CASE REPORT

Ruptured ectopic pregnancy with APLA syndrome – a case report

Raja Muhammad Bilal*, Azmat Riaz, MCPS, FCPS**,
Rao Ali Shan Khan, FCPS***

**Registrar; **Assistant Professor; ***Consultant Anesthesiologist*
Department of Anesthesiology, Intensive Care & Pain Management, Combined Military Hospital, Rawalpindi (Pakistan)

Correspondence: Major Raja Muhammad Bilal, Registrar Anesthesiology, Department of Anesthesiology, Intensive Care & Pain Management, Combined Military Hospital, Rawalpindi (Pakistan); E-mail: dr_rajabilal@yahoo.com

ABSTRACT

A case of anesthesia for emergency laparotomy due to a ruptured ectopic pregnancy in a patient with antiphospholipid antibody syndrome is presented. It is a pro-thrombotic disorder characterized by the presence of two auto antibodies, lupus anticoagulant and anticardiolipin antibody. The syndrome is characterized by platelet adhesion, expression of prothrombotic molecules, and local complement activation leading to arterial and venous thrombosis.

Key words: Antiphospholipid antibody syndrome; Autoimmune disorder; Thrombosis; Pregnancy, Ectopic

Citation: Bilal RM, Riaz A, Khan RAS. Ruptured ectopic pregnancy with APLA syndrome – a case report. Anaesth Pain & intensive Care 2014;18(4):461-463

INTRODUCTION

Antiphospholipid antibody syndrome is a pro-thrombotic, autoimmune disorder that results in both arterial and venous thrombosis. It is one of the most common causes of an acquired hypercoagulable state and changes the entire paradigm of anesthetic management if the patient presents with significant hemorrhage. A case with such paradoxical anesthetic implications is not only rare but challenging for any anesthetist. We present the perioperative course of a patient with antiphospholipid syndrome, who underwent an emergency laparotomy for a ruptured ectopic pregnancy.

CASE REPORT

A 32 years old multigravida, presented to the emergency department for sudden onset severe abdominal pain and drowsiness. She was in hypovolemic shock with a BP of 60/45 mmHg with generalized abdominal tenderness and distention. The patient was a diagnosed case of antiphospholipid antibody syndrome and was on tab. warfarin 5 mg once daily for the last 9 years. She sustained a CVA nine years ago and had residual weakness in her left upper and left lower limbs. She was overdue by 2 weeks. The patient had a bad obstetric history of $G_8T_2P_2A_5L_2$ with 5 miscarriages. Fluid resuscitation and urgent investigations were carried out. Urine pregnancy test was positive. Hb was 5.1 mg/dL, Hct 18%, platelets 116 x 10^9/L, PT 30/14, PTTK 50/34 and INR 2.45. Transabdominal ultrasound revealed a bulky uterus with free fluid in abdominal cavity. Patient was shifted for emergency laparotomy with a preliminary diagnosis of ruptured ectopic pregnancy.

On pre-op examination, her vital signs were HR 160/min, BP 73/43 mmHg, RR 32/min, temperature 97.8°F. She was drowsy but arousable to verbal command. Her airway assessment was unremarkable and she was assigned an ASA Physical Status 4(E). Blood products including 6 units of RCC, 12 units FFP and 6 units platelet were ordered. Volume resuscitation with warm crystalloids was continued till the arrival of blood products. FFPs were administered in a dose of 15 ml/kg to reverse the anticoagulant effects of warfarin. High risk informed consent was obtained and 4 IV lines, including a 16G neck line in

the left external jugular vein, were established. An arterial line was also established in the right radial artery. The patient was catheterized, warmed with a convective air warmer and Thromboembolism Deterrent Stockings (TEDS) were applied.

In view of hypovolemia and deranged coagulation profile; a general anesthetic technique was selected. Prophylactic antibiotic and tranexamic acid 1g stat were administered at the outset. The patient was premedicated with ranitidine, metoclopramide, dexamethasone, glycopyrrolate and fentanyl. Preoxygenation was performed for duration of six vital capacity breaths. RSI was carried out with ketamine and rocuronium. Anesthesia was maintained with isoflurane 0.8 MAC and intermittent boluses of 100 μg fentanyl (0.6 mg for total duration of the case). Atracurium was used for maintenance of neuromuscular blockade. A CVC was passed in the right internal jugular vein under USG guidance. Intraoperative monitoring included ECG, pulse oximetry, heart rate, $EtCO_2$, invasive blood pressure, central venous pressure, urine output, blood loss, Paw, V_{TE}, peripheral nerve stimulator at ulnar nerve, O_2 and end tidal volatile anesthetic analysis.

The intraoperative blood loss including evacuated clots was approximately 4100 ml. A total of 3400 ml of blood products were transfused including 5 x RCC, 10 x FFPs and 4 x Platelets, in addition to 1000 ml crystalloid solution. 10 ml of 10% calcium gluconate was given stat. Hypothermia was prevented using IV fluid warmer and active convective warming of the patient.

The emergence, extubation and recovery were uneventful. The vital signs after extubation were HR 87/min, BP 116/54 mmHg, RR 13/min, SpO2 98% on FiO2 0.35.

Postoperatively, patient was nursed in surgical ICU. Vigilance for bleeding and thromboembolic complications was maintained. Mechanical DVT prophylaxis employing TEDS and a pneumatic intermittent compression device was initiated immediately. Pharmacological prophylaxis was commenced 12 hours postoperatively with IV heparin 5000 IU BD, in consultation with medical specialist. Adequate analgesia was provided with inj. fentanyl infusion at 50 μg/hour to allow early mobilization. The patient remained stable and was shifted to HDU on the 1st postoperative day. UFH was replaced with LMWH (enoxaparin 40 mg SC BD). The patient remained stable and was discharged on the 3rd postoperative day on oral NSAIDs and tablet warfarin 5 mg OD with advice of follow up in OPD.

DISCUSSION

Antiphospholipid antibody syndrome (APLA, APS, Hughes' syndrome[1]) is a pro-thrombotic disorder characterized by the presence of two auto antibodies, lupus anticoagulant and anticardiolipin antibody. The pathophysiology involves antiphospholipid antibodies binding to β_2-glycoprotein I, which then bind to glycoprotein Iba on platelets, monocytes, and endothelial cells. These complexes cause platelet adhesion, expression of prothrombotic molecules, and local complement activation leading to arterial and venous thrombosis.[1-3]

The disorder was first recognized in the early 1980s.[2] It is a paradoxical disease state, characterized by in vitro prolongation of aPTT with a strong predilection for in vivo thrombosis. Primary APS presents with thrombotic phenomena in young patients in the absence of any other related disease while secondary APS is associated with SLE or Lupus like disease. It is a multisystem disorder characterized by recurrent systemic arterial and venous thrombosis, recurrent abortions, thrombocytopenia and neurological disorders. Cardiac valvular and skin involvement in the form of livedo reticularis may be seen.

24% of the thrombotic events occur during pregnancy and puerperium. Asherson[4] has described a rare syndrome that consists of precipitous multisystem organ thrombosis and failure, known as catastrophic antiphospholipid syndrome, which is triggered by pregnancy in 4% of cases.

Fetal survival and maternal thrombotic risk are improved when affected pregnant women are treated with low-dose aspirin and heparin which are discontinued at term and then again restarted after delivery for an indicated duration based on thrombotic risk stratification.

Perioperatively, APS leads to thrombosis which may be spontaneous or triggered by infection, surgical intervention, withdrawal of, or suboptimal anticoagulation, or introduction of OCPs. Paradoxically, long term anticoagulant therapy or associated thrombocytopenia or factor deficiencies predispose the patient to life threatening hemorrhage. Coexisting autoimmune disorders, secondary organ involvement, and thrombotic phenomena should be evaluated. The term lupus anticoagulant is a misnomer and in the absence of an underlying coagulation deficit or anticoagulant therapy, the prolonged aPTT does not suggest

a bleeding tendency, and neuraxial anesthesia may be administered safely. Parturients with antiphospholipid syndrome who undergo general anesthesia are at risk for venous thrombosis. Our patient presented with massive hemorrhage and deranged coagulation thus regional anesthesia was ruled out and general anesthesia was administered. Postoperatively, optimal analgesia for early mobilization should be ensured to prevent the associated risk of thrombosis.[5] Patients require close monitoring for both bleeding and thromboembolic complications postoperatively.[6]

To conclude, APS requires a multidisciplinary approach for its diagnosis and management. It is an important medical condition manifesting in pregnant women, where use of anticoagulation leads to the dilemma of its perioperative continuation.[7] Discontinuation of anticoagulation is a double edged sword and requires careful deliberation on part of the anesthetist to reduce the risk of both perioperative bleeding as well as thrombosis.[2]

REFERENCES

1. Hughes G. Hughes syndrome: The antiphospholipid syndrome—a clinical overview. Clin Rev Allergy Immunol 2007;32:3-12. [PubMed]
2. Garg P, Gaba P, Saxena KN, Taneja B. Anaesthetic implications of antiphospholipid antibody syndrome in pregnancy. J Obstet Anaesth Crit Care 2011;1:35-7. [Free Full Text]
3. Silver RM, Draper ML, Scott JR, et al. Clinical consequences of antiphospholipid antibodies: An historic cohort study. Obstet Gynecol1994;83:372-7 [PubMed]
4. Asherson RA. The catastrophic antiphospholipid (Asherson's) syndrome. Autoimmun Rev 2006;6:64-7. [PubMed]
5. Ralph CJ. Anaesthetic management of parturients with the antiphospholipid syndrome: A review of 27 cases. Int J Obstet Anesth 1999;8:249-52. [PubMed] [Free Full Text]
6. Madan R, Khoursheed M, Kukla R, al-Mazidi M, Behbehani A. The anaesthetist and the antiphospholipid syndrome. Anaesthesia 1997;52:72-6. [PubMed]
7. Alijotas-Reig J. Treatment of refractory obstetric antiphospholipid syndrome: the state of the art and new trends in the therapeutic management. Lupus. 2013 Jan;22(1):6-17 [PubMed] doi: 10.1177/0961203312465782.

★★★★★