Continuing Medical Education

Systemic Lupus Erythematosus and the obstetrical patient - implications for the anaesthetist

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Systemic lupus erythematosus (SLE) is a multisystem, chronic inflammatory disease characterized by autoantibody production. The disease is most frequently found in women of childbearing age and therefore may co-exist with pregnancy. The clinical manifestations of the disease are variable and depend on the severity of damage to organ systems such as musculoskeletal, renal, haematological, neurological, cardiac, and respiratory. Many patients require drugs such as aspirin or prednisone. The pregnant patient may experience exacerbations of the disease, neonatal loss, and obstetrical complications such as pre-eclampsia. Patients with the Lupus Anticoagulant are at risk for an abnormal perinatal course. The anaesthetic management will depend on the patient's clinical status and the well-being of the fetus. The patient should be examined to determine the extent of end organ damage, current medications, and the health of the fetus. Laboratory investigations such as a coagulation screen and tests of renal function should be performed before anaesthetic intervention if time permits. A multidisciplinary approach to care of the patient and resources to manage complications are essential to optimize the outcome for both mother and newborn.

Key words:

ANAESTHESIA: obstetrical;

COMPLICATIONS: SLE.

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Le lupus érythémateux est une maladie inflammatoire chronique multisystémique avec formation d'auto-anticorps. Il survient surtout chez les femmes en âge de procréer et on peut donc le retrouver lors d'une grossesse. Les manifestations de la maladie varient en fonction de l'atteinte des différents systèmes: musculo-squelettique, rénal, hématologique, neurologique, cardiaque et respiratoire. Plusieurs patientes prennent des médicaments tels l'aspirine ou la prednisone. Pendant la grossesse, la maladie peut s'aggraver, entraînant la mort fœtale ou d'autres complications telle la pré-éclampsie. De plus, les patientes affligées d'un anticoagulant lupique peuvent saigner en période périnatale. La démarche anesthésique dépendra de l'état clinique de la patiente et du fœtus que l'on devra évaluer avec une attention particulière portée aux organes cibles et à la médication. Avant une anesthésie on devra si possible mesurer l'intégrité des mécanismes de la coagulation et de la fonction rénale. Le sort de la mère et du nouveau-né dépendra en partie de la composition et de la capacité d'intervention de l'équipe soignante.

Systemic lupus erythematosus (SLE) is a multisystem inflammatory disorder characterized by autoantibody production. Antibodies are produced against host antigens and an inflammatory response is induced which results in tissue injury and the subsequent clinical symptoms. It has a strong female preponderance (10:1) and occurs primarily in the reproductive years.¹ The course and prognosis of the disease differ markedly among patients. However, over the past decade there has generally been a trend toward more favourable outcomes. Consequently, the obstetrical anaesthetist is likely to encounter pregnant lupus patients with increasing frequency. The purpose of this article is to provide a working knowledge of this disease and its implications for the practicing anaesthetist.

The immune system

The cardinal feature of this disease is immune mediated tissue injury. The major components of the immune system include cellular elements (lymphocytes, plasma cells and macrophages), humoral products of cells (e.g., immunoglobulins), and complement. Normally these three components interact to rid the body of foreign material and pathogenic organisms. However, detrimental effects can occur if in the process they cause inflammation and tissue damage or if the immune response is directed against autoantigens as in autoimmune disorders.²

Immune system injury can occur by four mechanisms: anaphylaxis (type I), cytotoxic (type II), circulating immune complexes (type III) and delayed hypersensitivity (type IV). SLE represents type III or immune complex disease. Antibodies combine with host antigens and are subsequently deposited in various tissues such as vascular beds, basement membranes and serous cavities. This causes complement activation and release of vasoactive and chemotactic factors. As a result phagocytosis of the complexes by polymorphs occurs followed by enzyme release and cell damage. Ongoing cell destruction is mediated by monocytes and T lymphocytes.²

The serum of patients with SLE contains an array of antibodies.³ A number of these bind diverse nucleic acid antigens such as DNA, RNA, various nucleoproteins and histones. One of these, known as anti RO/SSA, is of interest in that it is responsible for the syndrome of neonatal lupus. RO/SSA is an RNA protein complex whose function in the cell is presently unknown. Antibodies against it are IgG antibodies and are therefore able to cross the placenta and cause disease in the fetus. Forty per cent of SLE patients and 0.1% of normal subjects are positive for anti RO/SSA.

Another group of antibodies, the antilipid antibodies, react against various phospholipids and are responsible for the lupus anticoagulant activity, anticardiolipin antibodies and the false positive test for syphilis.

The lupus anticoagulant (LA) is of particular interest to the anaesthetist because of its effects on the coagulation screen. This is an antibody of the IgG or, occasionally, IgM class and it is estimated to be present in 10–34% of SLE patients.⁵ Its presence interferes with phospholipid dependent coagulation tests and results in a prolongation of the PTT and very rarely the PT, neither of which is corrected by the addition of normal plasma.⁶ This is an *in vitro* phenomenon and persons with LA do not have a bleeding tendency. Paradoxically patients are at increased risk of developing recurrent arterial or venous thromboembolic events.^{1,5–8} In addition, women who have circulating LA are at increased risk for recurrent abortion and intrauterine fetal death.^{4,9–11} This is possibly due to

decidual vasculopathy and placental infarction.¹² Although the isolated presence of LA does not present a risk of bleeding, other haematological abnormalities have been associated with LA and predispose the patient to haemorrhage.¹³ These include antibodies to coagulation proteins, particularly factor VIII and prothrombin. Also, both thrombocytopaenia and a qualitative platelet abnormality have been reported.

Diagnosis

The diagnosis of SLE requires evidence of disease in more than one organ system together with serological evidence of a disturbed immune system; that is, antibodies to one or more components of the cell nucleus.¹⁴ In particular, antibodies to double-stranded, versus single-stranded, DNA occur almost exclusively in SLE with levels frequently correlating with disease activity.

Clinical manifestations

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. Common sites of involvement include:

Musculoskeletal

Arthritis and arthralgia are the most frequent presenting complaints. Both small and large joints are affected but it is rare to have erosive changes as seen in rheumatoid arthritis.¹⁴

CUTANEOUS

The characteristic rash, "butterfly rash," is an erythematous rash over the face and bridge of the nose. It occurs in about 40% of patients. Raynaud's disease is also common and occurs in approximately one-fifth of patients. Other cutaneous manifestations include nasal and oral mucosal lesions, ulcers, and angioneurotic oedema.¹⁴

RENAL

Renal involvement is a major cause of morbidity and mortality and is most commonly marked by manifestations of glomerulonephritis (GN) such as proteinuria, hypertension, nephrotic syndrome, an active urinary sediment, azotaemia, and renal insufficiency.¹⁴ Several patterns of GN have been described with the course of the renal disease generally being better with the lower grades, such as focal or segmental, and worse with more diffuse proliferative involvement. Although GN is the most common lesion, vascular and tubulointerstitial disease have also been reported.

HAEMATOLOGICAL

Mild anaemia is a frequent occurrence and is usually

secondary to the decreased red cell production that accompanies inflammatory processes. Occasionally a more severe Coomb's positive haemolytic anaemia may occur.¹⁴ Leukopaenia, lymphopaenia, and thrombocytopaenia are common with active disease.¹⁴ In contrast clotting defects due to antibodies to factors II, VII, VIII, IX, and X have been reported but are infrequent.^{6,15} The presence of LA, resulting in a prolongation of the PTT, has already been discussed.

NEUROLOGICAL

More than 50% of lupus patients have some form of neuropsychiatric abnormality. The manifestations are diverse, are not specific to SLE and present a challenge in diagnosis and management. Seizures, strokes, peripheral neuropathies, psychosis, organic brain syndrome, and movement disorders are some examples. Despite early diagnosis and aggressive therapy these abnormalities remain a frequent cause of death.¹⁴

CARDIOPULMONARY

Pleuritis and pericarditis are the most common cardiopulmonary manifestations of lupus. However, myocarditis, bacterial endocarditis (Libman-Sachs), as well as coronary artery disease can occur.^{14,16-18} Although coronary vasculitis may cause obstructive lesions, accelerated atherosclerosis is the most common cause of coronary artery disease in SLE patients. Steroid therapy, hypercholesterolaemia, hypertension and immunological factors have been postulated to be contributing factors.^{16,17}

Pulmonary infections are the most common cause of lung infiltrates in patients with SLE. However, a small number of patients may develop diffuse alveolar infiltrates secondary to acute lupus pneumonitis while others may suffer from chronic interstitial fibrosis. Other manifestations include pulmonary hypertension, diaphragmatic dysfunction and pulmonary embolism.¹⁶

In addition to major organ involvement, constitutional symptoms such as fever, weight loss, malaise, and fatigue may mark the inflammatory state.¹⁴

Treatment

Treatment of SLE is aimed at achieving and maintaining adequate disease suppression while limiting unacceptable drug side effects. In light of its multifaceted nature many agents and treatment strategies have been utilized. Therapy can be divided into two approaches.¹⁴ The first is directed towards control of the disease manifestations and utilizes conventional agents such as antihypertensive and anticonvulsant drugs. The second is aimed at the hyperactive immune function and tissue inflammation. Here anti-inflammatory and immunosuppressive agents are the mainstay of therapy. Generally nonsteroidal anti-inflam-

matory drugs including ASA, are useful for the treatment of systemic symptoms, arthritis and mild serositis. Similarly, antimalarials are utilized for these complaints as well as for the skin manifestations of SLE. Steroids are employed for more serious and disabling manifestations while immunosuppressive and cytotoxic agents are reserved for major organ involvement that is progressive and unresponsive to glucocorticoids. On occasion, plasmaphoresis, in conjunction with immunosuppressive agents, may be used for severe life-threatening lupus or active major organ disease.

SLE and pregnancy

As lupus is predominately a disease of young women, clinical considerations include the possibility of pregnancy in this population. Generally, normal fertility rates have been reported.^{1,12,19} However, pregnancy is not without complications. Hazards include disease exacerbation, fetal death, neonatal lupus and obstetrical complications.²⁰

Disease exacerbation

The effects of pregnancy on SLE have been the subject of controversy. Studies have been fraught with numerous difficulties relating to their retrospective nature, control population and lack of known exacerbation rates in non-pregnant patients.^{10,21} Even prospective studies seem to be inconsistent, perhaps because there is no consensus for the diagnosis of lupus "flare."²⁰ However, exacerbations of maternal disease are said to occur in 30-50% of pregnancies.^{19,21,22} This frequency is two to three times greater if lupus is active at the time of conception with a higher proportion of the exacerbations occurring during the second and third trimesters and the post partum period.⁷ Pregnancy does not alter the long-term course of the disease and consequently pregnancy is not contraindicated.

It is accepted that active disease during pregnancy does not require abortion but should be treated with the necessary dosage of steroids required for disease control. Maternal deaths have only been reported in patients who were not treated with steroids and subsequently developed lupus crisis unresponsive to therapy.²¹ Mintz has recommended a minimum dose of prednisone for all pregnant lupus patients.¹⁹ In addition, some studies have suggested a several-fold increase of flares during the puerperium and prophylactic steroid therapy has been advised. However, *prophylactic* steroids during pregnancy or the puerperium are both controversial. Consequently their use varies considerably and is by no means universal.

Fetal death

Studies on fetal loss have found the fetus to be at high risk.

Spontaneous abortion is twice as frequent as in the general population and is not influenced by active or inactive maternal disease.¹⁹ Prematurity is a constant hazard occurring in 35–50% of pregnancies. It is associated with intrauterine malnutrition and is related to active maternal disease.²¹ Therefore, constant efforts to maintain the disease in an inactive state appear to be essential. Fetal death occurs in about one-third of lupus pregnancies. Patients with antibodies to anticardiolipin and LA are particularly at risk.^{10,11} Both anticardiolipin and an abnormal PTT can be identified early in pregnancy and are frequently associated with thrombocytopaenia. With high-dose steroid therapy the PTT frequently returns to normal while low platelet counts may be reversed with ASA.^{20,22} However, despite treatment, this subgroup of patients continues to have a fetal mortality rate greater than 50%. In patients with these antibodies and a poor obstetrical history, prophylactic treatment is undergoing international study.²³ In the interim, close monitoring of maternal disease activity and regular assessments of intrauterine growth are recommended. Deteriorating fetal well-being necessitates early delivery since premature infants of SLE patients usually die *in utero* rather than post partum.^{10,21,24}

Neonatal lupus

Neonatal lupus is a relatively infrequent clinical syndrome found exclusively in infants of mothers with anti Ro antibody.^{7,12,22} Transient clinical manifestations include dermatitis, anaemia, thrombocytopaenia, pneumonitis, hepatosplenomegaly and rarely glomerulonephritis. More permanent manifestations involve the cardiac tissue. Congenital heart block (CHB) is the most common manifestation.^{1,25} This usually presents as a stable bradycardia noted on routine obstetrical examination. Occasionally pericarditis and other structural cardiac abnormalities such as extensive endocardial fibrosis have been reported.^{12,26}

Obstetrical complications

Pregnancy-induced hypertension (PIH)

Patients with SLE are at an increased risk of developing PIH, irrespective of their pre-pregnant renal status.² This risk is increased if patients require more than 30 mg of prednisone daily. However, the distinction between PIH and lupus nephritis may be difficult. The presence of extrarenal disease, increasing anti-DNA titres, renal casts and low complement levels support lupus as the primary problem.^{7,12,27}

Thrombocytopaenia

A few patients may develop low platelets in the first

trimester of pregnancy, particularly those with a history of thrombocytopaenia or LA. This thrombocytopaenia is resistant to steroid therapy but may be reversed with ASA.²² It is associated with mid-trimester fetal death and spontaneously reverts to normal post partum. The development and new thrombocytopaenia during the latter weeks of pregnancy indicates PIH and should be managed as such.¹⁰

Prosthetic Joints

Ten per cent of lupus patients suffer from debilitating osteonecrosis most frequently involving the femoral head. The pathogenesis is unclear but its development is correlated with high-dose steroid therapy. There is no effective therapy and patients may require total joint replacement. With the softening of ligaments during late pregnancy these prosthetic joints may be endangered. Loosening and fracture have been reported.²⁰ In addition, infection has occurred and the routine use of antibiotics for these patients has been recommended.⁷

Management

Care of the pregnant lupus patient requires cooperation between the rheumatologist, obstetrician, neonatologist and anaesthetist. Although the ideal treatment strategy has yet to be defined, certain management principles are evident.

Since fetal survival is improved if pregnancy occurs during inactive disease patients should be advised to plan pregnancy, if possible, during a period when the disease has been quiescent for approximately six months.^{7,19}

Baseline CBC, BUN, Cr, anti-DNA titres, complement levels (C3, C4), urine sediment, 24 hr CCr and total urine proteins should be done before and after conception. These should be repeated monthly until two to three months post partum and more often if necessary.⁷ The PT, PPT, anti RO/SSA, and anticardiolipin antibody should also be measured early as these will help to identify those pregnancies which are at increased risk. Furthermore, if an elevated PTT is identified the cause must be determined. As the various assays and tests^(5,28) required to make this determination take time, this must be done well in advance of delivery since coagulation defects secondary to factor abnormalities or antibodies contraindicate regional anaesthesia while the presence of lupus anticoagulant does not.

Once pregnancy has been confirmed, low-dose steroid therapy may be considered^(7,21). Symptoms and laboratory manifestations of active disease must be sought throughout the pregnancy and in the first two months post partum. If an exacerbation occurs the patient should be treated with prednisone in doses necessary to control symptoms.

In addition to careful evaluation for signs of PIH, the obstetrical protocol must include measures to decrease intrauterine fetal mortality. Specifically, the growth and development of the fetus must be monitored.^{7,10} Once the fetus is viable, monitoring for fetal distress is required.²⁴ This is particularly important if the mother has a history of LA or anticardiolipin. Lockshin²² and Druzin²⁴ have noted a uniform pattern which is consistent with troubled pregnancies. At first there is an initial period of apparently normal progression. This is followed by a decrease in fetal growth, loss of normal fetal cardiac reactivity, a decrease in amniotic fluid volume and finally a spontaneous fetal bradycardia. At this point either the infant is delivered or dies.¹⁰ Consequently if fetal distress is diagnosed, interruption of the pregnancy is strongly advised. Consultation with a neonatologist regarding the potential for the survival of the fetus would be advisable at this time. If delivery is felt to be appropriate, induction of fetal lung maturity with betamethasone may be considered.

In patients who are positive for antiRO, evaluation of the fetus for evidence of bradycardia is essential. Fetal echocardiography is useful after the 28th week to identify an associated myocarditis. If CHB is suspected the newborn needs to be assessed regarding the need for a pacemaker and monitored for signs of congestive heart failure in the early neonatal period.^{7,12} A neonatal intensive care unit should be available at the time of delivery.

Anaesthetic management of the lupus patient includes all types of anaesthesia but must take into account the multisystem nature of the disease, the severity of the organ involvement and the drugs used in treatment. As well, the usual anaesthetic considerations for the pregnant patient apply.

A careful, systematic approach to the patient will identify those organs damaged by the disease and allow one to plan the anaesthetic appropriately. Similarly, preanaesthetic investigation and intraoperative monitoring must be tailored according to the disease manifestations in the patient. For example, if the patient has a history of coronary artery disease, management is guided by the same principles that apply to any patient with ischaemic heart disease. Likewise, the general management guidelines for patients with PIH are applicable. In addition there are a number of specific concerns that warrant mention. Ulcers on nasal and oral mucous membranes may increase the possibility of trauma with tracheal intubation while Raynaud's phenomena complicates the placement of arterial lines. The presence of peripheral neuropathies or fixed neurological deficits must be appreciated and documented when considering regional anaesthesia. A coagulation screen including PT, PTT, platelets and bleeding time should be done on all

patients. Elevation of PT or PTT may be due to antibodies to the various coagulation factors, most commonly factor VIII. These specific anticoagulants are diagnosed by performing factor assays. As there is a considerable risk of bleeding in their presence, regional anaesthesia is contraindicated. In contrast, an isolated elevation of the PTT secondary to LA is not associated with a bleeding tendency and therefore does not contraindicate epidural anaesthesia. Its presence is confirmed by demonstrating non factor specificity and documenting phospholipid dependency. However, the laboratory heterogeneity of LA is well documented in the literature.¹³ The correct diagnosis is often time-consuming and frequently requires more than one confirmatory test. Obviously the distinction between LA and factor abnormalities needs to be determined well in advance of labour and delivery. This *must* be done before considering regional anaesthesia since in both instances patients may be asymptomatic. The platelet count and bleeding time are necessary because both thrombocytopenia and/or thromboasthenia have been reported. Therapy during pregnancy with ASA may further confound the picture. Generally in these circumstances a normal bleeding time is reassuring. However, there have been two reports of haematomas following conduction anaesthesia in patients on aspirin therapy who had normal bleeding times.^{29,30} Therefore the benefits of epidural analgesia and anaesthesia in patients on ASA must be weighed against the improbable, but possible, risk of epidural bleeding.⁸ The benefits may well outweigh the risk but one must assess each case individually.

Although patients with valvular lesions, specifically Libman-Sach endocarditis, are frequently asymptomatic, antibiotic prophylaxis for labour and delivery is indicated. As previously mentioned, prophylaxis is also recommended for those with hip prostheses.⁷ In addition, careful positioning of these patients during vaginal delivery to avoid joint stress is advisable. High-dose steroid coverage during labour and delivery and the first two days post partum is necessary.⁷ Thereafter, patients should resume their antepartum dose for at least two months. Finally, blood should be cross-matched and typed in advance of delivery as irregular antibodies in the serum may cause matching difficulties.²

In summary, pregnancy in patients with SLE is considered to be high risk for both the infant and mother. Disease exacerbation, increased fetal loss, neonatal lupus and an increased incidence of PIH are the major challenges facing physicians. However, close monitoring of the mother and fetus, aggressive management of active disease and improved neonatal intensive care have decreased maternal and fetal morbidity and increased the survival of newborns.

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Multiple choice questions**TYPE I QUESTIONS: PICK ONE CORRECT ANSWER**

- 1 The major components of the immune system include all except
 - A plasma cells
 - B complement
 - C bradykinins
 - D macrophages
- 2 SLE represents an immune complex system injury of type
 - A I, anaphlaxis
 - B II cytotoxic injury
 - C III circulating immune complex disease
 - D IV delayed hypersensitivity
- 3 A Neonatal lupus is found exclusively in infants of mothers who are positive for anti RO/SSA.
 - B Asymptomatic patients with Libman Sach endocarditis do not require antibiotic prophylaxis for labour and delivery.
 - C When treating exacerbations of SLE during pregnancy, steroid therapy should ideally be limited to less than 30 mg of prednisone daily.
 - D Infants with congenital heart block do not require pacemakers.

TYPE II. QUESTIONS: CHOOSE ANSWER:

- A if only 1, 2, or 3 are correct
- B if only 1 and 3 are correct
- C if only 2 and 4 are correct
- D if only 4 is correct
- E if all are correct

- 1 1 SLE patient serum contains autoantibodies which bind DNA, RNA, and other nuclear substances.
 - 2 RO/SSA is an RNA protein complex which may stimulate the production of an IgG autoantibody.
 - 3 Anti RO/SSA is found in 40% of SLE patients and 0.1% of normal patients' serum.
 - 4 Anti RO/SSA does not affect the fetus.
- 2 1 LA is an IgG antibody
 - 2 LA is the cause of an isolated elevation of PTT in SLE patients.
 - 3 Patients with LA have an increased risk of spontaneous abortion and intrauterine fetal death.
 - 4 Epidurals are contraindicated in patients with LA who have an elevated PTT.

3 In SLE –

- 1 Coronary artery disease in young women most likely secondary to coronary vasculitis.
- 2 Serum levels of double stranded DNA increase with active disease.
- 3 A Coomb's positive haemolytic anaemia is the most common cause of anaemia.
- 4 Glomerulonephritis is a major cause of mortality.

4 1 Decreased fertility rates have been reported in SLE.

- 2 Thrombocytopaenia in the latter weeks of pregnancy is usually secondary to an exacerbation of SLE.
- 3 Patients with SLE who have *no* renal involvement have the same risk of PIH as the normal population.
- 4 Exacerbations of SLE during pregnancy are more likely to occur in the second and third trimesters.

5 Predisposing causes for bleeding complications in patients with LA include:

- 1 specific factor inhibitors
- 2 platelet dysfunction
- 3 thrombocytopaenia
- 4 concomitant drug therapy

ANSWERS

Type I Questions
1 C 2 C 3 A

Type II Questions
1 A 2 B 3 C 4 D 5 E