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BRIEF REPORT

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Brainstem hemorrhage in descending transtentorial herniation (Duret hemorrhage)

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Abstract *Objectives:* To review clinical and radiological findings in patients with Duret hemorrhages and to discuss the pathophysiology and differential diagnosis of these lesions. *Patients and methods:* We reviewed the case records of four patients with Duret hemorrhages who had been admitted to the neurological intensive care unit with supratentorial mass lesions. *Results:* Descending transtentorial and subfalcine herniations were present in all cases. Three patients were admitted with acute subdural hematoma and one with intraparenchymal hemorrhage. Computed tomography revealed the presence of blood in the mesencephalon and upper pons. Three patients died; one survived with severe disabilities. *Discussion:* Duret hemorrhages are typically located in the ventral and paramedian aspects of the upper brainstem (mesencephalon and pons). The pathophysiology of Duret hemorrhage remains under debate: arterial origin (stretching and laceration of pontine perforating branches of the basilar artery), versus venous origin (thrombosis and venous infarction). Multifactorial causation seems likely. *Conclusion:* Duret hemorrhages are delayed, secondary brainstem hemorrhages. They occur in craniocerebral trauma victims with rapidly evolving descending transtentorial herniation. Diagnosis is made on computed tomography of the brain. In most cases the outcome is fatal. On the basis of our observations we believe that arterial hypertension and advanced age are risk factors for the development of Duret hemorrhage.

Keywords Brain, computed tomography · Brain, hemorrhage · Brain, injuries · Brain, herniation · Duret hemorrhage

Introduction

Brainstem hemorrhages are commonly reported in autopsy series of severely head-injured patients [1, 2]. They can be classified as primary or secondary [2, 3]. Causes of primary brainstem hemorrhage include: direct laceration or contusion, penetrating injury, shearing injuries, petechial hemorrhages, disruption of the pontomedullary junction [3]. Secondary hemorrhages of the brainstem in craniocerebral trauma victims occur at a later stage as a result of descending transtentorial herniation [4]. These are known as Duret hemorrhages.

The neuropathological features of Duret hemorrhages are well known. However, the frequency of brainstem hemorrhages observed with computed tomography (CT) or magnetic resonance imaging (MRI) is much lower than that observed in autopsy series of head trauma victims [3]. Descriptions of Duret hemorrhages documented in vivo are rare. Most have been published as case reports [5] or included in articles reporting primary and secondary brainstem injuries [3, 6].

We present a series of four patients with Duret hemorrhages, review the pertinent clinical and radiological findings, and discuss the pathophysiology and differen-

tial diagnosis. Finally, we conclude that a previous medical history of systemic hypertension and advanced age constitute risk factors for the development of this condition.

Case reports

Patient A

A 55-year-old comatose woman was admitted to the emergency room. Glasgow coma scale (GCS) was 4/15. Clinical neurological examination showed dilated pupils, with brisk light reflexes. The patient had a previous medical history of arterial hypertension. Noncontrast CT of the brain revealed a massive hemorrhage in the left cerebral hemisphere. Midline structures were shifted to the right, and perimesencephalic cisterns were flattened. There was no brainstem hemorrhage at this time. Cerebral arteriography showed a small aneurysm at the M1 segment of the left middle cerebral artery. The patient's neurological condition continued to deteriorate. Repeat CT after 24 h showed subfalcine and descending transtentorial brain herniations. Moreover, a hematoma in the brainstem was seen (Fig. 1a). The aneurysm was successfully clipped on day 4 after admission. After 2 months the patient was discharged to a nursing home.

Patient B

A 76-year-old man was brought to the emergency ward in comatose state. GCS upon admission was 4/15. Blood pressure was 228/96 mmHg. Clinical neurological examination revealed anisocoria with nonreactive pupils and Kussmaul's breathing. The patient had suffered a head trauma 48 h before admission. Emergency CT revealed a large and recent left subdural hematoma (SDH), resulting in a subfalcine herniation (Fig. 1b). Additionally, there was a downward transtentorial herniation as evidenced by the inferior displacement of the pineal calcification. In the brainstem multifocal slitlike hemorrhagic foci were observed. The patient died during the evening of the same day due to hemodynamic instability.

Patient C

A 69-year-old man was found at home. He was unconscious with a GCS of 11/15. On arrival at the hospital GCS was 3/15. Pupils were asymmetric and nonreactive. The patient was known to have essential hypertension. Once previously he had suffered an acute myocardial infarction and a cerebrovascular accident with left hemiparesis. He was treated with oral anticoagulants and prothrombin time was within therapeutic range. Emergency CT upon admission disclosed a large left SDH with layering effect in the frontoparietal region. A midline shift with compression of the left lateral ventricle was also observed. Moreover, there was extensive bleeding in the mesencephalon and pons with extension into the left cerebellar hemisphere and fourth ventricle. Under the protection of intravenously administered vitamin K dependent clotting factors the SDH was evacuated neurosurgically. The patient was transferred to the neurointensive care unit and given maximal antiedematous treatment. However, he remained comatose; electroencephalography showed markedly decreased electrical activity over the left hemisphere suggestive of irreversible brain damage. The patient died on the fourth day.

Patient D

A 56-year-old comatose woman was brought to the emergency ward. GCS was 3/15 with unequal and nonreactive pupils. Previous medical history showed that the patient was known with severe hypertension. She had undergone coronary artery bypass

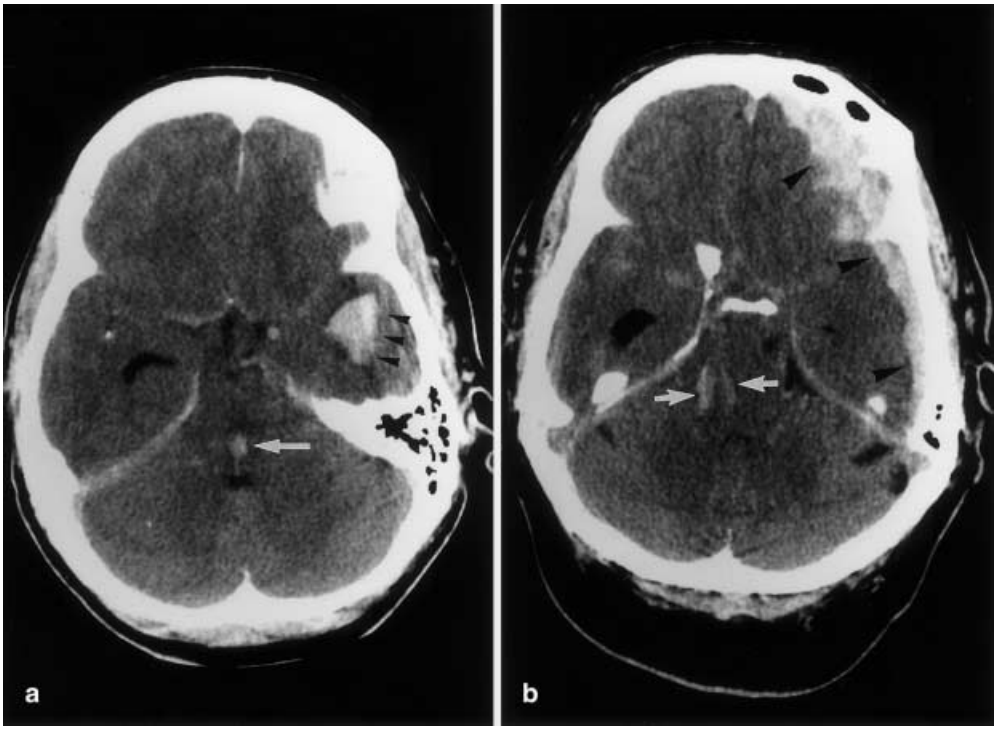

Fig. 1a, b Secondary brainstem hemorrhages (Duret hemorrhages) in two patients with descending transtentorial herniation due to a rapidly expanding supratentorial mass lesion. **a** Noncontrast axial CT in a 55-year-old woman (patient A) with a massive left cerebral hemisphere hemorrhage, extending into the temporal lobe (*black arrowheads*) 29 h after admission. There is a descending transtentorial herniation. There is a midline hemorrhagic focus in the upper brainstem, consistent with a Duret hemorrhage (*white arrow*). **b** Noncontrast axial CT in a 76-year-old man (patient B) after a fall on his head 2 days prior to admission. A left subdural hematoma is present (*black arrowheads*). Duret hemorrhage is observed in the upper pons (*white arrows*). Note the dilatation of the right temporal horn

grafting 4 years previously. Noncontrast CT of the brain disclosed an extensive left SDH with a mixed density pattern. There was a marked subfalcine shift of the midline structures to the right. The perimesencephalic cisterns were obliterated due to a downward transtentorial herniation. Infratentorially there was a large pontine hematoma, which had ruptured into the fourth ventricle. Emergency surgery was performed and the SDH was drained. The patient remained comatose during the postoperative period and finally died on the eighth postoperative day due to septic shock.

Discussion

Historical perspective and definitions

The Duret hemorrhage is named after Henri Duret, a nineteenth century French surgeon who worked in Charcot's laboratory in the Salpêtrière Hospital in Paris. Duret experimented on dogs to investigate the mechanisms of concussion. When he injected either gelatin or water inside the animal's skull, he observed that the swift increase in intracranial pressure caused multiple, minute hemorrhages in the brainstem near the floor of the fourth ventricle [5]. Discussion continues as to whether the hemorrhages observed by Duret in laboratory animals are the same as those that occur in the human brainstem with transtentorial herniation. Despite this uncertainty, the eponymous term "Duret hemorrhage" is now widely accepted to describe secondary brainstem hemorrhages due to descending transtentorial herniation of any cause.

Pathogenesis and risk factors

The pathogenesis of Duret hemorrhages remains controversial. Most authors support an arterial origin, but others consider venous congestion as a possible cause [7]. It is likely that more than one mechanism may be involved [2, 7]. A rapidly expanding supratentorial mass lesion causes severe increase in intracranial pressure and results in a descending transtentorial herniation. The most common causes include hematomas (epidural, subdural, intraparenchymal), and acute cerebral edema. The brainstem is pushed inferiorly, foreshortened and buckled. This leads to stretching, spasm, infarction, and hemorrhage of the central perforating arteries, which arise from the relatively immobile basilar artery [7]. Thus Duret hemorrhages usually commence in the midline of the mesencephalon and upper pons. Moreover, as a result of side-to-side compression there occurs an anterior-posterior elongation of the brainstem, which further stretches perforating arterial branches.

It is noteworthy that three of four of our patients had a previous medical history of arterial hypertension; the one patient without hypertension was 76 years old (relevant patient data are summarized in Table 1). The association of Duret hemorrhage and arterial hypertension has not been reported before. Our hypothesis is that, with arterial hypertension and advancing age, the elasticity of the blood vessel walls decreases, and the propensity to develop Duret hemorrhages increases. Venous congestion has also been reported as a possible cause of these hemorrhages [8]. Prolonged elevation in intracranial pressure could cause vascular thrombosis within the brainstem, which then can evolve to hemorrhage. The venous thrombosis and venous infarction theory is supported by the finding that small, thin-walled veins are more easily compressed and can undergo anoxic degeneration with subsequent vessel rupture and extravasation of blood.

Incidence

The reported incidence of secondary brainstem hemorrhages is significantly higher in neuropathological (30–60%) than in radiological studies (5–10%). Secondary brainstem damage was found in 51% of an autopsy

Table 1 Summary of relevant patient data

	Patient A	Patient B	Patient C	Patient D
Patient data	Woman aged 55 years	Man aged 76 years	Man aged 69 years	Woman aged 56 years
Previous medical history	Hypertension	Bronchogenic carcinoma	Essential hypertension, myocardial infarction	Hypertension
Cause of supratentorial mass effect	Intracerebral hemorrhage	Left SDH	Left SDH	Left SDH
Subfalcine herniation	+	+	+	+
Descending transtentorial herniation	+	+	+	+
Downward bowing of tentorium	+	+	+	+
Dilatation of contralateral lateral ventricle	+	+	+	+
Hemorrhage in mesencephalon	+	+	+	–
Hemorrhage in pons	–	+	+	+
Obliteration of 4th ventricle	–	+	+	+

series of 434 patients with nonmissile head injury [4]. Brainstem hemorrhage was observed in 37% of cases in a postmortem study of 132 fatal head injuries [9]. However, in CT studies the frequency of brainstem lesions was remarkably low. Secondary brainstem hemorrhages are found only in a small percentage of cerebral trauma patients with fatal prognosis. This discrepancy can be partially explained by the fact that up to 20% of secondary brainstem lesions are seen only microscopically [4]. Another factor may be the delayed development of secondary brainstem hemorrhages, occurring after the initial emergency CT. Finally, it could be possible that many of these severely injured patients do not survive long enough for a subsequent CT to confirm the diagnosis.

Differential diagnosis

Not all hemorrhagic lesions in the brainstem are Duret hemorrhages. Differential diagnosis includes primary traumatic brainstem hemorrhages (e.g., direct impact and penetrating injury, shearing injuries, disruption of the pontomedullary junction), as well as hypertensive bleeds and ruptured arteriovenous malformations. Direct impact hemorrhagic contusions of the brainstem occur with a lateral blow to the head, which causes a sudden acceleration-deceleration displacement of the brainstem relative to the tentorium. Patients with a narrow tentorial incisura are more vulnerable to this type of injury. Petechial shearing hemorrhages can be found in the posterior lateral quadrant of the brainstem [3, 10]; these are caused by a severe rotational force applied to the brainstem. Hypertensive brainstem hemorrhages usually arise in the dorsal region of the basis pontis. The pons is prone to hypertensive bleeds because it is supplied by direct perforating branches. This vascularization pattern is similar to that of the basal ganglia and thalamus, which are also sites of predilection. Hemorrhagic necrosis of the pons is a rare cause of brainstem hemorrhage and should be differentiated from pontine infarct, central pontine myelinolysis, and Duret hemorrhage. Brainstem hemorrhages can also occur due to rupture of an arteriovenous malformation. These instances are rare because clearcut arteriovenous malformations are infrequent in the posterior fossa, with the exception of cavernous malformations.

Conclusion

Duret hemorrhages are secondary brainstem hemorrhages that are caused by descending transtentorial herniation. They are typically found in the lower mesencephalon and ventral portion of the pons. The pathophysiology remains controversial; although most authors favor an arterial origin (stretching and laceration of perforating branches of the basilar artery), there are equally strong arguments for a venous origin (thrombosis and venous infarction). Intensive care physicians should understand the pathophysiology of descending transtentorial herniations. The delayed appearance of brainstem hemorrhage in a comatose patient with severe supratentorial hypertension is an ominous sign. Outcome is almost always fatal.

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