

Atypical bilateral cerebellar hemorrhage due to recreational drug use

Eğlence amaçlı uyuşturucu kullanımına bağlı atipik iki taraflı serebellar kanama

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Cerebellar hemorrhages may present with sudden onset posterior headaches, nausea, vomiting, gait disturbance, and impaired balance. They account for 5 to 10% of all intracranial hemorrhages (ICHs). Unilateral cerebellar hemorrhages are encountered more frequently than bilateral hemorrhages. Posterior fossa hemorrhages carry a high risk of mortality and morbidity due to fast-developing upward and tonsillar herniation. Acute bilateral cerebellar hemorrhages are rare, and outcomes are variable. We present a case of acute onset bilateral cerebellar hemorrhage, confined to the declive of the cerebellum in a young adult. Based on our review of the literature, we believe this is the first such case to be published.

A 42-year-old male with a history of hypertension and recent alcohol and cocaine use developed acute onset of headache, nausea, vomiting, and mild confusion at work. In the emergency room, the examination was significant for dysarthria/scanning speech, dysmetria, elevated systolic blood pressure greater than 220 mmHg, and the toxicological examination was positive for cocaine, benzodiazepines, and fentanyl. The initial computed tomography (CT) of head demonstrated an unusual appearing acute hemorrhage involving bilateral upper cerebellar hemispheres, specifically the declive segment of the posterior lobule. The head CT findings are shown in Figure 1. The patient was admitted to the neurology intensive care unit for

further management of persistent elevated blood pressure and close neurological monitoring. A CT angiogram of the head and neck was negative for aneurysms or vascular malformations. Workup of other underlying comorbidities included hemoglobin A1c, lipid panel, liver function test, and renal function, which were within normal limits.

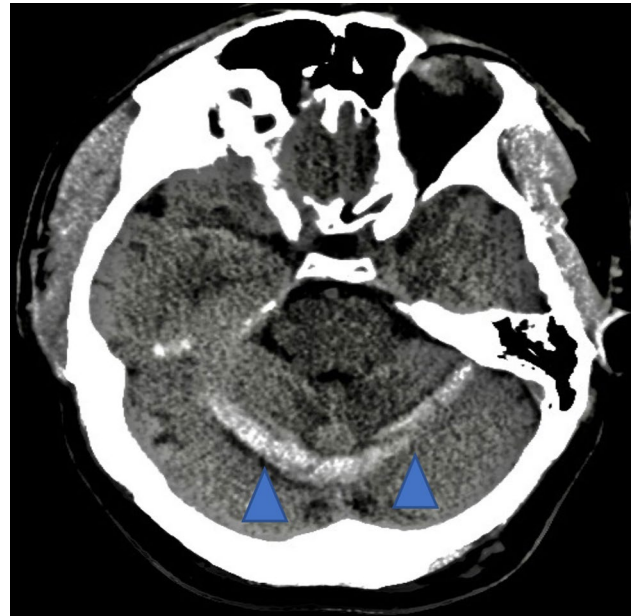


Figure 1. An axial section of the brain computed tomography displaying bilateral cerebellar hyperdensity corresponding to hemorrhage.

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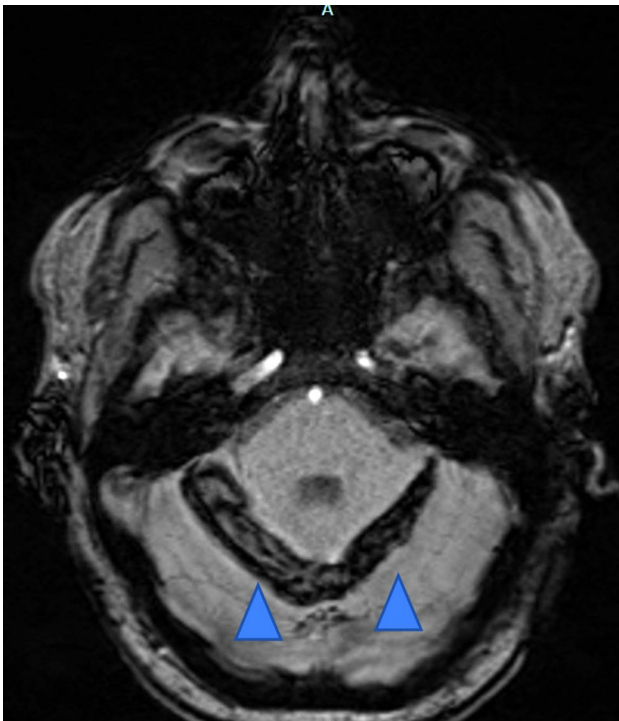


Figure 2. Susceptibility-weighted imaging revealing bilateral cerebellar hypointensity consistent with hemosiderin deposition.

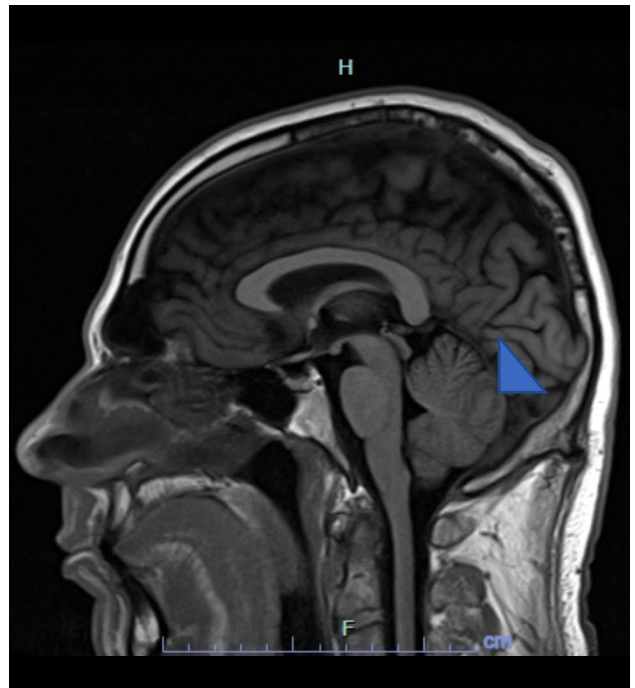


Figure 3. T1-weighted FLAIR magnetic resonance imaging demonstrating a minimal local mass effect on the adjacent brain parenchyma with subtle effacement of the sulci.

FLAIR: Fluid-attenuated inversion recover.

Magnetic resonance imaging of the brain demonstrated symmetric bands of susceptibility artifact in bilateral superior cerebellar hemispheres corresponding to the hyperdensity observed on the prior CT scans, patchy restricted diffusion, and associated FLAIR (fluid-attenuated inversion recover) hyperintensity and minimal local mass effect on the adjacent brain parenchyma (Figures 2, 3). No dural sinus thrombosis was observed on magnetic resonance venography.

The patient remained stable and was discharged home three days after the initial presentation, with examination still notable for dysarthria.

Intracranial hemorrhages represent 10 to 15% of stroke cases and may have a number of causes. These causes include hypertensive vasculopathy, cerebral amyloid angiopathy, coagulopathy, cerebral venous thrombosis, vasculitis, sympathomimetic drugs, aneurysm, and arteriovenous malformations. The putamen is the most common site of ICH with 35% of all intracranial bleeds occurring in this location, while cerebellar hemorrhages are infrequent. Most cerebellar hemorrhages are unilateral; however, bilateral cerebellar hemorrhages have occasionally been described.^[1,2]

Causative factors of cerebellar hemorrhage also vary. Hypertension is the most common risk factor for cerebellar hemorrhage. It is associated with Charcot-Bouchard microaneurysms involving branches of superior cerebellar arteries.^[3]

Cerebral venous thrombosis is another cause of cerebellar hemorrhage, be they unilateral or bilateral. Venous drainage of posterior fossa consists of four groups of veins, including the deep, superficial, bridging, and brainstem veins. Eng et al.^[4] reported a case of bilateral cerebellar hemorrhage due to straight sinus thrombosis. Lattanzi et al.^[5] reported bilateral cerebellar hemorrhage due to vermian vein thrombosis in a 67-year-old male who presented with sudden onset headache and gait issues.

There are several case reports and series reporting spontaneous cerebellar hemorrhages, including the study by Lalla et al.^[6]

Another possible cause of bilateral cerebellar hemorrhage is recreational drug use. Ahmed et al.^[7] published a case report in 2021 of a 51-year-old female with bilateral cerebellar hemorrhage due to opiate-induced toxic encephalopathy. Authors

reported that their case was in a spectrum of Chanter syndrome. This syndrome was reported by Jasne et al.^[8] as a combination of specific imaging findings, such as cerebellar, hippocampal, and basal ganglia involving diffusion-weighted changes with transient edema in patients with known history of recreational drug use.

Bilateral cerebellar hemorrhage may happen in settings of vascular malformations and as complications of intracranial procedures. Amini et al.^[9] reported a case series of remote unilateral and bilateral cerebellar hemorrhages that occurred in patients after undergoing supratentorial craniotomies for intracranial mass resection or aneurysm clipping. Sasani et al.^[10] reported a case of a 47-year-old female with remote cerebellar hemorrhage after spinal arteriovenous malformation surgery.

Mullaguri et al.^[11] reported a case of bilateral cerebellar hemorrhage within the posterior inferior cerebellar artery territory in a female patient with a history of cocaine use. Catheter angiogram showed patent vasculature and cocaine-induced vasospasm as a cause of the intracranial hemorrhage, as well as multifocal ischemic strokes remained as a top differential. Several cases of cocaine-induced unilateral or bilateral hemorrhages involving the cerebellum or basal ganglia have been published to date, with the mechanism thought to be possible vasospasms, hypoxia, or thrombosis.

In this report, the patient was found to have a cryptogenic bilateral cerebellar ICH confined to the declive of the cerebellum. Possible mechanisms include site-specific toxicity of the illegal drugs consumed prior to presentation, although no prior descriptions of drug-related, declive-specific hemorrhage are reported in the literature. The hemorrhage might also result from drug-induced hypertension with which the patient presented; however, it was unclear why the hemorrhage would have such an unusual pattern in this setting.

In conclusion, bilateral cerebellar hemorrhage is an infrequent entity that may be associated with recreational drug use. A complete workup for the cause of such hemorrhages is important to rule out other etiologies, such as vascular malformations and cerebral venous thrombosis. This report is, to the best of our knowledge, the first hemorrhage confined to the cerebellar declive reported in the medical literature.

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