



Livedoid Vasculopathy and Anesthetic Management in Cesarean Delivery

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ABSTRACT

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Livedoid vasculopathy (LV) is a hyalinizing vascular disease characterized by painful purple macules and papules that subsequently ulcerate. This vasculopathy may be associated with chronic venous insufficiency, deep venous thrombosis, factor V Leiden mutation, protein C deficiency, antiphospholipid syndrome, increased homocysteine levels, abnormalities in fibrinolysis, increased platelet activation and sickle cell disease. Difficult venous access, unreliable measurement of peripheral O₂ saturation and increased susceptibility to venous embolic events may be a challenge for anesthetists. There is limited data about anesthetic management of livedoid vasculopathy in the literature. This case report describes successful anesthetic management of two patients with livedoid vasculopathy.

Key words: Anesthesia, spinal, vasculopathy, livedoid, LMWH

ÖZET

Livedoid vaskülopatide anestezi ve düşük molekül ağırlıklı heparin kullanımı

Livedoid vaskülopati (LV) hyalinizan vasküler hastalık olup ağır mor maküller, papüller ve ulserasyonla karakterizedir. Bu vaskülopati kronik venöz yetmezlik, derin ven trombozu, Faktör V Leiden mutasyonu, protein C eksikliği, antifosfolipid sedrom, artmış homosistein seviyeleri, anomal fibrinoliz, artmış platelet aktivasyonu ve orak hücre hastalığı ile ilişkilidir. Venöz girişim güçlüğü, güvenilmez periferal O₂ saturasyonu venöz embolik olaylara artmış yatkınlık anestezistin başetmesi gereken olaylardır. Literatürde LV'de anestezi ile ilgili sınırlı bilgi bulunmaktadır. Bu vaka sunumu LV'li 2 hastanın başarılı anestezi yöntemini sunmaktadır.

Anahtar kelimeler: Anestezi, spinal, vaskülopati, livedoid, LMWH

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INTRODUCTION

Livedoid vasculopathy (LV) is a rare disease and primarily effect female individuals. This vasculopathy was firstly described by Bartd and Winkelmann in 1967 (1). It is characterized by chronic nature and periodic and recurrent exacerbations. It has a multifactorial nature and mainly affects the lower extremities. It is a hyalinizing vascular disease characterized by painful purple macules and papules that subsequently ulcerate. This

vasculopathy may associate with chronic venous insufficiency, deep venous thrombosis, connective tissue diseases, solid organ carcinomas, hematologic malignancies, factor V Leiden mutation, protein C deficiency, antiphospholipid syndrome, increased homocysteine levels, abnormalities in fibrinolysis, increased platelet activation and sickle cell disease (2). Difficult venous access, unreliable measurement of peripheral oxygen saturation and increased susceptibility to venous embolic events may be a challenge for anesthetists (3).

In literature there is limited knowledge about anesthetic management. This case report describes successful anesthetic management of two patients with LV using low molecular weight heparin (LMWH). The patients gave written permission for the authors to publish this report.

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CASE 1

A 27-years-old woman, weight 76 kg, height 163 cm, gestational week 39 was admitted to our hospital for planned cesarean delivery. Livedoid vasculopathy was diagnosed 8 years ago and treated with colchicine and low molecular weight heparin (LMWH) at active periods. She had active periods every summer for 8 years. During pregnancy at the first and second trimester she was treated with only acetylsalicylic acid 100 mg per os daily. At the third trimester she was no treated by anticoagulant. The first two trimesters were uneventful and at third trimester lesions activated during summer period. Laboratory exams were in the normal ranges except mild anemia (hemoglobin level was 8.1 g/dL). Protein S level and Protein C level were 85 and 88 IU/dL respectively. She had old maculopapular pustule, atrophie blanche at lower extremities and cutis marmoratus at all extremities. Her all four extremities skin was pale and cold. She had 1/6 diastolic murmur. Her effort capacity was New York Heart Association Class-II and metabolic equivalent task was 7. She had uneventful cesarean section by spinal anesthesia 4 years ago. Spinal anesthesia was decided due to patient clinical status. SpO₂ was monitorised and anesthesia was uneventful. SpO₂ level was normal during operation period. Twelve hours after the operation, the patient treated with acetylsalicylic acid 100 mg po and LMWH twice a day started. After 3 days, the patient was discharged home at usual time.

CASE 2

A 40-years-old man was admitted to our hospital for planned inguinal hernia repair. He had LV treated with colchicine, pentoxifylline, and Coumadin. Coumadin was discontinued 1 week before the operation. He had old maculopapular pustule, atrophie blanche at lower extremities. Twelve hours before the operation low-molecular-weight heparin (LMWH) (enoxaparin 0.4 ml sc) was administered. Before the anesthesia induction arterial line was placed to the left radial artery. After preoxygenation, anesthesia was induced and maintained with remifentanil infusion 0.1-0.25 mcg/kg/min and propofol infusion 3- 10 mg/kg/h. Airway maintenance was obtained with laryngeal mask airway.

Operation was uneventful and 24 hours after

operation the patient was treated with Coumadin. Plethysmography results were correlated with arterial oxygen saturation results from arterial blood gas analysis (pH:7.4, pCO₂:39.9, pO₂:99.8).

DISCUSSION

LV is characterized by chronic nature and periodic and recurrent exacerbations. LV is an uncommon condition worldwide and can occur during pregnancy due to the decrease in protein C and S levels. Our both cases had normal Protein C and S levels before surgery. The main pathogenic mechanism is thought to be due to vaso-occlusive phenomenon. Thrombosis of the dermal blood vessels leads to tissue ischemia as seen by a decrease in cutaneous oxygen tension (3). Therefore unreliable SpO₂ measurements can be seen at these patients.

Anesthetic management of patients with LV must be considered individually. We planned spinal anesthesia in first case because of unreliable SpO₂ measurements, prothrombotic state and choice of the patient. At the second case, patient refused regional anesthesia and we placed an arterial cannula for the confirmation of SpO₂ levels.

Most of LV patients are on anticoagulant therapy and should be evaluated carefully before surgery. In recent cases, despite the patients' normal coagulation profile, hypercoagulability and vascular thrombosis were the main risk factors because of increased susceptibility. In both cases patients received LMWH, 12 hours before operation and after the operation. Neuroaxial anaesthesia compared to general anesthesia is related with decreased risk of deep venous thrombosis and pulmonary embolism (4). Antiplatelet agents, warfarin, unfractionated heparin and LMWH are options for long term therapy against LV. LMWH has been widely used for long term remissions (5). Warfarin and unfractionated heparin use is associated with excessive surgical bleeding and has difficulties within monitorisation. LMWH use at the perioperative period has been shown to decrease the thromboembolic events with minimal increase surgical bleeding.

Spinal or general anaesthesia are both suitable options for LV patients when risk factors are fully evaluated. For the risk of bleeding or thromboembolic events, screening of the coagulation profile, cessation of

long acting anticoagulant drugs, transition to LMWH therapy during perioperative period, antiembolic stockings, and anesthetic strategy for early ambulation must be kept in mind.

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