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Research Article

Intracranial Acute Subdural Hematoma Induced by Increased Intraperitoneal Pressure: Two Case Reports

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Abstract

Case 1: An obese (BMI 40.6 kg/m^2) 31-year-old Japanese woman who experienced a sudden headache and nausea when squatting to feed her pet was transported by ambulance. The CT scan of her head revealed a right Acute Subdural Hematoma (ASDH). She was discharged without neurological deficit after conservative treatment.

Case 2: A 35-year-old Japanese man who became comatose after an elbow punch to his right flank by an opponent during a soccer match was transported by ambulance. The CT scan of his head revealed a massive right ASDH. He was saved by emergency hematoma removal and external decompression. Active venous bleeding from the direction of the superior sagittal sinus was stopped by oxidized cellulose packing, and no brain contusion was observed intraoperatively. Left paralysis of 4/V in a Manual Muscle Test (MMT) and left hemianopia has remained.

Discussion: The intracranial ASDH in these two cases was caused without internal hemorrhagic risk factors or the mechanism of direct head trauma. These cases indicate that elevated intraperitoneal pressure may induce an intracranial ASDH, probably resulting from increased intracranial venous pressure. It has been reported that insufficiency of the internal jugular valve (which prevents the intracranial venous pressure from increasing by elevated intraperitoneal or intrathoracic pressure) is recognized in 25%–45% of healthy individuals. The present two cases are noteworthy because they suggest a new mechanism of intracranial ASDH.

Keywords: Acute subdural hematoma, ASDH, venous pressure, intraperitoneal pressure, internal jugular valve

Abbreviations

Acute Subdural Hematoma (ASDH); Body Mass Index (BMI); Computed Tomography (CT); Diffusion Weighted Imaging (DWI); Fluid Attenuated Inversion Recovery (FLAIR); Glasgow Coma Scale (GCS); Manual Muscle Test (MMT); Magnetic Resonance Angiography (MRA); Magnetic Resonance Imaging (MRI); T1-Weighted Imaging (T1WI); T2-Weighted Imaging (T2WI)

Introduction

A non-traumatic intracranial acute subdural hematoma (ASDH) may be caused by vascular malformations such as cerebral aneurysms, cerebral arteriovenous malformations, a dural arteriovenous fistula, brain tumor, and blood coagulation/fibrinolysis abnormalities [1-7]. We treated two patients with an intracranial ASDH without these factors or a direct head injury, and we speculated that the ASDHs were induced by increased intraperitoneal pressure.

Spinal ASDH due to increased pressure of spinal veins (which have poor valve structure) was reported to occur when the intrathoracic pressure rises accompanied by an elevation of intraperitoneal pressure [8-10]. The pressure of intracranial veins, which do not have a valve structure, is not easily increased because of the internal jugular vein valve, even if the central venous pressure rise when the intrathoracic pressure elevates [11]. However, internal jugular vein valvular insufficiency, which results in an elevation of the intracranial venous pressure accompanied by central venous pressure elevation due to the increase in the intrathoracic and intraperitoneal pressure, has been identified in 25%–45% of healthy individuals [12-15].

We report two cases of intracranial ASDH which were presumed to be triggered by an elevation of intraperitoneal pressure.

Case Presentation

Case 1

Case 1 was an obese 31-year-old Japanese woman (height 157 cm, weight 100 kg, body mass index [BMI] 40.6 kg/m^2) who consulted us with the chief complaints of severe headache and vomiting which occurred suddenly when she squatted down to feed her pet. She was under medical treatment for depression, but no antithrombotic drug was prescribed. On admission, her Glasgow Coma Scale (GCS) score was 15 without neurological deficit. Blood tests revealed no abnormalities in the blood count, biochemistry, or coagulation system. Head Computed Tomography (CT) showed a right ASDH (Figure 1A,B). She underwent conservative treatment from the admission day and cerebral angiography on the 3rd disease day which did not detect any intracranial organic vessel abnormality. The ASDH did not increase, and no etiology of bleeding was detected on magnetic resonance imaging (MRI) (T1-weighted imaging [T1WI], T2-

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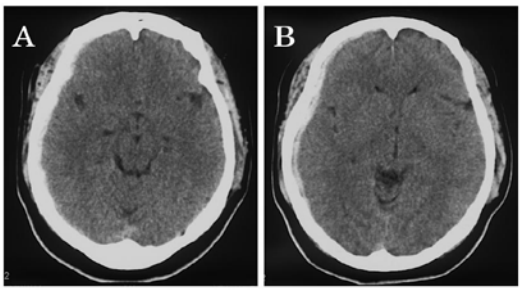

Figure 1: C ase 1, an obese 31-year-old woman. Head CT (axial image). CT shows a thin right ASDH.

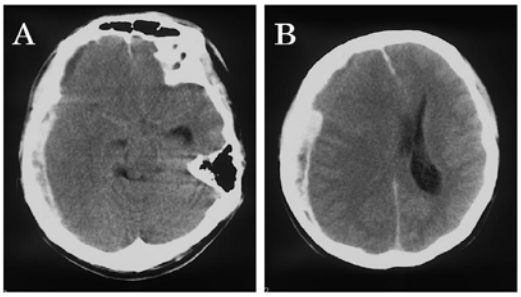

Figure 2: Case 2, a 35-year-old man injured in a soccer match. Head CT (axial image). CT shows a right ASDH and mid-line shift.

Weighted Imaging [T2WI], and Fluid Attenuated Inversion Recovery [FLAIR]) performed on the 4th disease day. She was discharged to home on the 5th disease day with no symptoms. CT performed on the 46th disease day indicated that the ASDH had disappeared.

Case 2

Case 2 was a 35-year-old Japanese man with no remarkable medical history who was transported to our hospital due to a disturbance of consciousness that occurred after an opponent's elbow hit his right flank strongly during a soccer match. Although he was injured at the flank, he was not injured at the head and walked out of the soccer field by himself without falling. On admission he was comatose with decerebrate rigidity and right mydriasis (8 mm dia.) without light reflex. Blood tests showed no abnormality in the blood count, biochemistry, or coagulation system. A head CT scan showed a right ASDH accompanied by uncus herniation, and it did not show brain contusion (Figure 2A,B). He underwent an emergency hematoma removal and external decompression. During the operation, active venous bleeding from the direction of the superior sagittal sinus was recognized but brain contusion was not observed. The venous bleeding was stopped by oxidized cellulose packing into the subdural space.

Head MRI (T1WI, T2WI, FLAIR, and Diffusion Weighted Imaging [DWI]) performed on the 10th disease day showed an intensity change in the right frontal lobe, temporal lobe, occipital lobe and corona radiata (Figure 3A–D). However, even with Magnetic Resonance Angiography (MRA), we did not observe any organic intracranial lesions (which can be the cause of an ASDH). The patient's consciousness became clear and he was transferred for rehabilitation on the 51st disease day. Left paralysis of 4/V in a Manual Muscle Test (MMT) and left hemianopia has remained as of this writing at 8 months after the injury.

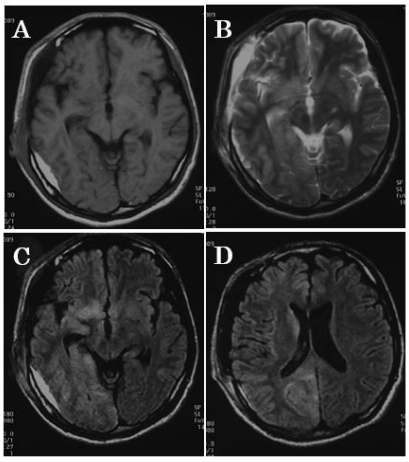

Figure 3: Case 2. Head MRI (axial image). A: (FLAIR). B: (T2WI). MRI shows the remaining thin right subdural hematoma. C, D: (DWI) MRI shows high-intensity change in the right frontal lobe, temporal lobe, occipital lobe, and corona radiata.

Discussion

Intracranial ASDHs are categorized into complicated subdural hematomas and simple subdural hematomas. Complicated subdural hematomas are caused by injured cerebral surface vessels accompanied by cerebral contusion. Simple subdural hematomas are caused by an injured bridging vein due to rotational acceleration without cerebral contusion. Shenkin reported that 61.5% of ASDHs resulted from injured cortical arteries that could cause a complicated subdural hematoma, 25.6% of ASDHs resulted from a venous hemorrhage that could cause a simple subdural hematoma, and other ASDHs had an unknown etiology [16].

It was reported that a non-traumatic intracranial ASDH may be caused by intracranial organic disease such as a cerebral aneurysm, brain tumor, cerebral arteriovenous malformation, or dural arteriovenous fistula, and blood coagulation/fibrinolysis abnormalities including HELLP syndrome and antithrombotic drug administration [1–7,17]. The ASDHs of our two patients occurred without direct head injury, intracranial organic disease, or blood coagulation/fibrinolysis abnormalities as shown by their clinical histories, brain images and blood tests. The ASDH in Case 1, an obese woman, occurred when she was squatting and the ASDH in Case 2 occurred while he was straining after an elbow punch to his right flank. Thus, both of the ASDHs occurred in a situation in which the patient's abdominal pressure was increased.

Elevated intraperitoneal pressure has been reported to increase intracranial venous pressure. Sugerman et al. reported the details of six women with brain pseudotumors and high intracranial pressure (293 ± 80 mmH_2O) due to remarkable central obesity (BMI ≥ 35 kg/m^2) [18]. They speculated that the elevated intraperitoneal pressure due to the patients' obesity increased the intrathoracic pressure, resulting in increased central venous pressure and intracranial venous pressure.

Although, to our knowledge, intracranial ASDH due to elevated

intraperitoneal and/or intracranial venous pressure has never been reported, it has been suggested that elevated intraperitoneal and/or intracranial venous pressure is related to headaches and transient global amnesia [12-15]. Intraperitoneal pressure can rise due to coughing, squatting, and holding one's breath even in daily life and especially due to obesity or pregnancy, and elevated intraperitoneal pressure has resulted in venous spinal ASDHs resulting from increased intrathoracic pressure [9,10]. It is thought that spinal veins without a valve structure can be affected by venous congestion [8]. In contrast, the intracranial veins, which also have no valve structure, are not easily affected by elevated intrathoracic pressure thanks to the internal jugular vein valves [11].

However, increased intrathoracic pressure can elevate the intracranial venous pressure with internal jugular vein valvular insufficiency. Nowadays, the internal jugular vein valves are evaluated during a Valsalva procedure with ultrasonography, which is less invasive than venography [12]. Cejas et al. reported that 79.5% of patients with transient global amnesia had internal jugular vein valvular insufficiency [13]. Chung et al. reported that intracranial venous regurgitation was recognized in 50% of patients with transient global amnesia by three-dimensional time-of-flight MRA [19]. As noted above, internal jugular valvular insufficiency was recognized even in 25%-45% of healthy individuals [12–15].

We speculated that the increased intraperitoneal pressure of the obese Case 1 when she squatted caused the increases in intrathoracic pressure, internal jugular vein pressure and intracranial venous pressure leading to the intracranial venous ASDH. Yamashita et al. reported that the subdural bridging veins, which lack collagen, are susceptible to bleeding due to their fragility [20]. In addition, Drake et al. reported that 10 of 11 patients with an intracranial arterial ASDH were in a coma; two of these 10 patients fell into a coma immediately after injury and the other eight patients became unconscious progressively. Drake et al. noted that a characteristic of the intracranial arterial ASDHs was progressive neurological deterioration, which was not observed in case 1 [21]. Therefore, the intracranial ASDH in case 1 can be thought to be a venous ASDH.

The intracranial ASDH in Case 2 occurred without a direct head injury or rotational acceleration to the head. The patient did not receive any blow to his head and did not fall down. We speculated that the increased intraperitoneal and intrathoracic pressure due to the elbow punch to his right flank and his straining due to the pain resulted in increased intracranial venous pressure leading to the intracranial ASDH due to the rupture of a fragile subdural bridging vein. The intensity change observed in postoperative MRI in his case is thought to be a cerebral infarction caused by the microcirculatory disturbance due to the compression of the ASDH, as Schroder et al. described [22].

There were no direct injuries to the head in either of the present cases, and there were no underlying diseases that could cause a non-traumatic subdural hematoma. Although we could not confirm the internal jugular vein valvular insufficiency in our patients, it is worth reporting the possibility that an intracranial ASDH can be induced by increased intraperitoneal pressure without external force to the head.

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