Anesthetic management of patient with systemic lupus erythematosus and antiphospholipid antibodies syndrome for laparoscopic nephrectomy and cholecystectomy

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ABSTRACT

We report a case of a female having systemic lupus erythematosus and antiphospholipid antibodies syndrome, who was on immunosuppressant therapy. We discussed the preoperative evaluation and perioperative management who underwent nephrectomy and cholecystectomy.

Key words: *Anesthetic management, antiphospholipid syndrome, systemic lupus erythematosus*

INTRODUCTION

Systemic lupus erythematosus (SLE) is an autoimmune disease with heterogeneous presentation and with systemic involvement in which tissues and multiple organs are damaged by pathogenic autoantibodies and immune complexes.[1] It is characterized by the presence of autoantibodies directed against nuclear antigens. The prevalence of SLE varies with ethnicity, but is estimated to be about 1/1000 overall; with female to male ratio of 10:1, the peak age of onset being between 15 and 40 years.[2-4]

We need the presence of four of the following to diagnose SLE: Discoid rash, photosensitivity, oral ulcers, arthritis, serositis (pleuritis and pericarditis), renal involvement, neurologic disorder (seizures and psychosis), immunologic disorder (hemolytic anemia, leucopenia, and thrombocytopenia), characteristic facial malar rash, and immunologic disorder. Hypercoagulable state may be present.[3] Antinuclear antibody and anti-double stranded DNA (dsDNA) are positive with very high titers, and serum complement levels (C3, C4) are low. As it is a multisystem disorder, a thorough preanesthetic evaluation is essential for safe anesthesia management. Anesthetic plan must be individualized based on the degree of the involvement of the various systems, current medications the patient is taking, and on the laboratory investigations.[3] We present the perioperative management of a patient with a history of SLE posted for laparoscopic nephrectomy and cholecystectomy.

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CASE REPORT

We report a case of a 21-year-old, 70 kg female having SLE and antiphospholipid (APL) syndrome for 2 years, who was scheduled for laparoscopic nephrectomy and cholecystectomy. She was in her usual state of health before 2 years when she developed right superficial femoral and popliteal deep venous thrombosis twice, and she was anticoagulated with warfarin for 2 months. After 3 months, she developed two episodes of left hemiparesis due to right frontal lobe ischemic stroke. Thrombophilic screening done only anticardiolipin antibodies were positive. After 1 year she developed sudden acute chest

pain, and cardiac enzymes were mildly elevated. After 3 months of that event she presented in an emergency with epigastric ad right flank pain, ultrasonography abdomen showing right hydronephrosis. Renal function tests were moderately elevated, and emergency right nephrostomy tube was placed. On further evaluation, she diagnosed to have lupus nephritis chronic kidney disease stage 3 and positive APL antibodies and dsDNA antibodies. Right ureteric stricture was present and on dimercaptosuccinic acid cortical scan right kidney function was 20%. Multiple gall bladder stones noticed.

Preoperatively patient is having hypertension, lupus nephritis, APL syndrome, SLE, and perimyocarditis. Laboratory results were within normal limits. Chest X-ray was unremarkable. Electrocardiogram (ECG) showing left ventricular hypertrophy and echocardiogram was normal. She was on low molecular weight heparin, calcium channel blocker, angiotensin converting enzyme inhibitor, azathioprine, hydrocortisone, and opioid analgesics. All medications continued up to day of surgery. After applying all standard monitoring, she induced with good dose opioid, etomidate, and cisatracurium. Difficulty encountered in passing even 7.0 mm endotracheal tube (ETT), so intubated with 6.5 mm ETT, and excessive neck movements were avoided. Radial arterial cannulation done. Foley urinary catheter and orogastric tube placed and patient positioned for surgery. Depth of anesthesia was maintained with sevoflurane and infusions of remifentanil and cisatracurium. Intermittent intravenous boluses of labetalol given to lower the blood pressure. Degree of muscle relaxation monitored by peripheral nerve stimulator and urine output monitored per hour basis. Intraoperative blood loss was 500 ml and urine output was 80 ml/h and 300 ml positive fluid balance. After 3 h of surgery patient extubated and transferred to high dependency unit, postoperative pain managed effectively with morphine patient-controlled analgesia.

DISCUSSION

From our experience and systematic review of the literature[1,3] suggests that perioperative management must be tailored to the individual patient. Anesthetic management plan was made after taking into account severity of the disease, the potential drug interactions with immunosuppressants, an unexpected difficult airway with subglottic stenosis or laryngeal edema, and coagulation profile of the patient.

Patients with SLE have a variety of abnormalities of varying intensity. Therefore, there are a host of presentations, and the course is highly variable, ranging from relatively mild and uncomplicated to major life-threatening disease. Cardiovascular involvement could be in the form of pericarditis, myocarditis, arthrosclerosis, and the myocardial ischemia.[1] Pulmonary involvement could vary from pleuritis, pleural effusion, alveolar hemorrhage, and interstitial lung disease.[5] Renal involvement is seen in the form of lupus nephritis characterized by proteinuria, hematuria, and abnormal urinary segments.[6] As the present in our case, patients with SLE are at high risk of hypertension renal status. The risk may increase in case the patient requires more than 30 mg of prednisolone daily.[2] Our patient was on high-dose prednisolone and developed hypertension.

A 37-95% of SLE patients may manifest central and peripheral nervous system complications. American College of Rheumatology recommends the term neuropsychiatric SLE to encompass all possible manifestations which may vary from headaches, seizures, cerebrovascular disease, psychosis, acute confusional states to even demyelinating disease states.[3] Our patient suffered twice left hemi paresis and improved by physical rehabilitation. Hematological manifestations commonly seen in SLE include anemia, thrombocytopenia, and leucopenia. Anemia is found in about half of SLE patients with the most common cause being anemia of chronic disease; however, other causes include autoimmune hemolytic anemia, iron deficiency anemia, anemia of chronic renal failure, and cyclophosphamide myelotoxicity.[7] Nonerosive arthritis is seen in patients with SLE. Prolonged glucocorticoid use for immunosuppression could cause osteoporosis. Incidence of atlantoaxial subluxation has been reported.[8]

Antiphospholipid syndrome may occur secondary to SLE and is characterized clinically by recurrent pregnancy loss and by presence of lupus anticoagulant antibodies which may falsely prolong activated partial thromboplastin time in such individuals.[9]

The preoperative visit aims at the activity of the lupus, organ damage, medication exposure, thorough preanesthetic assessment, and laboratory test. Care of the high-risk patients requires a multidisciplinary approach.

As SLE symptoms are nonspecific, the investigations become mainstay in monitoring. Complete blood count has to be done in all patients alongside coagulation profile. Platelet count should be repeated every month because of high risk of thrombocytopenia in lupus patients.[7] Electrocardiography may be done when suspecting pericarditis, myocarditis, and chest X-ray may be reserved for extreme cases where pleural effusion or interstitial pneumonitis is seen clinically.[2] For patients with renal involvement, every month creatinine clearance

and 24 h urine protein should be checked. If the patient is on steroids then, a close watch on blood glucose levels is advocated. Anticardiolipin antibody, lupus anticoagulant, anti-β2 glycoprotein should be done to rule out any secondary involvement in succeeding months.

Monitoring during anesthesia includes five-lead ECG, noninvasive blood pressure, pulse oximetry, and invasive monitoring should be used in patients with myocarditis, valvular involvement, or conduction abnormalities. Renal protective strategies and maintenance of urine output, avoidance of nephrotoxic drugs are the goals during anesthesia. Adequate pain management and corticosteroid cover should be given intraoperatively to prevent adrenal suppression. Antibiotics are to be given to prevent infection. Patient should be positioned with care to avoid joint stress.

Difficult airway should be anticipated in all the patients, smaller sized tubes, and laryngeal mask airway must be available considering the potential laryngeal and the subglottic involvement. Laryngeal involvement could vary from mild inflammation to laryngeal edema, epiglottis, and vocal cord paralysis[10] to acute airway obstruction. The pathophysiology of laryngeal inflammation of SLE is not well-understood although the tissue deposition of immune complexes with activation of complements is less likely the cause. Compression of recurrent laryngeal nerve by dilated pulmonary artery has been reported as the cause of left palsy in patients with SLE. Secondary nerve vasculitis is believed to be a cause especially in vocal cord palsy involving right side.[11] There is a significant risk of failed intubation and airway trauma during instrumentation. Pharmacological interactions between anesthetic drugs and immunosuppressant drugs should warrant consideration. Azathioprine, an antimetabolite immunosuppressor, may interact with muscle relaxants, and dose increases of 37% with cisatracurium, 20% with vecuronium, and 45% with pancuronium were required in one-study.[12]

To conclude, with proper understanding of the pathophysiology and systemic organ involvement with SLE, these patients can be managed successfully.

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