- 9. Garcha PS, Mohan CV, Sharma RM. Death after an inadvertent intrathecal injection of tranexamic acid. *Anesth Analg* 2007;**104**:241–2.
- Mahmoud K, Ammar A. Accidental intrathecal injection of tranexamic acid. *Case Rep Anesthesiol* 2012. http://dx.doi.org/ 10.1155/2012/646028.
- Mohseni K, Jafari A, Nobahar MR, Arami A. Polymyoclonus seizure resulting from accidental injection of tranexamic acid in spinal anesthesia. *Anesth Analg* 2009;**108**:1984–6.
- Murkin JM, Falter F, Granton J, Young B, Burt C, Chu M. High-dose tranexamic acid is associated with nonischemic clinical seizures in cardiac surgical patients. *Anesth Analg* 2010;110:350–3.

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- 13. Yamaura A, Nakamura T, Makino H, Hagihara Y. Cerebral complication of antifibrinolytic therapy in the treatment of ruptured intracranial aneurysm. Animal experiment and a review of literature. *Eur Neurol* 1980;19:77–84.
- 14. Furtmüller R, Schlag MG, Berger M, et al. Tranexamic acid, a widely used antifibrinolytic agent, causes convulsions by a gammaaminobutyric acid(A) receptor antagonistic effect. *J Pharmacol Exp Ther* 2002;**301**:168–73.
- Herroeder S, Schönherr ME, De Hert SG, Hollmann MW. Magnesium-essentials for anesthesiologists. *Anesthesiology* 2011;114:971–93.
- Euser AG, Cipolla MJ. Magnesium sulfate for the treatment of eclampsia: a brief review. *Stroke* 2009;40:1169–75.

Early postpartum mitral valve thrombosis requiring extra corporeal membrane oxygenation before successful valve replacement



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ABSTRACT

Pregnancy is associated with an increased risk of thrombosis in women with mechanical prosthetic heart valves. We present the case of a 29-year-old woman who developed early postpartum mitral valve thrombus after an elective cesarean delivery. The patient had a mechanical mitral valve and was treated with warfarin in the second trimester, which was replaced with high-dose dalteparin during late pregnancy. Elective cesarean delivery was performed under general anesthesia at 37 weeks of gestation. The patient was admitted to the intensive care unit for postoperative care and within 30 min she developed dyspnea and hypoxia requiring mechanical ventilation. She deteriorated rapidly and developed pulmonary edema, worsening hypoxia and severe acidosis. Urgent extra corporeal membrane oxygenation was initiated. Transesophageal echocardiography revealed a mitral valve thrombus. The patient underwent a successful mitral valve replacement after three days on extra corporeal membrane oxygenation. This case highlights the importance of multidisciplinary care and frequent monitoring of anticoagulation during care of pregnant women with prosthetic heart valves.

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Introduction

In patients with mechanical heart valves, pregnancy increases the risk of valve thrombosis. As anticoagulation is needed, maternal hemorrhage and fetal complica-

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tions, such as congenital malformations, hemorrhage and death may occur.¹⁻⁴ The choice of an appropriate anticoagulation agent during pregnancy in these women remains a concern. In a retrospective study between 1997 and 2008, the risk of a thromboembolic event in women with mechanical prosthetic heart valves was found to be 14.9% (7/47) and in the majority it was associated with enoxaparin treatment.³ An older review published in 2000, in which a large number of women had older generation and more thrombogenic valve prostheses, showed higher rates (64%) of fetal malformation in women treated with oral anticoagulation in the first trimester than in women who were switched to heparin (3.4%).⁵ It is clear that the management of women with mechanical heart valves during pregnancy requires risk counseling, judicious monitoring and multidisciplinary care. Moreover, when complications arise, these patients can deteriorate rapidly as this case report reveals.

Case report

The patient was a 29-year-old-woman with a body mass index of 25 kg/m^2 . At the age of six, she underwent a mitral valve replacement (Björk-Shiley 27 mm) for mitral regurgitation due to rheumatic valve disease. She had a history of one first trimester miscarriage. Two years before the current pregnancy she underwent an urgent cesarean delivery at 35 weeks of gestation. She had been admitted with severe preeclampsia and developed hemolvsis, elevated liver enzymes and low platelets (HELLP) syndrome less than 24 h after admission. Cesarean delivery was performed under general anesthesia after reversal of warfarin with prothrombin concentrate 2000 IU and vitamin K 2 mg. Postoperatively she was treated with an unfractionated heparin (UFH) infusion. On the first postoperative day, she underwent a laparotomy because of severe venous bleeding.

During the current pregnancy, she was cared for by a multidisciplinary team consisting of obstetrics, cardiology, hematology and anesthesia and her anticoagulation was under strict control according to European guidelines for anticoagulation in pregnant women with prosthetic valves.⁶ Pre-pregnancy she was on warfarin but anticoagulation was changed to dalteparin in the fourth week of pregnancy. During the second trimester this was changed back to warfarin. At 34 weeks of gestation, anticoagulation was switched back to dalteparin. In addition, she was treated with acetylsalicylic acid 75 mg throughout pregnancy. Weekly dose adjustments of dalteparin were made according to anti-factor Xa measurements which were taken before and 4 h post subcutaneous injection. The patient was receiving subcutaneous dalteparin 12500 U twice daily and acetylsalicylic acid at 37 weeks when an elective cesarean delivery was performed under general anesthesia. She received her last dalteparin injection 24 h before surgery.

An arterial line was placed before induction of anesthesia. A remifenatanil infusion at 0.5 µg/kg/min was given for 45 s before administration of thiopental 500 mg followed by succinvlcholine 100 µg. The patient was intubated and anesthesia was maintained with sevoflurane and a remifentanil infusion. A baby girl was delivered with Apgar scores of 8 and 10 at 1 and 5 min, respectively. At the completion of surgery, the patient was extubated and transferred to the intensive care unit (ICU). Total blood loss was 1000 mL. She received 500 mL colloid and 600 mL crystalloid during surgery. Within 30 min of arrival in the ICU she complained of dyspnea and her oxygen saturation started to decrease. Transthoracic echocardiography (TTE) was performed which revealed a pressure gradient through the mitral valve which was three times higher than on previous TTE: median pressure was 25 mmHg. No thrombus was visualized. A chest X-ray showed pulmonary congestion. The patient deteriorated dramatically and developed pulmonary edema with a saturation of 83% despite intravenous furosemide and non-invasive ventilation with 100% oxygen. Arterial blood gases showed pH 7.03, base excess -11.1 mEq/Land lactate 2.8 mmol/L. She was intubated and mechanical ventilation started. Transesophageal echocardiography (TEE) confirmed a high pressure gradient through the mitral valve prosthesis but the suspected thrombus was not seen, possibly due to poor visualization. The left ventricle was found to be hyperdynamic and small. Her metabolic acidosis worsened and she was severely hypoxic with a saturation of 70%. Arterial blood gases revealed a PaO₂ of 6.03 kPa on an FiO₂ of 1.0 and a PaCO₂ of 11.5 kPa. She was hypotensive and started on a low-dose norepinephrine infusion (<0.1 µg/kg/min) which resulted in a relatively stable blood pressure of between 90 and 120/60 and 65 mmHg.

An UFH bolus of 5000 U and infusion of 1250 U/h was started and veno-arterial extra corporeal membrane oxygenation (ECMO) was established via peripheral cannulation approximately 2 h after arrival in the ICU. The activated partial thromboplastin time (APTT) target was >100 s. Her APTT was measured every 4 h and values were between 92 and 122 s. The ECMO system provided satisfactory circulatory and respiratory support. The patient was transferred to the cardiothoracic ICU. Within 6 h her norepinephrine infusion was no longer needed and her blood gases were pH 7.42, PaO₂ 13.3 kPa and PaCO₂ 5.35 kPa. Her lactate was 2.4 mmol/L. A fluoroscopic examination demonstrated severe dysfunction of the prosthetic mitral valve with obstruction of disc movement. Systemic thrombolytic therapy was not given because of the risk of bleeding due to her recent cesarean delivery.

On day 1 postoperatively, following an unsuccessful ECMO weaning attempt, TEE demonstrated a thrombus obstructing the mechanical mitral valve prosthesis, Download English Version:

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