. If the dissection extends to the subadventitial plane, extensive SAH may result. The walls of the intracranial arteries are thin

compared with vessels of similar size elsewhere, and the lack of external elastic lamina allows the dissecting hemorrhage to rupture into the subarachnoid space. Most patients with vertebrobasilar dissection present with headache, neck pain homolateral to the side of the dissection and preceding the onset of the more severe neurological symptoms by a few hours.

Basilar artery thrombosis is associated with a high mortality rate [2]; however, the prognosis is dependent on several factors that enable an estimate of its course and outcome. Factors associated with poor outcome include decreased level of consciousness, dysarthria, pupillary disorders, bulbar symptoms, diplopia, bilateral cerebellar lesions, and a cardiac cause of embolism [4]. Up to 90% of patients with no such factors have a good functional outcome, while all patients with such factors either die or will have severe disability. The mortality rate is consistently greater than 90%. Although the basilar artery occlusion is rare in children, Nakatomi and his colleague's have reported basilar artery occlusion without any involvement of the vertebral arteries in a 7-year-old boy without any basic disorders [12]. Yoshimoto and his colleagues presented 10 cases of basilar artery dissection [19] among them six cases with unruptured basilar artery dissection and all were treated conservatively i.e., without any surgical or endovascular intervention. Functional outcome of these patients was one patient with vegetative state and others with severe disability. A review of literature revealed that basilar artery dissection carried significant morbidity and death, whether they presented with ischemia or subarachnoid hemorrhage [17].

Subarachnoid hemorrhage from ruptured intracranial vertebrobasilar dissection is known mainly from Japanese publications and seems to affect primarily the vertebral arteries [6, 7, 11, 15]. The natural history of vertebrobasilar dissecting aneurysms presenting with SAH is notable for a high incidence of recurrent bleeding within the first hours or days after the ictus. In a review of 60 cases in the literature, Aoki and Sakai [1] found that 18 patients (30%) were documented with rebleeding, predominantly during the acute stage, with a temporal profile similar to that observed in patients with ruptured intracranial saccular aneurysms. Mizutani et al. [10] reported a series of 42 patients with SAH from vertebrobasilar dissecting aneurysms treated at a single institution; they noted a remarkable 69% incidence of recurrent bleeding before surgical or endovascular therapy. Fifty-seven percent of the recurrent hemorrhages occurred within 24 hours and 80% within 7 days of the initial hemorrhage. The mortality (46.7%) of patients with a second episode of bleeding was significantly higher than that (8.3%) of those who bled only once. The authors concluded that a delay before surgical treatment resulted in a high mortality, as the result of subsequent ruptures was devastating.

Risk factors of vertebral and basilar artery dissection include hypertension (primary or secondary), trauma, sepsis, bacterial endocarditis, arteritis, hypercoagulable state, fibromuscular dysplasia, cystic medial necrosis, Ehlers-Danlos syndrome, Marfan syndrome, and rheumatoid arthritis [8, 17]. Extracranial vertebral artery dissection can be spontaneous or traumatic, whereas intracranial vertebral artery dissection is mostly spontaneous and presents with posterior circulation infarctions or SAH [10].

Diagnosis of vertebro-basilar artery dissection is classically based primarily on angiographic studies, even though the use of magnetic resonance imaging (MRI) can enable a diagnosis to be made non-invasively. The issue of management of ruptured dissection in the vertebral artery is still unsettled [3, 13]. The MRI findings of dissection include an intramural hematoma, an intimal flap, and the enhancement of the artery wall and septum. MRA can clearly show abrupt luminal stenosis and the disappearance of flow signal at end distal to the dissection [9]. MRA can also enable the age of the hematoma to be estimated. An intramural hematoma has an intermediate signal on T1-weighted images and a high signal on T2-weighted images in the first few days of dissection. Subsequently, there is a high signal on both T1 and T2-weighted images and finally, the hyperintensity resolves [18]. A high-intensity signal within the arterial wall on T1- and T2-weighted images provides evidence of acute intramural hematoma, corresponding with the area of focal narrowing (the “string sign”) observed on conventional angiography. The signal intensity of the intramural hematoma diminishes over time, resolving within 6 to 8 weeks. The two vertebral arteries with the basilar artery are the main blood supply to the posterior circulation of the brain. Depending on the site of the involvement, vertebral artery dissection is classified as V1 to V4, or extracranial and intracranial [14]. Extracranial dissection includes V1 (proximal to entry into the transverse foramen), V2 (within the transverse foramen from C6 to C2), and V3 (after exit from the C2 transverse foramen). Intracranial or V4 vertebral artery dissection refers to dissection that occurs after the vertebral artery has entered the dura [16]. It has been reported that 40% of posterior fossa infarctions are caused by vertebral artery dissection [5].

In conclusion, we describe a rare case of basilar artery thrombosis and infarction of the superior surface of the cerebellum and brain stem, caused by a dissection involving the basilar trunk and associated clinicopathologic features.

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