Case Report

Anesthetic Management of a Case of Ventricular Bigeminy Posted for Elective LSCS

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Abstract

Ventricular bigeminy is a type of dysrhythmia which can complicate any pregnancy and labor. These dysrhythmias occur most commonly as sporadic occurrences in view of anxiety and can be treated by reassurance. Nevertheless, in certain cases where if they occur in an increased frequency even after adequate reassurance and primary supportive care can lead to dangerous morbidities and mortalities. We present the management of a case of 21-year-old female patient presenting with ventricular bigeminy posted for elective lower segment cesarean section in our institution. General anesthesia was considered the anesthetic modality of choice and injections loxicard and metaprolol IV were used intraoperatively to tackle the occurrence of ventricular ectopics. In conclusion, thorough history, clinical examination, and judicious use of antiarrhythmic agents recommended during pregnancy can prevent an asymptomatic ventricular ectopy transforming into a fatal arrhythmia in managing a case of ventricular bigeminy.

Key words: Arrhythmia, lower segment caesarean section, pregnancy, ventricular bigeminy, ventricular ectopy

Introduction

Ventricular bigeminy is a form of ventricular tachydysrhythmias that is characterized by the occurrence of two wide QRS ventricular premature complexes (VPCs) in quick succession alternated by a compensatory pause and normal sinus rhythm.[1] These VPCs are not only common in daily clinical practice but they are largely asymptomatic in more than half of the presenting population of patients. In the absence of heart disease, VPCs are associated with little or no increase in developing into a dangerous arrhythmia.[1] But if they occur in a significantly frequent number they can cause upsetting symptoms in patients and might also lead to life-threatening arrhythmias.[2] Especially if such VPCs complicate pregnancy and parturition, special care has to be taken to avoid dangerous morbidities and mortalities both to the mother and the fetus. This case report can provide the reader, an approach to manage a case of ventricular bigeminy complicating a primi pregnancy.

Case Report

A 21-year-old primigravida at 38 weeks of gestation in labor was admitted to our hospital for elective lower segment caesarean section (LSCS). Her antenatal history revealed spontaneous conception with no complications in the first trimester. She was admitted in a private hospital for her safe confinement where the doctors detected the presence of multiple ventricular premature contractions and then she was referred to our tertiary-care facility. The patient presented with complaints of occasional palpitations with New York Heart Association (NYHA) grade 2 breathlessness during the early second trimester. Electrocardiogram (ECG) findings of the patient showed multiple VPCs more than 8 per min, often in bigeminy with no ST–T changes. She never gave any history of abortions, chest pain, or wheezing and was not a known case of diabetes, hypertension, ischemic heart disease (IHD), or bronchial asthma. She was of moderate built and nourished with bilateral pitting pedal edema below knees.

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On examination, her pulse rate was 109 beats per min and BP 130/100 mmHg. Respiratory system showed bilaterally equal, normal vesicular breath sounds with no rhonchi or crepitations. Her hemoglobin level was 10.5 gm%. Human immunodeficiency virus (HIV), serum electrolytes, random blood sugar, urea, creatinine, Venereal Disease Research Laboratory (VDRL), hepatitis B surface antigen (HbsAg) reports were normal. Her blood group was A positive. Her ECG showed sinus tachycardia, premature ventricular contractions (8–10 per min). Holter study was done and it revealed 200–300 VPCs over a period of 24 h. Her Echocardiography was normal with left ventricular ejection fraction (LVEF) 57%.

Anesthesia technique

After councelling, the patient for nil by mouth the previous night, Alprax tablet (0.25 mg) was prescribed orally. On the day of surgery, informed high risk consent was obtained from the patient in view of recently diagnosed ventricular premature contractions. An 18-gauge branula was secured and anti-aspiration prophylaxis was injected, ranitidine (50 mg) and ondansetron (4 mg) were injected through intravenous (IV) route. The patient was then shifted to the operating room and monitors like ECG, pulse oximetry, and NIBP were attached. A baseline reading of the blood pressure and saturation and heart rate were recorded. The patient was premedicated by injecting glycopyrrolate (0.2 mg) through IV route. The patient was preoxygenated with 100% oxygen for 3 min before induction of anesthesia. Lignocaine (2 mg/kg) was administered through IV route in graded doses. After cessation of the VPCs [Figures 1 and 2], anesthesia was induced by injecting thiopentone through IV route in titrated doses till the loss of eyelash reflex and succinylcholine (100 mg) was given through IV route to facilitate tracheal intubation with an endotracheal tube (ETT) of 7-mm internal diameter (ID). Cricoid pressure was maintained throughout induction till bilateral air entry was confirmed. Anesthesia was maintained with oxygen, nitrous oxide, and isoflurane mixture with intermittent atracurium injection in boluses of 7.5 mg IV dose when the patient had respiratory efforts. Intraoperative tachycardia was managed by injecting metoprolol (1 mg) through IV route based on the response.

A 2.5-kg weight baby was delivered after 6 min which cried immediately at birth with an Apgar score of 9. After fetal extraction, oxytocin was administered as 10 IU in 500 mL NS as a slow IV infusion as IV boluses can cause transient hypotension and tachycardia. Intraoperative hemodynamic status was maintained stable with a systolic blood pressure of 110–100 mmHg, diastolic blood pressure of 60–50 mmHg, and heart rate between 80 and 90 beats per min. No desaturation occurred during the intraoperative phase, end tidal carbon dioxide (EtCO2) level was maintained between 36 mmHg and 40 mmHg, and airway peak pressure did not exceed 25 mmHg. Extubation was performed on the operating table after injecting lignocaine (40 mg) through IV route, and residual neuromuscular blockade was reversed by injecting glycopyrrolate (0.4 mg) and neostigmine (2.5 mg) through IV route. The patient was awake and comfortable after extubation and was shifted to intensive care unit (ICU) for postoperative monitoring and hemodynamic management.

The postoperative period was uneventful. Postoperative analgesia was provided by injecting diclofenac (75 mg) slowly through IV route and paracetamol also through IV route (15 mg/kg/6 h). The patient was monitored in a high dependency unit for two days postoperatively during which she had sporadic episodes of asymptomatic VPCs. A cardiology review of the patient was done on the third day and metoprolol (50 mg) BD was prescribed for rate control, and she was asked to come for follow-up after a month and patient was discharged.

Discussion

Arrhythmia in pregnancy is common and may cause concern for the wellbeing of both the mother and the fetus. The cause for these dysrhythmias in most cases is anxiety which will often only require reassurance.[3] The incidence and severity of atrial and ventricular ectopy are reported to increase during pregnancy.[4,5] Isolated atrial and ventricular ectopics in

Figure 1: ECG showing ventricular bigeminy

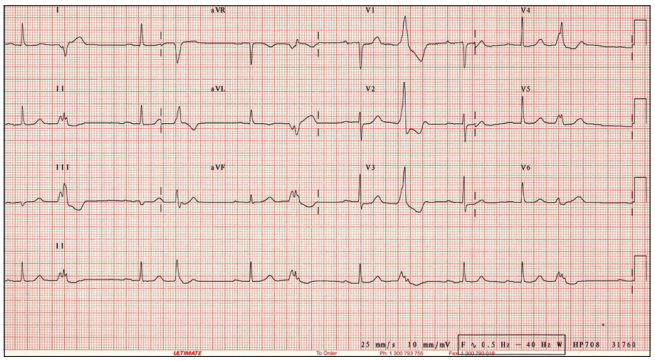

Figure 2: Intraoperative ECG with ventricular bigeminy

pregnant woman without pre-existing heart disease are usually benign.[6] During pregnancy cardiovascular system undergoes significant changes in adaptation to pregnancy, including an increased heart rate and cardiac output, reduced systemic resistance, increased plasma catecholamine concentrations and adrenergic receptor sensitivity, atrial stretch, and increased end-diastolic volumes due to intravascular volume expansion as well as hormonal and emotional changes. A combination of these and the heightened visceral awareness experienced in pregnancy may lead a woman to seek advice on symptoms that are within the normal range and may otherwise have been ignored. The pregnant state is unlikely to generate a new arrhythmia substrate; however, such physiological changes may render a pre-existing substrate capable of sustaining an arrhythmia. Most tachycardia episodes are initiated by ectopic beats, and the occurrence of arrhythmia episodes may therefore increase during pregnancy in line with the increased propensity to ectopic activity.[3]

A thorough history and examination of the patient is paramount. It should be remembered that in the third trimester, patients may become more symptomatic with activity and thus even minor arrhythmias may present with associated symptoms such as breathlessness or chest pain. Palpitations are the most common symptoms and while syncope or pre-syncope may reflect a cardiac cause, it may also result from the physiological drop in blood pressure because of peripheral vasodilatation that is maximal in the second trimester. A thorough study of the patient's family history concentrating particularly on any sudden premature or unexplained deaths that may identify a genetic propensity to life-threatening arrhythmias is also important.[3]

Premature ventricular complexes are more prevalent with increasing age and occur in association with a variety of stimuli such as acute and chronic IHDs such as cardiomyopathy, valvular heart disease, and mitral valve prolapse. Noncardiac causes, such as stimulants (caffeine, cocaine, alcohol) and metabolic abnormalities with acidosis, hypoxemia, and electrolyte abnormalities, have also been implicated in causing VPCs. It is important to determine whether underlying structural heart disease is present and left ventricular function is impaired. The routine investigations should include resting 12-lead ECG, echocardiography, ambulatory Holter recordings, and exercise tolerance test. Echocardiography is important as both ventricular function and the presence or absence of structural heart disease are important considerations in assessing the need for further intervention and treatment.[2]

Attempts have been made to estimate the risk of chronic premature ventricular complexes based on their frequency and waveforms. Several studies have demonstrated an increased risk for life-threatening arrhythmias with 10 or more ectopic impulses per hour or the presence of impulse salvos (i.e., three to five consecutive impulses). However, structural heart disease and poor left ventricular function are the key factors in determining the need for treatment and assessing the prognosis.[7,8]

In our case, we have taken extreme caution in administering pharmacological agents for the VPCs as there are various considerations for antiarrhythmic drug usage during pregnancy.

The decision to treat a woman depends upon the frequency, duration, and tolerability of the arrhythmia. The greatest risk to the fetus is during organogenesis and this is complete by the end of the first trimester. The smallest recommended dose should be used initially and be accompanied by regular monitoring of maternal and fetal clinical condition. Various drugs have been used to terminate arrhythmias, providing useful safety data. However, the literature is limited to single or small case series. These include digoxin, adenosine, amiodarone, flecainide, procainamide, propranolol, propafenone, quinidine, sotalol, and verapamil. The majority of drugs available, however, only have limited evidence for use in pregnancy. Therapeutic levels of drugs may also be difficult to maintain in the pregnant woman as pregnancy tends to decrease the concentration of drugs because of an increased volume of distribution and increased drug metabolism. This may explain why women previously stable on therapy have breakthrough arrhythmias during pregnancy. The safety profile of every antiarrhythmic drug used for the management of LSCS should also be tailored to the response. If patients with multiple VPCs have severe disabling symptoms, Beta blockers are the safest initial choice.[1] Most antiarrhythmic drugs are safe for lactation as well, and if a beta blocker has to be chosen, an agent other than acebutolol should be used.[1]

For our patient we had selected IV lignocaine for its membrane-stabilizing effect that can prevent a sustained VPC that can culminate into a ventricular tachycardia. Intravenously administered lignocaine, given first in a 100-mg bolus (or 1 mg/kg) and then in an infusion at 1–4 mg/min can prevent a ventricular ectopic beats turning into pulseless ventricular tachycardia.[1]

Conclusion

Thus, a thorough clinical history and examination, understanding the disease progression and severity, a vigilant intraoperative monitoring, and selecting drugs to be safely used in the lowest possible doses to avoid an impending cardiac mortality in a parturient posted for LSCS, is vital in managing any cardiac arrhythmia during pregnancy and labour. Knowledge, skill, and expertise in managing arrhythmia in pregnancy cannot only save one life but two.

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Conflicts of interest

There are no conflicts of interest.

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