

Subdural Hematoma after Open-Heart Surgery

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Four cases are described of acute subdural hematoma that occurred after valve replacement in patients receiving anticoagulant therapy. All four patients experienced rapid deterioration of consciousness or neurological dysfunction, to varying degrees, between two and 42 days after valve replacement; emergency brain computed tomography scanning demonstrated the presence of subdural hematoma.

A variety of neurological complications may occur following an open-heart operation, especially when cardiopulmonary bypass (CPB) is used. The incidence of stroke and neurological complications after open-heart surgery has been recognized to range between 1.6% and 5.2% (1). Most strokes are embolic (1-3), and hemorrhagic events such as subdural hematoma or intracerebral hemorrhage have been rarely reported (3-7). Herein, four cases are described of acute subdural hematoma that occurred after valve replacement in patients receiving anticoagulant therapy.

Case reports

Clinical data on the four patients are listed in Table I; additional information relating to the individual patients is summarized below.

Preoperatively, one patient had a history of a syncope attack accompanied by a fall which resulted in what was considered to be minor head trauma; however, no neurological dysfunctions were present and computed tomography (CT) brain scanning showed no cerebral infarction or hemorrhage in any of the four patients. Anticoagulant therapy, which consisted of warfarin with or without aspirin, was discontinued in all patients at least seven days before surgery. At sur-

The neurological problems were completely resolved by removal and drainage of the hematoma in three patients, while conservative management was performed with no aggravation of neurological symptoms in the fourth patient.

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gery, the standard technique of CPB with moderate hypothermia was used, and the activated clotting time was maintained at over 400 s with heparin during CPB. Protamine reversal of heparinization was carried out routinely after termination of CPB.

Postoperatively, the intravenous administration of heparin was started on the first postoperative day and continued until the target International Normalized Ratio (INR) could be regulated by oral anticoagulation with warfarin. Low-dose aspirin (100 mg/day) was also used for anticoagulation. None of the patients had experienced overt head trauma prior to the onset of neurological dysfunction after surgery.

Patient 1

A 61-year-old woman was admitted for treatment of severe mitral and tricuspid regurgitation. She had a history of pacemaker implantation for sick sinus syndrome 12 years previously, and had been receiving anticoagulation with warfarin since that time. At surgery, the patient underwent mitral valve replacement (MVR) with a mechanical valve, tricuspid valve replacement (TVR) with a bioprosthetic valve, and replacement of a catheter electrode with a myocardial electrode. Her postoperative hemodynamic condition was stable, and anticoagulant therapy with warfarin and aspirin was started on postoperative day 1. On postoperative day 2, the patient had massive diuresis of over 3000 ml (75 ml/kg), leading to a loss in body weight of 1.1 kg during a 24-h period. On postoperative day 23, although she remained alert, a left hemiparesis developed after three days of severe headache.

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Table I: Clinical data for patients who developed subdural hematoma after open-heart surgery.

Patient no.	Age (years)	Gender	Operative procedure	Anticoagulant therapy	Head trauma	Onset (POD)	INR at onset	Neurological symptoms	Surgical treatment	Outcome
1	61	F	MVR+TVR s/p PMI	WF + ASA	No	23	2.3	Headache Hemiparesis	Removal Drainage	Survived
2	70	F	re-AVR s/p AVR+MVR	WF + ASA	No	42	2.6	Headache Hemiparesis	Removal Drainage	Survived
3	68	F	AVR s/p MVR+TVR	WF + ASA Heparin	No	2	1.52	Somnolence	No	Survived
4	77	F	re-AVR s/p AVR+MVR	WF + ASA Heparin	Yes	3	1.27	Stupor Hemiparesis	Removal Drainage	Survived

ASA: Aspirin; AVR: Aortic valve replacement; INR: International Normalized Ratio; MVR: Mitral valve replacement; PMI: Pacemaker implantation; POD: Postoperative day; TVR: Tricuspid valve replacement; WF: Warfarin.

Emergency brain CT scanning demonstrated the presence of a subdural hematoma in the right convexity space. After discontinuation of warfarin with reversal by vitamin K, removal and drainage of the subdural hematoma were performed. Subsequently, the patient's neurological dysfunction showed a complete recovery.

Patient 2

A 70-year-old woman was admitted for replacement of a malfunctioning prosthetic valve in the aortic position. The patient had a history of aortic valve replacement (AVR) and MVR with a mechanical valve 24 years previously, after which she had remained on anticoagulant therapy with warfarin. At surgery, the aortic prosthetic valve was replaced with a bioprosthetic valve. Weaning from CPB was achieved with circulatory assistance using right-heart bypass and intra-aortic balloon pumping (IABP). A coagulation disability with thrombocytopenia (platelet count $4.4 \times 10^4 / \text{mm}^3$) required delayed sternal closure. The right-heart bypass and IABP were stopped on postoperative days 3 and day 5, respectively. Anticoagulation was effected with intravenous infusion of heparin during these six days, and subsequently maintained with warfarin and aspirin. On postoperative day 42, after a two-day period of severe headache, the patient's level of consciousness suddenly deteriorated and she fell into a stupor. Anisocoria of the pupil and right hemiparesis were also identified. Emergency brain CT scanning demonstrated subdural hematoma in the right convexity space (Fig. 1). After discontinuation of warfarin with reversal by vitamin K, removal and drainage of the subdural hematoma were performed under local anesthesia, whereupon the patient's consciousness and neurological symptoms promptly recovered. Anticoagulant therapy with heparin was restarted two days after surgery for the hematoma, and was switched to warfarin.

Patient 3

A 68-year-old woman was admitted for treatment of severe aortic valve stenosis. The patient had undergone MVR and TVR with a mechanical valve for rheumatic valve disease at the age of 42 years, and thereafter had remained on anticoagulation with warfarin. Hematologic examination and liver function tests indicated pancytopenia (platelet count $7.4 \times 10^4 / \text{mm}^3$) and Child-Pugh class A liver cirrhosis caused by type C hepatitis. At surgery, the aortic valve was replaced with a mechanical valve. The patient's postoperative hemodynamic condition was stable, and anticoagulant therapy with warfarin and aspirin was

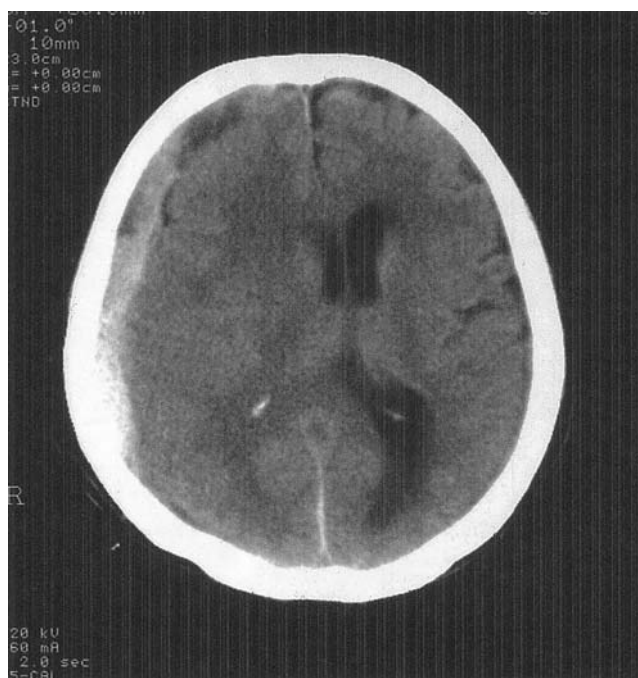


Figure 1: Patient #2. Brain computed tomography scan showing subdural hematoma in the right convexity space with a leftward shift of the midline.

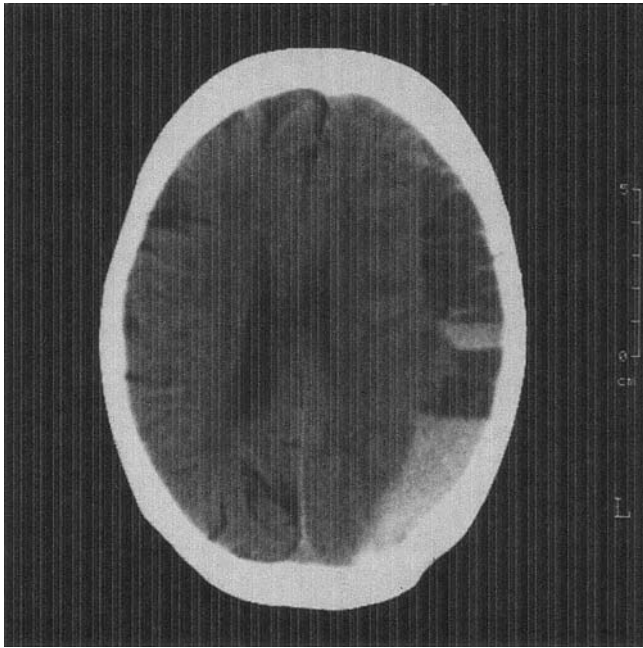


Figure 2: Patient #4. Brain computed tomography scan showing subdural hematoma.

restarted on postoperative day 1. On the next day, she fell into a state of somnolence without any neurological symptoms. Emergency CT scanning of the brain demonstrated subdural hematoma in the bilateral convexity space. The patient was managed conservatively by withdrawal of warfarin and aspirin, and continuous intravenous infusion of heparin was used to regulate the level of anticoagulant therapy. No expansion of the subdural hematoma was found by serial CT scanning, and no further aggravation of the neurological symptoms was observed. Anticoagulant therapy with warfarin was restarted on postoperative day 6.

Patient 4

A 77-year-old woman was admitted for prosthetic valve endocarditis in the aortic position, caused by *Staphylococcus capitis*. The patient had undergone open mitral commissurotomy at the age of 50 years, MVR at the age of 68 years, and AVR with a bioprosthetic valve seven months previously. She also had a history of a syncope attack accompanied by a fall two months before the present admission to hospital. At surgery, debridement of the infected annulus and replacement of the bioprosthetic valve with a new bioprosthesis were performed. The patient's postoperative hemodynamic condition was stable, and anticoagulant therapy with warfarin and aspirin was restarted on postoperative day 1. On postoperative day 3, her level of consciousness deteriorated and she fell into a stupor with right hemiparesis. Emergency CT scanning of the brain

demonstrated subdural hematoma in the left convexity space (Fig. 2). After stopping anticoagulation, removal and drainage of subdural hematoma were performed as an emergency procedure; the patient's consciousness and neurological symptoms promptly recovered. Anticoagulant therapy with heparin was restarted two days after the craniotomy, and was switched to warfarin.

Discussion

A variety of neurological complications may occur following an open-heart surgery, especially when CPB is utilized intraoperatively. The incidence of stroke and neurological complications after open-heart surgery has been recognized as being between 1.6% and 5.2% (1). However, most of the strokes are embolic events and hemorrhagic brain injury is rarely reported (3-7). Although acute subdural hematoma generally occurs as a result of trauma, it may also occasionally develop during or immediately after open-heart surgery (4,6). According to a Japanese study (3), eight cases (21%) of subdural hematoma were found among 39 cases of major cerebral dysfunction occurring within three years after cardiovascular surgery. In the present authors' experience, subdural hematoma occurred in the present four patients (0.4%) among a total of 1,100 who underwent cardiovascular surgery using CPB at their institution during the past five years.

Although, previously, postoperative over-anticoagulation has been suggested as the cause of subdural hematoma, the latter condition - in addition to cerebral infarction - has been suggested recently as a possible neurological complication directly related to cardiovascular surgery (3).

The exact etiology of subdural hematoma remains uncertain, however, and three pathogenic mechanisms have been proposed after cardiovascular surgery with CPB: (i) intraoperative or postoperative anticoagulant therapy with heparin or warfarin; (ii) head trauma; and (iii) rapid alterations in cerebral volume due to fluid shifts, leading to a tearing of the dural bridging veins (4,6).

A causal relationship between anticoagulant therapy and subdural hematoma has been documented. Subdural hematoma is clearly more frequent (1%) in patients receiving anticoagulant therapy than in those (0.03%) not receiving such therapy (8); moreover, the most common neurological complication of anticoagulant therapy is subdural hematoma, with an incidence of between 12 and 38% (9). In the present patients, a combination of warfarin and low-dose aspirin was used as postoperative anticoagulant therapy, according to recommendations in the ACC/AHA Practice Guidelines (10). A slight increase in risk of bleeding

with aspirin, mainly of gastrointestinal hemorrhage, has been indicated (10,11); however, the combination of warfarin and aspirin has further reduced both, the risk of thromboembolism and mortality due to other cardiovascular diseases (10), in patients with mechanical prostheses. Yet, the risk of bleeding with aspirin was increased when doses exceeding 500 mg/day were administered in combination with high-intensity warfarin (10,11). Although the lack of head trauma preceding subdural hematoma has been confirmed in half of those patients receiving anticoagulant therapy (8), trivial, forgotten trauma, and some type of external force on the body clearly exceeding everyday events has often been responsible in such cases. In the present four patients, in addition to anticoagulant therapy with warfarin and aspirin, the following events may have caused the subdural hematoma; a rapid decrease in fluid volume in patient #1; a coagulation disability or thrombocytopenia in patients #2 and #3; and the syncope attack accompanied by a fall in patient #4.

The diagnosis of subdural hematoma following open-heart surgery is often difficult because neurological complications may manifest in several ways, including failure to regain consciousness, prolonged drowsiness or stupor, or frank lateralizing neurological deficits. A variety of mechanisms has also been suggested to explain the development of these neurological phenomena. At present, subdural hematoma can only be accurately diagnosed by CT scanning. Hence, as brain injury is often slight in subdural hemorrhage after open-heart surgery, an early diagnosis by CT scanning and prompt treatment are of major importance to prevent catastrophic hemorrhagic brain injury in these cases.

In conclusion, four cases of acute subdural hematoma are described that occurred after valve replacement in patients receiving anticoagulant therapy. It is very important to bear in mind the possibility of an intracranial hematoma, whenever any neurological problems, such as disturbances of consciousness, are recognized after open-heart surgery and/or during anticoagulant therapy.

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