Cerebral Infarction after Deflation of a Pneumatic Tourniquet during Total Knee Replacement

Yukimasa Ogino, M.D.,* Yoshihisa Tatsuoka, M.D.,† Ryota Matsuoka, M.D.,‡ Kumi Nakamura, M.D.,§ Hitoshi Nakamura, M.D.,‡ Chiaki Tanaka, M.D.,∥ Nobuyuki Kamiya, M.D.,∥ Yuko Matsuoka, M.D.*

THROMBOEMBOLIC complications are common in orthopedic procedures when a tourniquet is used, but paradoxical embolism is rare. We report a case of cerebral embolism that developed immediately after tourniquet deflation in a total knee replacement procedure in a patient diagnosed with an asymptomatic atrial septal defect (ASD) postoperatively.

Case Report

A 76-yr-old woman was scheduled to undergo total left knee replacement during spinal anesthesia. The patient’s height and weight were 150 cm and 56 kg, respectively. Neither neurologic disease nor cardiovascular disease was evident in preanesthetic assessment. Preoperative chest radiography showed no active lesions, and her electrocardiogram (ECG) was of a normal sinus rhythm with a left axis deviation. The hemoglobin and hematocrit concentrations were 10.9 g/dl and 32.9%, respectively. The other laboratory examinations were normal.

Spinal anesthesia was established with dibucaine (3.6 mg) with epinephrine (0.2 mg). Oxygen was delivered via a face mask at 3 l/min. Electrocardiography, indirect blood pressure, and pulse oximetry were monitored.

After limb exsanguination, and wounding with an Esmarch bandage, a pneumatic tourniquet was inflated and kept at 350 mmHg for 108 min. Approximately 20 min after bone-cement implantation, the tourniquet was deflated promptly. Immediately after the deflation, the patient became unresponsive, even though she had been completely responsive up until then. Trembling of the lips, excessive salivation, and rightward conjugate deviation of the eyes were observed. Neither hypotension nor decreased oxygen saturation (SpO₂) were noted. At the end of the operation, 1 h after tourniquet deflation, further neurologic examination revealed left facial palsy, left upper-limb paresis, and global aphasia, with impairment of consciousness. (Her left lower limb paresis was confirmed later, after the effect of spinal anesthesia had worn off.)

Considering possible respiratory suppression during transfer of the patient, the trachea was intubated. Computed tomography of the brain was performed 2.5 h after the onset of the clinical deficits and showed no evidence of intracranial hemorrhage. Then, the arterial blood gas analysis was performed: pH: 7.27; pressure of oxygen (Pao₂): 68.9 mmHg; pressure of carbon dioxide (Paco₂): 43.7 mmHg; base excess: −6.1 mEq (fractional inspired oxygen tension [FiO₂]: 0.4). Mechanical ventilatory support was commenced later in the intensive care unit.

A selective antithrombin drug, argatroban (60 mg/day) and 10% glycerin were drip-infused. The activated partial thromboplastin time was between 45.4 and 81.8 s (normal values, 30.0–45.0 s), but no hemorrhagic complications were noted. On the second day postoperatively, mobility of the left upper and lower limbs showed some improvement. On the third day postoperatively, the patient regained consciousness and the trachea was extubated. The patient recovered with no remaining neurologic deficits on the fourteenth postoperative day.

Transesophageal two-dimensional, color flow Doppler echocardiography on the sixth postoperative day showed a normal right atrium size and an ASD of less than 3 mm in diameter, with a left-to-right shunt. Lung perfusion scintillation on the eighth postoperative day showed partial defects in the bilateral lower-lung regions, and no intrapulmonary shunt.

Although repeated computed tomography of the brain postoperatively showed no remarkable changes, magnetic resonance imaging on the fifty-eighth postoperative day revealed multiple subcortical small infarct lesions (fig. 1).

Discussion

It is well known that, because of venostasis distal to a tourniquet, microthrombi are formed in extremities during tourniquet inflation. The incidence of deep venous thromboembolism is reportedly higher than 50% in total knee replacement procedures. A thromboembolus that forms in extremities may cause pulmonary embolism when a tourniquet is deflated. However, cerebral embolism after tourniquet deflation has rarely been reported. In one reported case, a patent foramen ovale and systemic clot emboli were revealed at autopsy.
Paradoxical embolism can be caused in the existence of intracardiac shunts such as ASD or patent foramen ovale. Moreover, these shunts can be clinically asymptomatic. Shub et al. reported that 3.8% of ASDs are clinically unsuspected (“silent” ASD). According to the autopsy study of a Mayo Clinic group, the overall incidence of patent foramen ovale was 27.3% in normal hearts.

In our case, postoperative echocardiography showed a small ASD with a left-to-right shunt. Pulmonary embolism, if it occurred during the operation, could induce transient pulmonary hypertension, which might lead to right-to-left shunting. Therefore, we speculated that right-to-left shunting occurred transiently after tourniquet deflation.

Other authors have shown that, using transesophageal echocardiography, at the moment of tourniquet deflation, showers of echogenic materials commonly appear in the right side of the heart, and sometimes in the left side. It appears that, after tourniquet deflation in limb surgery, fragments of thromboemboli formed in veins distal to a tourniquet can enter the systemic circulation to a greater or lesser degree. In our case, small particles could have passed through the ASD when a transient right-to-left shunt occurred.

Aggressive antithrombin therapy with argatroban was commenced only 2 h after the operation had ended. This synthetic thrombin inhibitor, known to prevent cerebral spasm induced by thrombin, may have enhanced the patient’s rapid recovery of neurologic indicators. Conversely, Mohr et al. called the phenomenon of a short-lived deficit after the abrupt onset of a major hemispheric syndrome in patients with cardiogenic brain embolism, “a spectacular shrinking deficit (SSD).” Mineyama et al. suggested that, from their angiographic findings, an embolus initially introduced in a large artery of the carotid system migrated rapidly to its distal branches. In our case, although occlusion of major cerebral arteries was not evident, rapid recovery from a major hemispheric syndrome was compatible with spectacular shrinking deficit.

In conclusion, we suggest that potential risk of paradoxical embolism should be considered in the management of orthopedic procedures during tourniquet control because the incidence of clinically asymptomatic ASD or patent foramen ovale is not negligible.

References