Massive right main pulmonary embolism caused by tricuspid valve infective endocarditis

We describe a rare case of bacterial endocarditis with large vegetation and substantial pulmonary embolism. A 29-year-old woman who had acute renal failure after a septic abortion developed tricuspid valve endocarditis with large vegetation, which subsequently resulted in massive embolism to the right main pulmonary artery. The patient presented with symptoms of dyspnoea. Physical examination disclosed non-laboured breathing, with equal breath sounds bilaterally. Laboratory tests showed only leucocytosis and increased levels of C-reactive protein. Chest radiography showed a peripheral right and left infiltrate. Results of electrocardiography and other laboratory tests were unremarkable.

The initial transthoracic echocardiogram (TTE) detected the presence of a mobile mass (26 mm in diameter) attached to the posterior leaflet of the tricuspid valve and another mobile mass (24 mm in diameter) on the anterior leaflet. During subsequent TTE, the large mass on the posterior leaflet of the tricuspid valve was not seen. A CT scan of the chest was performed (figure 1A), showing the presence of right pulmonary artery emboli, which had originated from the tricuspid valve vegetations.

The patient was sent for emergency surgery. During surgery, extensive vegetation on the anterior leaflet of the tricuspid valve was found (figure 1B). The massive amount of vegetation and embolus was found in the right pulmonary artery, and pulmonary embolectomy was performed (figure 2A). The tricuspid valve was repaired with pericardium (figure 2B). No bacterium

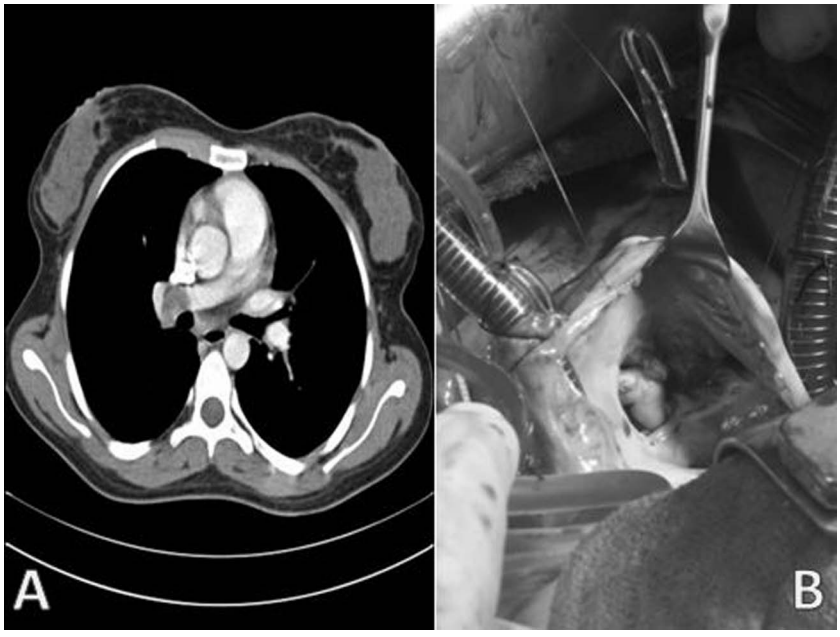

Figure 1 (A) CT scan showing right pulmonary artery embolism. (B) Extensive vegetation on the anterior leaflet of the tricuspid valve.

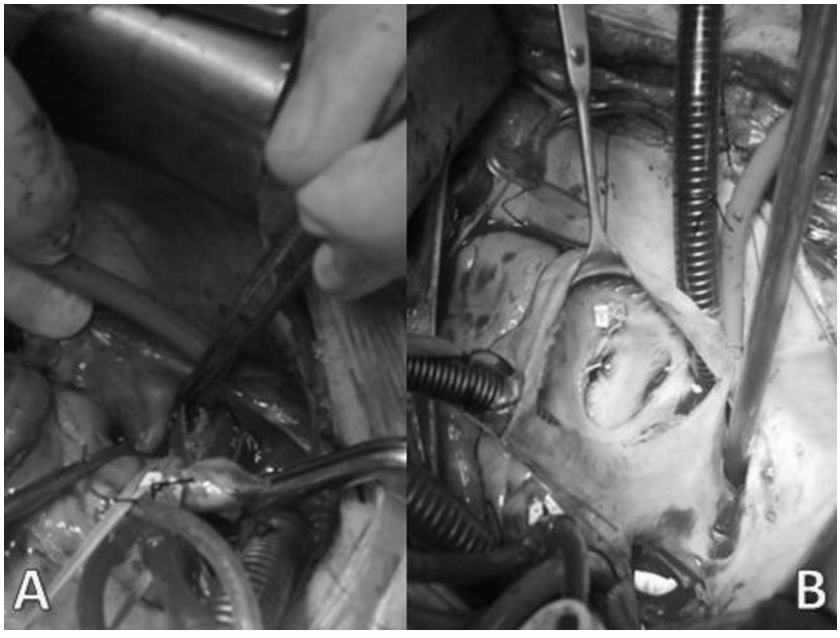

Figure 2 (A) At surgery, a massive embolism was seen at the right pulmonary artery. (B) Tricuspid valve reconstruction with pericardium.

was grown from the blood culture and specimen, but pathological specimens of the tissue showed fibrinous exudates with colonies of numerous cocci and chronic active inflammation. A combination of ampicillin and gentamicin was continued. After 6 weeks of combination treatment with antibiotics, the patient was discharged. After 6 months, a follow-up TEE showed that there was minimal tricuspid valve insufficiency.

In conclusion, in patients with endocarditis, a search for clinical and radiological signs of embolisation is necessary. The presence of both pulmonary and systemic embolisation resulting from bacterial endocarditis may cause death.

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