Epidural Anaesthesia in a Parturient with Guillain–Barre Syndrome

Dear Sir,

The choice of anesthesia in parturients with Guillain–Barre Syndrome is difficult as they have weakness of limbs due to involvement of nerves which may progress to respiratory paralysis. So, general anesthesia was the technique of choice in these patients. Recently, there has been an increase in interest in the administration of neuraxial anesthesia in the form of epidural and spinal anesthesia in these patients. The technique of epidural anesthesia is safer as the extent of block could be diligently extended so as to avoid sudden hemodynamic changes and respiratory compromise. We report a case of a parturient with GBS who presented to us for cesarean section. Epidural anesthesia was administered with 0.375% bupivacaine (lesser than the routinely used anesthetic concentration).

A 25-year-old parturient, G3P2 was diagnosed to have GBS and was referred to us for elective cesarean at 38 weeks of gestation. GBS was diagnosed during her 7th month of gestation when she had gradual progressive weakness of all four limbs and dyspnoea. She was admitted in intensive care unit and required ventilatory support and intravenous immunoglobulin. Ultrasound of fetus showed normal fetal cardiac activity. She was gradually weaned off from ventilatory support and discharged after 25 days of intensive care without any residual weakness or difficulty in breathing. Decision was taken to carry out the cesarean section under epidural anesthesia. Under all aseptic precautions, epidural catheter was inserted in T12-L1 interspace with 18G Tuohy’s needle. Epidural test dose of 45 mg lignocaine and 15 mcg of adrenaline was administered. After 10 minutes, 14 cc of 0.375% bupivacaine (lesser than routinely used 0.5% bupivacaine as anesthetic concentration) and 20 mcg of fentanyl were given through epidural catheter in graded doses of 8 ml followed by 6 ml after 40 minutes of the initial 8 ml dose. The epidural block was established till T8 dermatomal level. There were no episodes of hypotension, bradycardia or dyspnoea throughout the surgery. 90 micrograms of Buprenorphine was given through epidural catheter at an interval of 12 hours for postoperative analgesia. Since she did not develop weakness/sensory disturbance/respiratory compromise, she was discharged on the 8th day after surgery.

As GBS patients have greater sensitivity to local anesthetics,[1] sudden hemodynamic changes in the form of profound hypotension, bradycardia, and cardiovascular collapse may occur after spinal anesthesia. Also, postoperative neurodeficit is a feared complication of subarachnoid block due to increased sensitivity to local anesthetic agents, interaction between local anesthetics, and myelin and direct damage to nerve roots.[2] Pregnancy necessitates rapid sequence induction if general anesthesia is administered. Succinyl choline must be avoided as it leads to hyperkalemia.[3] As GBS is a demyelinating polyradiculoneuritis, sensitivity to non-depolarizing muscle relaxants increases as well; this might result in delayed recovery and postoperative ventilatory support.[4] From this case, we infer that epidural anesthesia with 0.375% bupivacaine would be a more logical choice in a parturient with GBS presenting for elective cesarean section as it is associated with less cardiovascular and respiratory repercussions.

The case described here is a representative case and further studies may be required to prove the safety profile of epidural anesthesia in GBS.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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Dear Sir,

I would like to take the opportunity to address the correspondence to JOACC regarding the manuscript titled thrombotic thrombocytopenic purpura during pregnancy.

First, I would like to start by clarifying the major differences between immune thrombocytopenia (ITP), referred at letter to editor, and thrombotic thrombocytopenic purpura (TTP), presented at the manuscript.

Immune thrombocytopenia (ITP), also called idiopathic thrombocytopenic purpura, or immune thrombocytopenic purpura, is an acquired thrombocytopenia caused by autoantibodies against platelet antigens.

ITP can be primary due to autoantibody-mediated platelet destruction or secondary to an underlying condition including, among others, HIV infection, hepatitis C virus (HCV) infection, systemic lupus erythematosus, and chronic lymphocytic leukemia. Some cases of ITP can be preceded by viral infection. Antibodies against viral antigens may cross-react with normal platelet antigens (a form of molecular mimicry). Infection with HIV, HCV, cytomegalovirus, and varicella-zoster virus have been proposed to cause secondary ITP by this mechanism. Less commonly, preceding bacterial infections may be implicated. Bacterial products, such as lipopolysaccharide, may attach to platelet surfaces and can increase platelet phagocytosis. Helicobacter pylori infection may contribute to the development of ITP in some cases by an unknown mechanism that might include molecular mimicry, immune alterations, and activities of bacterial products such as cytotoxin-associated gene A (CagA).[1]

Observational studies have suggested that therapy for H. pylori infection may improve platelet counts in some individuals with moderate to severe ITP who are from endemic geographical regions such as Japan and Italy.[2,3]

On the other hand, TTP, an example of thrombotic microangiopathy, unlike ITP, is associated with microangiopathic hemolytic anemia, as inferred by the presence of schistocytes on the peripheral blood smear, along with thrombocytopenia, and signs of organ injury.[4] It can be acquired due to an inhibitor (autoantibody) directed against ADAMTS13 or hereditary due to inherited ADAMTS13 mutations. Diffuse microvascular thrombosis cause thrombocytopenia from platelet consumption in thrombi. Contrary to individuals with ITP, who are otherwise well, patients with TTP are often quite ill. Literature review did not reveal a correlation between TTP and Helicobacter pylori infection.

Concerning preeclampsia, a meta-analysis of observational studies that examined the relationship between maternal infection and preeclampsia reported that the risk of preeclampsia was increased in pregnant women with urinary tract infection and periodontal disease. There were no associations between preeclampsia and presence of antibodies to Chlamydia pneumoniae, Helicobacter pylori, and cytomegalovirus; treated and nontreated HIV infection; malaria; herpes simplex virus type 2; bacterial vaginosis; or Mycoplasma hominis.[5]

Low-dose aspirin is the only drug for which there is convincing evidence of benefit in reducing the risk of preeclampsia.[6] In populations with low calcium intake, elemental calcium supplementation for pregnant women can reduce the risk of preeclampsia.[7] Preconception weight loss can reduce the risk of developing preeclampsia.[8]