Case Report

Anesthetic management of urgent LSCS in patient of APLA syndrome with mobile right heart thrombus - a case report

Surinder Singh Sodhi DNB¹

¹Deptt. of Critical care, Max Super Specialty Hospital, Shalimar Bagh, New Delhi, India

Email: sodhi11499@gmail.com

How to cite: Sodhi SS. Anaesthetic management of urgent LSCS in patient of APLA syndrome with mobile right heart thrombus - a case report. Onco Critical Care 2024;104-107

The antiphospholipid syndrome (APS) is a complex autoimmune systemic disease which is characterized by the presence of antiphospholipid antibodies (aPL). The vascular involvement with APS not only results in thrombotic complications but may also involve multiple organ systems including heart. The cardiac involvement due to thrombosis results in immune-mediated injury. Cardiac manifestations include valvopathies, valve thickening through non-bacterial thrombotic endocarditis, regurgitation and severe valvular damage, and coronary artery disease (CAD). Other less common cardiac manifestations include myocardial dysfunction, pulmonary hypertension and intracardiac thrombus.¹

When associated with pregnancy, APS can often lead to morbidities due to its association with thrombotic complications (recurrent fetal loss and placental insufficiency). Cardiac involvement can make the anaesthesia management difficult.² Mobile right heart thrombus (MRHT) formation with APS is rare and life threatening complication. We present here a case of a pregnant lady who presented with ischemic placental insufficiency.

Case Report

A 31-year-old, 36 weeks pregnant female, a known case of antiphospholipid syndrome with ischemic placental insufficiency, was planned for lower section cesarean section. Patient's bad obstetric history that included a missed abortion, a preterm (29 weeks gestation) and LSCS following imminent eclampsia. Previous LSCS was associated with pulmonary edema in the post operative

period and loss of neonate on seventh postoperative day due to necrotizing enterocolitis.

Diagnosis of APS was made on the bases of detection of significant lupus anticoagulant and cardiolipin antibodies in blood. Beta 2 glycoprotein levels was normal. Echocardiography that was done at 26 weeks of gestation, detected focal thickening & calcification of leaflets resulting in moderate regurgitation. A mobile mass lesion of size 15 x 8mm attached to interatrial septum on right auricle side was noticed. There had been no regional wall motion abnormality and cardiac ejection fraction was normal. Therapeutic doses of Heparin 60mg, Ecosprin 150mg along with Predisolone 10mg/day and HCQs were started. Repeat echocardiography following anticoagulant therapy revealed complete resolution of mobile thrombus 1 month later and the patient was put on inj clexane 0.6 mg subcutaneously once daily.

Repeat echocardiography during preoperative evaluation revealed reformation of mobile mass lesion (23 x 7mm) on the same location. Lower limb venous Doppler study was done which did not suggest deep vein thrombosis in lower limbs.

A multidisciplinary team (Anesthesiologist, Cardiologist, Obstetrician, Vascular Surgeon, Physician and Patient) meeting was called to address the treatment options and risk assessment. Concerns regarding semi emergent nature of surgery, systemic anticoagulation, surgical retrieval of mobile thrombus, preoperative placement of IVC filter, temporary embolization of uterine arteries and intraoperative management of possible

symptomatic pulmonary embolism, General Anaesthesia/Subarachnoid block, treatment option for mobile right heart thrombus, consent for hysterectomy, blood & blood products availability, CTVS/Cardiology backup etc were discussed.

It was decided that after continuing the daily dose of clexane, the surgery would be taken up next day under general anaesthesia. Symptomatic thromboembolic event would be treated immediately with administration of thrombolytic agents. The resulting surgical site bleeding would be addressed by manual compression, ligation of uterine arteries, administration of blood and blood products and if needed, hysterectomy.

After obtaining written informed consent which included possibility of thromboembolism, hysterectomy and fetal loss, the patient was taken up for LSCS under general anesthesia. Patient received inj clexane 0.4 ml subcutaneously 12 hour before surgery. After attaching the standard monitors, wide bore peripheral venous line, invasive arterial and central venous line were established under local anesthesia. Rapid sequence induction with propofol and succenylcholine was done for tracheal intubation. Immediately after opening the peritoneum, bilateral uterine arteries were identified and untied sutures were placed around the arteries. The subsequent course of anaesthesia and surgery remained uneventful. The surgery lasted for 40 min and a healthy male baby of 2.34 kg weight was delivered and the patient was transferred to intensive care unit for observation. Clexane 0.6 ml s/c 12 hourly, tab ecosprin 150 mg twice daily were started. Echocardiography done on 2nd postoperative day showed the persistence of MRHT of 22 X 7 cm at the same location. The subsequent postoperative course in the hospital remained uneventful and the patient was discharged from the hospital on 3rd postoperative day and advised to follow in OPD.

Discussion

APS is a complex systemic disease which probably results from immune mediated injuries and manifests itself in various clinical forms. Primary APS and Secondary APS have been defined depending upon the absence or presence of underlying connective tissue disorder respectively. Obstetric APS can affect both mother and fetus.³⁻⁵ Typically, the pregnant women

present with varying history of early miscarriages, still births, intrauterine growth retardation (IUGR), premature births complicated with pre-eclempsia, eclempsia, placental insufficiency, non-reassuring fetal surveillance tests or abnormal Doppler flow velocimetry. Pregnant women with APS have an increased risk of thrombosis, thrombocytopenia, and HELLP syndrome (hemolytic anemia, elevated liver enzymes, and low platelet count).⁶

It has been suggested that the pro-inflammatory and procoagulant activity of aPL on vascular endothelial cells might be responsible for valvular heart lesions and atherosclerosis. Mitral valvular involvement is the most common cardiac manifestation of APS and includes valvular thickening and valve vegetations (also referred as Libman-Sacks endocarditis). ⁷⁻⁸Intra-cardiac thrombus occurs rarely but is potentially life- threatening. In a case series of APS patients, the overall prevalence of intra-cardiac thrombi was found to be 1.8%. ⁹

High incidence of venous thromboembolism (VTE) and pulmonary embolism (PE) associated with pregnancy puerperium (0.05–0.20% and 0.03% respectively) are amongst the most common causes of maternal death. In a European cohort of 247ob-APS (EUROAPS), early and severe PE together with HELLP syndrome appeared in more than 18% of these women.¹⁰ Therefore, ob-APS patients require close surveillance and tailored treatment before, during, and after the pregnancy. Since deep vein thrombosis (DVT) is left-sided in >85% of cases due to compression of iliac vein by gravid uterus, >2 cm calf circumference difference in left leg during the second or third trimester should raise a strong possibility of DVT.¹¹ Positive D-dimer test in pregnancy is not necessarily indicative of VTE as D-dimer levels increase physiologically with each trimester. Stepwise approach for diagnosis in suspected cases include compression ultrasonography and magnetic resonance venography. 12 Where PE is suspected and all other investigations are normal, low-dose CT should be undertaken.13

Anticoagulants play a major role in overall management of ob-APS. Vitamin K antagonists (VKA) cross placenta and can cause embryopathy or fetopathy. Unfractionated heparin (UFH) or low molecular weight heparin (LMWH) combined with low dose of aspirin (LDA) (75–100 mg/day) remain the standard of care for

prophylactic or therapeutic management of VTE despite the associated risks of bleeding and thrombocytopenia. 14-15 Ob-APS frequently present for emergency LSCS due to maternal and fetal risks and give limited time for optimization. Furthermore, the presence of MRHT in the perioperative settings of LSCS makes the management difficult.¹⁶ MRHT can embolize at any moment and have a dismal prognosis. However, the most appropriate therapeutic approach for MRHT management remains an unresolved issue. Heparin infusion is time consuming and may be unsuitable in with' formed unstable patients clot'. embolectomy is costly and is associated with mortality rates of 20-50%. Percutaneous procedures carry risks of radiation exposure, damage to the puncture site, perforation of cardiac structures, tamponade, and contrast reactions.

Thrombolytics are considered to be relatively contraindicated during pregnancy and peripartum. Further, their use during LSCS can possibly lead to uncontrolled uterine bleeding at the site of placental separation. However, when PE is associated with desaturation and hypotension, administration of thrombolytics can be a reasonable lifesaving option. Thrombolysis results in immediate MRHT lysis, rapidly reduces pulmonary artery pressures and at least partly treats the accompanying DVT. However, with these short lasting effects, the lysed clot-fragments can migrate and recurrence of embolism following partial dissolution of the venous thrombus can happen. Unfractionated heparin infusion after thrombolysis is desirable.¹⁷

The anesthetic management of Mobile right heart thrombus is complex and challenging. MDT should be established. All goals and outcomes should be discussed. Backup plans should be available. Possibility of Hysterectomy and subsequent complications should be discussed in detail with the attendant/Patient.

Conflict of interest: Not declared

Reference

 Alfirevic, Z., Roberts, D. and Martlew, V. How strong is the association between maternal thrombophilia and adverse pregnancy

- outcome? A systematic review. Eur. J. Obstet. Gynecol.Reprod. Biol2002; 101: 6–14.
- 2. Tektonidou MG, Andreoli L, Limper M, Amoura Z et al. EULAR recommendations for the management of antiphospholipid syndrome in adults. Annals of the rheumatic diseases. 2019;78:1296-304.
- 3. Rodziewicz M, D'Cruz DP. An update on the management of antiphospholipid syndrome. Therapeutic Advances in Musculoskeletal Disease. 2020;12:1759720X20910855.
- 4. Farquharson RG, Quenby S, Greaves M. Antiphospholipid syndrome in pregnancy: a randomized, controlled trial of treatment. Obstetrics & Gynecology. 2002;100:408-13.
- D ware BR, Silver RM, Blackwell JL, Reading JC, Scott JR. Outcome of treated pregnancies in women with antiphospholipid syndrome: an update of the Utah experience. Obstetrics & Gynecology. 1992;80:614-20.
- Branch D.W, Silver R.M, Pierangeli SS, Van Leeuwen, Harris E.N. Antiphospholipid antibodies in women with recurrent pregnancy loss, fertile controls, and antiphospholipid syndrome. Obstet. Gynecol1997; 89, 549–555.
- Carreras LO, Forastiero RR, Martinuzzo ME.Which are the best biological markers of the antiphospholipid syndrome? J. Autoimmun.2000; 15, 163–172.
- 8. R. Cervera, J.C. Piette, J. Font, et al., Antiphospholipid syndrome: clinical and immunologic manifestations and patterns of disease expression in a cohort of 1,000 patients, Arthritis Rheum.2002; 46; 1019–1027.
- J.S. Levine, D.W. Branch, J. Rauch, The antiphospholipid syndrome, N. Engl. J. Med.2002 346;752–763.
- 10. R.S. Rai, L. Regan, Antiphospholipid syndrome and pregnancy loss, Hosp. Med. 1998;59: 637–639.
- 11. C.B. Chighizola, M. Gerosa, L. Trespidi, et al., Update on the current recommendations and outcome in pregnant women with antiphospholipid syndrome, Expert Rev. Clin. Immunol.2014; 10:1505–1517
- 12. W.A. Wilson, A.E. Gharavi, T. Koike, et al., International consensus statement on preliminary classification criteria for defi nite antiphospholipid syndrome: report of an

- international workshop, Arthriti Rheum. 1999;42: 1309–1311.
- 13. Carson, SA, Branch, DW. Management of recurrent early pregnancy loss. Compendium of Selected Publications. American College of Obstetricians and Gynecologists, Washington, DC, USA, 2002:372–383.
- 14. Chamley, L.W., McKay, E.J. and Pattison, N.S. Inhibition of heparin/antithrombin III cofactor activity by anticardiolipin antibodies: a mechanism for thrombosis. Thromb. Res.1993; 71:103–111.
- 15. Spinnato JA, Clark AL, Pierangeli SS, Harris EN. Intravenous immunoglobulin therapy for the antiphospholipid syndrome in pregnancy. Am. J. Obstet. Gynecol.1995; 172: 690–694.
- 16. Wilson WA, Gharavi AE, Piette JC. International classification criteria for antiphospholipid syndrome: synopsis of a post-conference workshop held at the 9th International (Tours) APL Symposium. Lupus 2001; 10:457–460.
- 17. Yetman DL, Kutteh, WH. Antiphospholipid antibody panels and recurrent pregnancy loss: prevalence of anticardiolipin antibodies compared with other antiphospholipid antibodies. Fertil. Steril. 1996; 66:540–546.