more precise, as in visible bone. Furthermore, the surgeon needs only 1 assistant or none.

A simple action using radiopaque materials lets surgeons perform reduction more exactly in short time, and patients avoid high costs and lots of radiation exposure.

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Delayed Acute Subdural Hematoma Associated With Percutaneous Coronary Intervention

Nobuhiko Arai, MD, Akiyoshi Nakamura, MD, PhD, Masanao Tabuse, MD, PhD, and Hiromichi Miyazaki, MD, PhD

Background: Delayed acute subdural hematoma (DASH) is a subdural hematoma which is detected later. An initial computed tomography (CT) does not reveal any intracranial hemorrhage at all. Few patients of DASH after mild traumatic brain injury associated with percutaneous coronary intervention (PCI) have been published.

Patient Presentation: A 63-year-old woman presented with cardiac pulmonary arrest due to acute myocardial infarction and lethal arrhythmia. She had hit her head on the road. The initial CT did not reveal any hemorrhage in the intra-cranium. She fully recovered after PCI. However, 1 hour after PCI, she lost consciousness and immediate CT showed acute subdural hematoma and subarachnoid hemorrhage. The period from losing consciousness to brain herniation presenting as anisocoria was very short—only 30 minutes in our patient. Although emergent evacuation of hematoma and external decompression were performed, the patient died 1 day after the operation.

Conclusion: The authors encountered a patient of DASH after PCI that resulted in death. Clinicians should be aware that subdural hemorrhage can occur after PCI if no hemorrhage is noted in the initial head CT, and the operation should be performed as soon as possible when the consciousness level decreases.

Key Words: Cardiac pulmonary arrest, delayed acute subdural hematoma, mild traumatic brain injury, percutaneous coronary intervention

Mild traumatic brain injury (TBI) is very common in clinical practice and is the cause of most admissions due to head injuries. Head injuries are sometimes related to acute myocardial infarction (AMI) because of sudden loss of consciousness when it is accompanied by lethal arrhythmia. In such patients, percutaneous coronary intervention (PCI) is usually performed and an antithrombus agent is administered. Delayed acute subdural hematoma (DASH) is a subdural hematoma that is detected later, in which an initial computed tomography (CT) does not reveal any intracranial hemorrhage. To the best of our knowledge, this is the first patient of DASH which developed after PCI.

PATIENT PRESENTATION

A 63-year-old woman presented with cardiac pulmonary arrest (CPA). She lost her consciousness and fell down on the pavement. She hit the occipital site of her head on an asphalt road. Some pedestrians overlooked this fact and verified her CPA. Before arrival of rescue workers, automated external defibrillator was administered once and return of spontaneous circulation was achieved. When rescue workers examined her in an ambulance, she was alert and oriented to time, place, and person. She denied any pain or dyspnea. She has a history of diabetes mellitus and was taking an oral hypoglycemic agent. She had no family history of heart disease. In the emergency room electrocardiography revealed ST elevations in the V1-3 electrodes. In addition, echocardiography showed septal hypokinesis. Acute myocardial infarction was strongly suspected. Before coronary angiography, whole body CT was performed to detect other injured sites including the head and to rule out pulmonary embolism and aorta dissection. Computed tomography did not reveal any intracranial abnormality, including hemorrhage (Fig. 1). Coronary angiography showed acute occlusion of the anterior descending branch. Plain old balloon atherectomy was performed, and a drug-eluting stent was implanted in the lesion. It took 2 hours to complete the procedure. Furthermore, 5000 units of unfractionated heparin were administered in the emergency room, and an additional 5000 units were administered at the beginning of the procedure. The activated clotting time was not estimated during the procedure because the coronary angiography was completed in a short duration and the amount of a heparin was considered to be adequate. After the procedure, she was oriented to time, place, and person and could communicate with her family

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This clinical note is involving human. We have been in compliance with the Helsinki Declaration and given approval by the ethics committee of Hiratsuka City Hospital.
The authors report no conflicts of interest.
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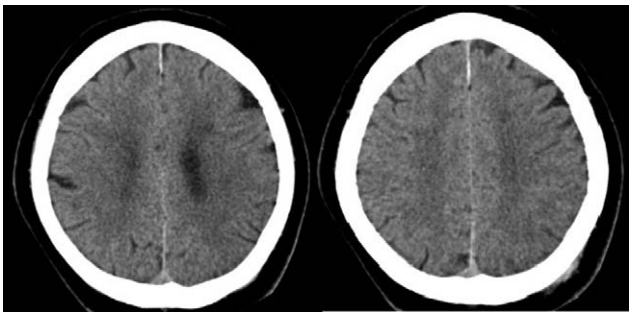

FIGURE 1. Initial computed tomography did not show any hemorrhage.

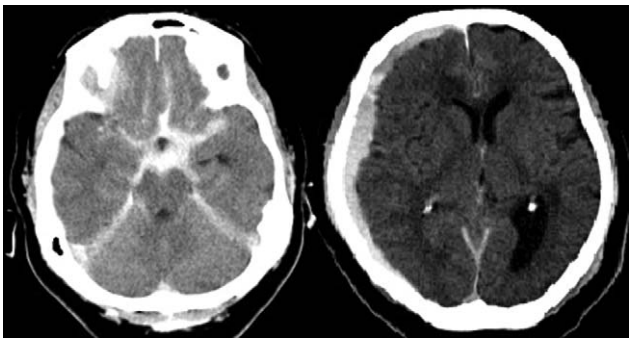

FIGURE 2. Computed tomography performed 1.5 hours after percutaneous coronary intervention revealed massive subarachnoid hemorrhage and acute subdural hematoma.

members. However, 1 hour after PCI, her consciousness decreased and she went into coma. Emergency follow-up head CT revealed a massive subarachnoid hemorrhage and right acute subdural hematoma (Fig. 2). This image was acquired 9 hours after the head trauma. Computed tomography angiography was performed to detect an aneurysm, which showed no aneurysm. Continuous heparin drip was discontinued soon after the CT. It is noted that only 30 minutes was taken from her consciousness-level down to brain herniation which presented as anisocoria. Emergency hematoma evacuation and external decompression were performed. However, intraoperatively, the whole brain looked severely pale and no longer pulsated. She died 1 day after the operation.

DISCUSSION

Universally, mild TBI is defined by the following criteria: initial Glasgow Coma Scale score of 13 through 15, loss of consciousness of <20 minutes, absence of focal neurological deficits, and post-traumatic amnesia of <24 hours.[1] Mild TBI is very frequent in clinical practice and accounts for 80% of all patients of hospital admissions due to head injuries.[2] In most patients, patients with mild TBI are permitted to go home if no lesion is detected in the head CT. However, some patients exacerbate and need surgical treatment. In almost all such patients, the cause is delayed intracranial hemorrhage. Many reports about delayed intracerebral hemorrhage and epidural hematoma have been published.[3–5] However, patients of delayed acute subdural hematoma are uncommon.

Delayed acute subdural hematoma, first described by Cohen and Gudeman,[6] is defined as an acute subdural hematoma (ASDH) that is not apparent on the initial CT scan, but appears on a follow-up CT scan. Delayed acute subdural hematoma can occur in about 0.5% of ASDH patients treated with surgery at the hospital.[6] Delayed acute subdural hematoma patients have rarely been discussed in detail, and only 7 previous patients have been published thus far.[7–11] The characteristics of all 8 patients including our 1 are summarized in Table 1. Patient 1 was not examined using CT but cerebral angiography, which did not reveal any abnormalities. This patient resulted in death due to DASH, which was found at autopsy. The other patients were examined by CT. The time interval to the following CT scan ranges from 9 hours to 72 hours. The prognosis is almost poor (5 deaths and 2 in disability) except for patient 7 (good recovery). The inducing factor is probably antithrombotic agent because 5 patients are related to that.

Itshayek et al[11] considered the administration of anticoagulants to elderly patients as a risk factor for DASH. Our patient was considered to be closely related to PCI, which is a very effective and established procedure to treat coronary lesions. Conversely, some common complications such as peripheral embolism, acute coronary occlusion, or puncture side related complications can occur. The anticoagulant heparin is imperative to avoid embolic complications and thrombosis. In this patient, we administered 10,000 units of heparin in total, which was an appropriate amount considering her weight. As reported in the study above, anticoagulant use seems to be related to DASH. This patient suggests that PCI may induce DASH, even after mild TBI. Percutaneous coronary intervention procedure for CPA due to AMI is imperative. Therefore retrospectively this complication, subdural hematoma, was unavoidable. However, at least clinicians should explain this complication to the patients and their family prior to the procedure and recognize it as soon as possible.

TABLE 1. Lists of Delayed Acute Subdural Hematoma

No	Reference	Age/Sex	Event	Initial CT	Second CT	Interval	Intervene	Outcome	Supplement
1	Cassin and Spitz[7]	19/F	TC	Skull fracture contusion	ASDH	15 h	Conserve	Death	Alcoholic
2	Koumtchev et al[9]	70/M	TC	Normal	ASDH	24 h	Conserve	Death	
3	Itshayek et al[11]	86/M	Fall	Normal	ASDH	72 h	Craniotomy	Death	Aspirin, enoxaparin
4	Itshayek et al	69/M	Fall	Normal	ASDH	9 h	Craniotomy, evacuation of hematoma	Death	Coumadin, ESRD
5	Itshayek et al	65/M	Fall	Normal	ASDH	24 h	Craniotomy, evacuation of hematoma	Moderate disability	Coumadin INR 2.99
6	Itshayek et al	72/W	Hitting	Normal	ASDH	24 h	Conserve	Mild disability	Wf INR 3.03
7	Matsuda et al[10]	18/M	Hitting	Normal	ASDH	48 h	Conserve	GR	
8	Present patient	63/F	Fall	Normal	ASDH SAH	9.5 h	Craniotomy	Death	PCI

ASDH, acute subdural hematoma; ESDR, end stage renal disease; GR, good recovery; INR, international normalized ratio of prothrombin time; PCI, percutaneous coronary intervention; SAH, subarachnoid hemorrhage; TC, traffic crash; Wf, warfarin.

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Some mechanisms, by which a subdural hematoma occurs later, are described below. First, owing to hypotension, the intracranial hemorrhage was so tiny that it could not be detected on the initial CT. Therefore, when a patient's blood pressure increases, some bleeding appears in the cranium. Second, elevated intracranial pressure or cerebral edema prevents bleeding from the capillary. This mechanism is likely to be involved in patients with severe TBI. Other mechanisms such as vasoparalysis resulting from local hypoxia[12] or local metabolic disorders that may injure the vessel wall[13] have also been suggested. The patient was in CPA at the onset of TBI. It is possible that the initial intracranial hemorrhage was so tiny owing to the absence of circulation. After PCI, blood pressure was in the normal range. In addition to this, antithrombus agent use probably resulted in increasing the size of the hemorrhage, which caused brain herniation. A notable thing in this patient is the very short duration in which brain herniation developed. The interval from coma onset to brain herniation was far shorter than that from TBI to coma onset. Thus, immediate surgical treatment should be administered.

CONCLUSIONS

We encountered a patient of DASH following successful PCI 9.5 hours after mild TBI. The initial CT showed no intracranial hemorrhage. However, delayed new acute subdural hematoma and subarachnoid hemorrhage was detected. And it took only 30 minutes for brain herniation to occur after the appearance of coma. Clinicians should be aware that DASH can occur after PCI, especially in CPA recover patients. The operation should be performed as soon as possible when DASH-related PCI occurs because of its exacerbation-period shortness.

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Efficacy of Topical Timolol as Primary Monotherapy in Cutaneous Facial Infantile Hemangiomas

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Abstract: Recent studies have shown that infantile hemangiomas (IHs) undergo a rapid growth phase between 5.5 and 7.5 weeks of life and do not usually proliferate beyond 6 months; growth thereafter is usually proportionate to the child's growth. This review assesses the evidence for topical timolol as primary monotherapy for cutaneous facial IHs before 12 months of age, and to determine the differences in outcome between early (before 6 months) and late initiation (after 6 months) of timolol. A review of English language articles published up to November 2015 was performed using selected key words. Articles identified were further reviewed for relevance. The full text of studies included for final analysis was perused to include pertinent patient details, treatment protocol with timolol, complications (if any) reported, and response to treatment. Four studies met the inclusion criteria. In children before 12 months of age, the efficacy of topical timolol for the treatment of cutaneous facial IHs in achieving clinically significant improvement as defined by a standardized Global Assessment Score score of 3 and above ranged from 47% to 88%. One study also showed that IH regression was greater in patients started on timolol before 6 months of age compared with those started later ($P < 0.05$). Topical timolol initiated in children before 12 months of age appears to be safe and clinically effective. There was insufficient data for detailed analysis of outcomes in patients who commenced treatment before and after 6 months of age.

Key Words: Beta-blocker, infantile hemangioma, timolol, topical

The serendipitous observation of the effect of beta-blocker therapy on hemangioma regression in a child with concomitant cardiomyopathy back in 2008 by Léauté-Labrèze et al[1] has led to a paradigm shift in the management of infantile hemangiomas (IHs), which is the most common benign tumor in children with an incidence of approximately 5%.[2] Corticosteroids, which were the

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