ANESTHETIC CONSIDERATIONS FOR THE PATIENT WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Systemic lupus erythematosus (SLE) is a complex disorder characterized by dysregulation of pathogenic autoantibodies and immune complexes that leads to multisystem chronic inflammatory processes[1]. SLE is not a rare condition; the estimated prevalence is 100 physician-diagnosed patients per 100,000 people. The disease may present at any age, although it primarily affects women of reproductive ages. The ratio of female to male patients is 9:1.

The prevalence of SLE is described as having an ethnic component, with black women affected 3 times more than whites[2]; in addition, blacks and Hispanics are reported to have higher rates of morbidity[3]. Although a classical presentation of SLE has been described, clinically there are many variations of the disease. For example, elderly patients tend to have a less-severe form involving fewer organ systems overall; men usually experience less photosensitivity but have a higher rate of mortality[4,5].

SLE was first documented in the Middle Ages when it was termed lupus ("wolf" in Latin) to describe the appearance of the classical facial (malar) rash. It was suggested that the rash resembled the fur on the forehead and muzzle of the wolf. Others have suggested that the disease may have been named after a veil (loup) used by women in France to cover facial blemishes. It was not until 1872 that Móric Kaposi, a Hungarian dermatologist, began to recognize and describe the systemic manifestations of the disease[6].

A groundbreaking advance in the study of lupus was made when Malcolm Hargraves, a hematologist at Mayo Clinic in 1948, described the lupus erythematosus or LE cell found in the bone marrow of patients. Ten years later, George Friou, MD, developed a test using fluorescent antihuman globulin demonstrating the antigen–antibody reaction, thus advancing the immunologic study of the disease[7]. Although SLE is largely attributed to autoimmune processes, its pathogenesis can be induced by drugs. This feature of SLE was discovered at the Cleveland Clinic in 1954, when a patient who was treated with hydralazine for hypertension subsequently developed SLE[8].

Clinical Manifestations

Considerable variation exists in the clinical presentation of SLE, ranging from acute features with the classical malar, erythematous "butterfly rash" to a progressive fatal illness most commonly caused by complications of renal, cardiovascular, pulmonary, and central nervous

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system (CNS) pathologies[9]. Across all age groups, SLE is characterized by chronic, inflammatory, multiorgan symptoms caused by immune complexes and antibodies against cell surface molecules or serum constituents[10]. The level of involvement of each organ system varies (Figure).

Mucocutaneous involvement is the most commonly reported clinical feature. It can appear as a rash (acute to chronic), alopecia, photosensitivity, and pathology of mucous membranes[4]. SLE patients commonly have manifestations found in other autoimmune diseases, such as Raynaud's phenomenon, sclerodactyly, rheumatoid nodules, and erythema multiforme[11]. These manifestations are secondary to activation of the membrane attack complex and immune complex deposition[12].

Musculoskeletal symptoms play a major role in the pathogenesis of SLE, affecting 53% to 95% of cases[4]. These include arthritic, arthropathic, myositic, and necrotic processes. Some complications arise from immunoglobulin deposition; others may be a result of corticosteroid treatment or hematologic pathogenesis.

Hematologic involvement is a common characteristic of SLE. It is defined variously as anemia, leukopenia, thrombocytopenia, and antiphospholipid syndrome. Most patients with SLE have anemia secondary to many causes, including immune-mediated hemolysis, chronic disease, renal insufficiency, aplastic anemia, hypersplenism, blood loss, myelodysplasia, myelofibrosis, and medication use. Thrombocytopenia in these patients can result from platelet destruction, microangiopathic hemolytic anemia, hypersplenism, bone marrow suppression, and thrombopoietin antibodies. SLE is commonly complicated by leukopenia-either neutropenia or lymphocytopenia. Antiphospholipid syndrome may coexist with SLE, causing thrombosis and vascular disease[4].

Renal symptoms affect 40% to 70% of patients[4]. Mild, asymptomatic disorders of the urinary system affect many patients. A small percentage of cases progress to chronic renal insufficiency, a renal vasculitis syndrome, or severe lupus glomerulonephritis[13].

Perhaps the most debilitating complications seen in SLE are those affecting the peripheral nervous system and CNS. The American College of Rheumatology (ACR) classifies these manifestations as neuropsychiatric systemic lupus erythematosus syndromes (NPSLE)[14]. The ACR has designated 19 syndromes within the NPSLE group. CNS syndromes include cerebrovascular disease, demyelinating syndrome, myelopathy, seizure disorder, psychosis, and aseptic meningitis. Peripheral nervous system syndromes include Guillain-Barré syndrome, mononeuropathy, myasthenia gravis, and cranial neuropathy[4].

Cardiovascular involvement is variable. Chronic inflammation and autoantibodies accelerate atherosclerosis by injuring endothelial cells and altering lipoproteins[15]. The correlation between early, severe atherosclerosis and SLE, leads to an increased prevalence of coronary artery disease, myocardial infarction, and stroke in these patients[16]. Approximately 25% of patients with SLE develop pericarditis, whereas myocardial pathology is reported in less than 5% of patients[4]. Additionally, SLE increases the risk for valvular heart disease defined as aortic and mitral valve thickening, vegetations, regurgitation, and stenosis[17].

Pulmonary involvement includes pleural, parenchymal, vascular, and muscular manifestations. The most common respiratory finding is pleuritic pain. Pleuritis is reported in more than 50% of patients with SLE. Clinically insignificant pleural effusions often are diagnosed. A more debilitating complication, although rare, is interstitial lung disease; its severity ranges from mild inflammation to extensive fibrosis[18]. Reports of other types of parenchymal involvement include acute pneumonitis secondary to alveolar wall necrosis, bronchiolitis obliterans, pulmonary hypertension, and infection due to immunosuppression[19]. Perhaps most worrisome is pulmonary hemorrhage secondary to inflamed capillaries, a relatively rare complication that has a mortality rate as high as 90%[20]. A late pulmonary consequence of SLE is diaphragmatic pathology. This complication, known as "shrinking lung syndrome", causes decreased total lung capacity and volume[21].

Infections play an important role in the morbidity and mortality in SLE. Disruption of normal immunity, chronic inflammation, and immunosuppressive therapy make these patients particularly susceptible. Complement deficiencies occur in SLE due to immune

complexes activating the classical pathway. These deficiencies increase vulnerability to encapsulated bacteria and disseminated *Neisseria* infections[22]. Tuberculosis and Herpes Simplex Virus infections are also more prevalent among SLE patients. However, most often the respiratory and urinary tracts are involved by gram-negative and gram-positive bacteria[23,24].

As supported by recent evidence, patients with SLE have an increased risk for cancer, including non-Hodgkin's lymphoma and lung, breast, and cervical malignancies. Although there is an association between malignancy and SLE, the pathogenic mechanisms are unknown. It has been suggested that genetic and environmental factors play a role[25].

Pathogenesis

The pathogenesis of SLE is complex. Main factors include genetic patterns, gender, and environmental risks. Despite the different presentations of SLE, each patient shares a common dysregulation of autoantibody activity and an increased amount of immune complexes. Functionality at every level of the immune system is affected. Abnormalities in B cells, T cells, and immunoregulatory pathways have been described. [The unchecked production of these self-destructing molecules causes widespread inflammatory processes leading to a common theme of damaged organ systems]. The unchecked activation of inflammatory processes and resultant production of cytokines, most notably INF-α, are responsible for the systemic damage of organs[26,27].

In SLE, many autoantibodies have a pathogenic role, targeting DNA, RNA, cell membrane structures, the cellular surface, and intracellular molecules[1]. The most prevalent self-destructing molecules are within the antinuclear antibody (ANA) group. The hypothesis supported by increasing evidence is that these antibodies originate from antinucleosomal antibodies[28]. A main concern is the effect of the anti-DNA antibodies on renal parenchyma. The antibodies directly bind to or form complexes with various renal components, such as heparin sulfate proteoglycan, laminin, α-actinin, histone proteins, and glomerular basement membrane collagen[29,30]. In SLE, anti-DNA molecules also attack the CNS. These antibodies target neurons and cause apoptosis, leading to cognitive impairment, altered mental status, and deterioration in mood[31]. Anti-phospholipid antibodies also contribute to neurologic dysfunction and are associated with an increased risk of strokes, seizures, and migraines[32].

Other autoantibodies specific to cellular types cause complications in patients. A common self-destructing molecule with up to 25% prevalence is the anti-Smith autoantibody, another ANA subtype. Highly specific for SLE, this autoantibody acts as an accelerator of disease[1]. Similarly, anti-Ro autoantibody has a particularly important part in the pathogenesis of SLE and is associated with nephritis, dermatitis, vasculitis, neonatal lupus, and Sjögren's syndrome[1].

With a variable severity of disease, some autoantibodies cause hematologic pathology; antibodies against platelets cause thrombocytopenia. More specifically, these antibodies are targeted against platelet cytoplasmic and surface components, including phospholipids and glycoproteins II and III[33]. The immunoglobulin G non-Rhesus antibody against an erythrocyte surface molecule contributes to SLE by causing hemolysis and anemia[34]. Additionally,

Table 1
Environmental Factors Associated With Pathogenesis of SLE

Ultraviolet B light
Epstein-Barr virus
Estrogen and prolactin: Predilection for females (9:1 ratio, female: male)
Lupus-inducing medications Hydralazine Procainamide Isoniazid Hydantoins Chlorpromazine Methyldopa Penicillamine Minocycline Tumor necrosis factor-α inhibitors Interferon-α
Dietary factors: Alfalfa sprouts and related sprouted foods containing canavanine, pristane
Infectious agents other than Epstein-Barr virus
Bacterial DNA
Human retroviruses
Endotoxins, bacterial lipopolysaccharides

SLE, systemic lupus erythematosus
Adapted from reference 1.

Table 2
ACR Classification Criteria for SLE

Criteria	Description
ANA	Abnormal titer of ANA by immunofluorescence or equivalent assay at any time and in the absence of drugs known to be associated with drug-induced lupus syndrome
Arthritis	Non-erosive arthritis involving 2 or more peripheral joints and characterized by tenderness, swelling, or effusion
Discoid rash	Erythematous, raised patches with adherent keratotic scaling and follicular plugging; atrophic scarring occurs in older lesions
Hematologic disorder	Hemolytic anemia with reticulocytosis, or Leukopenia: <4,000/mm^3, or Lymphopenia: <1,500/mm^3, or Thrombocytopenia: <100,000/mm^3 in the absence of contributing medications
Immunologic disorder	Anti-DNA: antibody to native DNA in abnormal titer, or Anti-Smith: presence of antibody to Smith nuclear antigen, or Positive finding of antiphospholipid antibodies based on: 1) abnormal serum concentration of IgG or IgM anticardiolipin antibodies; 2) positive test result for lupus anticoagulant using a standard method; or 3) false-positive serologic test for syphilis known to be positive for at least 6 months and confirmed by Treponema pallidum immobilization or fluorescent treponemal antibody absorption test
Malar rash	Fixed erythema, flat or raised, over the malar eminences, tending to spare the nasolabial folds
Neurologic disorder	Seizures in the absence of contributing medication or known metabolic derangements (eg, uremia, acidosis, or electrolyte imbalance) Psychosis in the absence of contributing medication or known metabolic derangements (eg, uremia, acidosis, or electrolyte imbalance)
Oral ulcers	Oral or nasopharyngeal ulceration, usually painless, observed by a physician
Photosensitivity	Skin rash as a result of unusual reaction to sunlight, by patient history or physician observation
Renal disorder	Persistent proteinuria, >0.5 g per day, >3+ if quantitation is not performed, or Cellular casts: may be red blood cell, hemoglobin, granular tubular, or mixed
Serositis	Pleuritis: convincing history of pleuritic pain or rub heard by physician or evidence of pleural effusion, or Pericarditis documented by ECG or rub or evidence of pericardial effusion

ACR, American College of Rheumatology; ANA, antinuclear antibody; ECG, electrocardiography; Ig, Immunoglobulin; SLE, systemic lupus erythematosus.
Adapted from reference 29.

anticardiolipin antibody and lupus anticoagulant target phospholipids, inducing vascular thrombosis[1].

Numerous genetic factors affect pathogenesis of SLE. Monozygotic twins appear to have an increased prevalence of SLE[35]. First-degree relatives have a reported 29-fold relative risk[36]. A predisposition to develop SLE is thought to involve expression of multiple genes and gene regions, including autoantibody production, several human leukocyte antigens, and non-leukocyte antigens[1].

The clinical picture of each patient differs according to unique and multiple stimuli, and the pathogenesis of the disease may be influenced by a number of environmental factors[1]. These include exposure to viruses (most notably Epstein-Barr), ultraviolet light, certain medications (including procainamide, hydralazine, isoniazid, hydantoins, chlorpromazine, methyldopa, penicillamine, minocycline, tumor necrosis factor blockers, and interferon-α), and certain dietary components (Table 1).

Diagnosis

The ACR has established clinical criteria for the diagnosis of SLE (Table 2). A patient must exhibit at least 4 of the following 11 features: serositis, manifested as pleuritis or pericarditis; oral ulcers, including nasopharyngeal lesions; arthritis; photosensitivity; hematologic abnormalities, including hemolytic anemia or any blood component deficiency; renal pathology, such as proteinuria or cellular casts;

presence of immunologic disorders such as anti-Smith, anti-double-stranded DNA (anti-dsDNA), anti-histone, anti-U1RNP, anti-Ro/SSA, or anti-La/SSB; positive antinuclear antibodies; neurologic disorders; malar rash; and discoid rash. These standard criteria confer 95% specificity and 85% sensitivity for SLE diagnosis[37]. Positive ANA is the most sensitive and optimal test for SLE screening. However, ANA is commonly seen in other autoimmune disorders, while anti-dsDNA and anti-Smith antibodies are more specific to SLE[38].

Diagnosing SLE may be a tedious process; however, many laboratory studies, imaging studies, and histologic tests are available. A Coombs' test measures erythrocyte-specific antibodies in patients with anemia. Anti-histone screening may confirm drug-induced lupus in a patient whose prescription history is pertinent[1]. Other tests are used to determine levels of certain biological markers to support the diagnosis. For example, an increased level of creatine kinase supports myositis; an elevated C-reactive protein level or erythrocyte sedimentation rate indicates an inflammatory state; and depressed levels of the complement proteins C3 and C4 suggest immune complex activity.

Lupus band test (LBT) is a useful diagnostic test that detects the deposition of immunoglobulins and complement in the dermal-epidermal junction by direct immunofluorescence. Specificity of LBT is very high, making it useful in the differentiation of SLE from other skin lesions, as well as, from other ANA positive diseases. A positive LBT in a patient without dermal involvement can aid in making the early diagnosis of SLE and can predict a decreased prognosis if involvement is seen in areas not exposed to UV-light[39].

The use of various molecular biology techniques to test for antibodies coupled with the clinical picture may distinguish SLE from other connective tissue disorders or determine coexisting disease. For example, the presence of anti-Ro/SSA or anti-La/SSB indicates Sjögren's syndrome and is associated with neonatal lupus. Anti-RNP antibodies suggest scleroderma, whereas anti-cardiolipin antibodies have been described in the pathogenesis of antiphospholipid antibody syndrome1 (Table 3).

Table 3
Clinical Manifestations and Associated Autoantibodies in SLE

Manifestation	Autoantibody
Nephritis	Anti-dsDNA Anti-Ro Anti-C1q
Dermatitis	Anti-Ro Anti-dsDNA
Vasculitis	Anti-Ro
CNS	Anti-ribosomal P Antineuronal Anti-NR2
Hematologic Lymphopenia Hemolysis Thrombocytopenia Clotting	 Antilymphocyte Antierythrocyte Antiplatelet Antiphospholipid
Fetal loss	Antiphospholipid
Sjögren's syndrome	Anti-Ro
Neonatal lupus	Anti-Ro

CNS, central nervous system; dsDNA, double-stranded DNA; SLE, systemic lupus erythematosus
Adapted from reference 1.

Prognosis and Treatment

Prior to advancements in screening tests, diagnostic laboratory studies, and treatment options, the prognosis was dismal for patients with SLE. Currently, the survival rate exceeds 90% in patients diagnosed 10 years previously[40]. Although the pathogenesis of SLE is different for each patient, there is an established correlation of increased mortality with infection, accelerated atherosclerosis, CNS involvement, and renal disease. For the younger patient, infection seems to be a main cause of death, whereas complications related to atherosclerosis decrease survival in older patients[4]. Etiologic factors that increase mortality include age greater than 50 years, male gender, and low socioeconomic status[41].

Current therapeutic options for SLE are treatments directed at systemic inflammation, immune-cell targets, signaling pathways, and cytokines. Antimalarial drugs, corticosteroids, cyclophosphamide, MMF, Methotrexate, and Aziothioprine are effective in suppressing inflammation. Agents that target B lymphocytes to prevent production of autoantibodies include rituximab (anti-CD20), epratuzumab (anti-CD22), belimumab (anti–B-cell lymphocyte-activating factor [BAFF]), and atacicept (anti-BAFF and a

proliferation-inducing ligand [APRIL]). Anti-cytokines, such as anti-TNF, anti-interleukins, and anti-INFα, interfere with immune cell signaling and activity[42].

The treatment and management of patients with SLE varies. Disease severity and organ involvement determine a suitable treatment regimen. Treatment is induced during relapses in an effort to prevent exacerbations. Patients with mild SLE, defined by musculoskeletal and cutaneous involvement, generally are treated with antimalarials, glucocorticoids, and nonsteroidal anti-inflammatory agents. Patients in whom there is major organ involvement, including renal, hematologic, pulmonary, cardiac, and nervous systems, are considered to have moderate to severe SLE. These patients benefit from more intense treatment with immunosuppressive, cytotoxic, and biologic agents.4 Clinical guidelines set forth by the ACR recommend drug therapy with azathioprine, mycophenolate mofetil, cyclophosphamide, methotrexate, and cyclosporine, with appropriate monitoring for toxicity[4]. In addition, a new drug belimumab, a monoclonal antibody that inhibits B lymphocyte differentiation and autoreactivity, shows promising results in patients with active disease[43].

Anesthetic Considerations

Preoperative Assessment

Because of extensive, multiple organ dysfunction that can develop in SLE, the preoperative assessment of a patient with this disease may be extensive. Patient history, thorough physical examination, laboratory testing, and imaging are indicated. Cardiovascular function should be assessed with chest radiography, echocardiography, and electrocardiography to determine the presence of pericarditis, endocarditis, myocarditis, congestive heart failure, and conduction blocks.

In addition to cardiovascular evaluation, pulmonary function and arterial blood gas tests should be conducted if respiratory symptoms are present. Other complications such as lupoid hepatitis can be uncovered by liver function tests, a gastrointestinal series, and determination of albumin/globulin ratios and bilirubin levels. Anemia, thrombocytopenia, and leukopenia can be assessed by hematologic studies, including complete blood count, platelet count, prothrombin time, and partial thromboplastin time.

For CNS involvement, electroencephalography and a computed tomography scan may be necessary. Renal involvement can be evaluated by urinalysis, renal ultrasound and scan, blood urea nitrogen level, and creatinine, albumin, and total serum protein levels[44]. Identifying specific organ dysfunctions and the clinical picture will determine the appropriate anesthetic plan (Table 4).

Intraoperative Assessment

There are many perioperative issues to consider in the patient with SLE-from organ pathology to anatomic change. As mentioned, multiple manifestations of the disease may alter anesthetic management of the patient. Renal or hepatic involvement may affect the metabolism and efficacy of common drugs, including IV and inhaled anesthetics, analgesics, neuromuscular inhibitors, cholinesterase inhibitors, and muscarinic antagonists. Patients treated with cyclophosphamide may require a longer period of anesthesia induction because of an inhibitory effect on cholinesterase that may lengthen the response to succinylcholine[45]. Intubation, extubation, and maintaining an airway may be difficult in some patients because of SLE-induced upper airway obstruction and laryngeal involvement[10].

Airway Maintenance

Airway protection is a major concern in all patients undergoing anesthesia. Patients with SLE may have mucosal ulceration, cricoarytenoid arthritis, laryngeal pathology including recurrent laryngeal nerve palsy, or temporomandibular joint dysfunction that results in a difficult intubation[10]. Avoiding intubation when possible or using fiber-optic techniques are alternative approaches[44].

Pulmonary

In patients with SLE, respiratory involvement may include acute pneumonitis, chronic alveolar infiltrates, and recurrent infectious pneumonia[4]. Perioperatively, pulmonary function and oxygenation should be carefully assessed. Avoidance of hypoxia, hypercapnia, and catecholamine release maintains pulmonary blood flow and reduces pulmonary vascular resistance. Arterial cannulation for blood gas analyses, and placement of a pulmonary artery catheter to assess

Table 4
Preoperative Assessment of the Patient With SLE

System	Effects	Assessment by History	Physical Examination	Tests
Cardiovascular	Pericarditis Endocarditis Myocarditis CHF Conduction blocks	Chest pain Palpitations	Murmur Effusion Diastolic noncompliance Pericardial friction rub	ECG CXR Echocardiography
Respiratory	Infiltrates Restrictive PFTs ↑ A-a gradient Atelectasis	Pleuritic pain Dyspnea Cough Hemoptysis	Friction rub Effusion Cyanosis Normal peak flow	CXR PFTs ABGs
Gastrointestinal	Perforated viscus Pseudo-obstruction Liver congestion Lupoid hepatitis	Nausea/vomiting Peritonitis Pancreatitis Abdominal pain Ileus	Dilated loops of bowel Peritoneal free air Hepatomegaly Jaundice	Gastrointestinal series LFTs Bilirubin level A/G ratio
Hematologic	Hemorrhage Thromboembolism Anemia	Bruising Thrombosis	Lymphadenopathy Splenomegaly Anemia	CBC Platelet count PT, PTT
Renal	Glomerulitis Nephrotic syndrome Renal insufficiency Renal failure	Polyuria Oliguria Hematuria Fever	Costophrenic tenderness Edema	Urinalysis Renal US Renal scan BUN, Cr, TP, albumin
CNS	Confusion Hallucinations Psychoses Seizures	Paranoid states Hyperirritability Numbness Hemiparesis	Psychosis Nystagmus, ptosis, diplopia Aphasia Peripheral neuropathy	EEG CT scan Neurologic, psychiatric evaluations
Musculoskeletal and dermatologic	Vasculitis Symmetric arthritis Joint immobility Aseptic necrosis	Photosensitivity Atrophic/scarred lesions Ecchymosis Purpura Joint pain Immobility	Malar or butterfly rash Perioral ulcerations Reduced range of motion Hip pain	Hip x-rays Antinuclear antibody

A-a, alveolar-arterial; ABG, arterial blood gas; A/G, albumin/globulin; BUN, blood urea nitrogen; CBC, complete blood count; CHF, congestive heart failure; CNS, central nervous system; Cr, creatinine; CT, computed tomography; CXR, chest x-ray; ECG, electrocardiography; EEG, electroencephalography; LFT, liver function test; PFT, pulmonary function test; PT, prothrombin time; PTT, partial thromboplastin time; SLE, systemic lupus erythematosus; TP, total protein; US, ultrasound
Adapted from Robinson DM. Systemic lupus erythematosus. In: Roizen MF, Fleisher LA, eds. Essence of Anesthesia Practice. 2nd ed. Philadelphia, PA: WB Saunders; 2002.

pulmonary hypertension, may be indicated[44]. A rare complication in these patients is alveolar hemorrhage, in which case pulmonary capillary exchange, oxygenation, and airway pressure must be monitored; suction should be readily available.

Renal

Glomerulitis, nephrotic syndrome, renal insufficiency, and renal failure may develop. Renal involvement poses a significant challenge in patients with SLE and may alter standard administration of anesthetics[44]. Drugs requiring renal excretion, including some opioids, benzodiazepines, and neuromuscular blocking agents, may accumulate. The lingering, toxic metabolites lead to prolonged sedation, paralysis, and an increased recovery period. Additionally, the kidneys

or other organ systems may be further damaged. In cases of extreme end-organ damage, the use of remifentanil and cisatracurium-both metabolized via processes that are end organ-independent-is indicated.

Cardiovascular

Premature and accelerated atherosclerosis increases the risk for cardiovascular disease[46]. Patients are predisposed to potentially catastrophic events such as intraoperative myocardial infarction. Every effort should be made to maintain hemodynamic stability.

Management of the Case Presented

A detailed medical history of the patient was obtained, and a physical examination completed. Her airway was characterized as Mallampati class II with good cervical range of motion. Her lungs were clear to auscultation; heart sounds were regular without murmurs. Other findings of the physical examination were within normal limits, except for alopecia, which was moderate.

After a discussion with the patient about the risks and benefits of general anesthesia, regional anesthesia, and supplemented local anesthesia, the latter was chosen. After IV administration of 2 mg of midazolam and 50 mcg of fentanyl, the surgeon locally injected a mixture of bupivacaine and lidocaine. A propofol infusion of 40 mcg/kg per minute was started. The patient also received 4 mg of ondansetron. The procedure lasted 45 minutes. The patient was discharged to undergo dialysis, and later to home.

Conclusion

SLE is a complicated autoimmune disease with variable systemic manifestations. Because of the complexity and potentially wide-ranging clinical presentations of SLE, anesthetic management of patients is challenging. The inherent heterogeneity of SLE necessitates extensive preoperative assessments of patients, in addition to obtaining detailed histories and physical examinations. Careful anesthetic planning and intraoperative monitoring of all affected organ systems-particularly renal, pulmonary, and cardiovascular function-are required.

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