Atrioventricular nodal reentrant tachycardia occurring during both primary and secondary cesarean deliveries

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INTRODUCTION

Atrioventricular (AV) nodal reentrant tachycardia (AVNRT) is a dysrhythmia caused by a reentry circuit within, or near, the AV node. Typically, the heart rate increases rapidly to 120 – 250 bpm. It is the most common type of paroxysmal supraventricular tachycardia (PSVT). It is also the most common cardiac dysrhythmia in pregnancy.[1] We report a patient who experienced this during both her primary and subsequent secondary cesarean deliveries.

CASE DESCRIPTION

This is a case of a 30 year old G2P1 woman, with a history of palpitations, who presented at 39 weeks gestation for repeat cesarean delivery (CD). The patient stated that her first dysrhythmia occurred immediately before her first CD: "after receiving some medication though the epidural."

Her previous CD record was reviewed. At that time, after administration of 10 ml of lidocaine 2% with epinephrine through her labor epidural, her EKG showed a narrow complex tachycardia. Her heart rate remained in the 160s despite the use of carotid massage, adenosine, esmolol, and phenylephrine. The fetal heart rate decreased to 100 bpm and an emergent CD, under general anesthesia, was performed due to a patchy epidural.

Cardiology was consulted intraoperatively and the patient was diagnosed with AVNRT. Five mg of metoprolol IV was administered. Her heart rate returned to a sinus rhythm at 70 bpm. A postoperative EKG showed PACs with aberrant conduction. A TTE showed no anatomic abnormalities and the patient was discharged on metoprolol 100 mg PO daily.

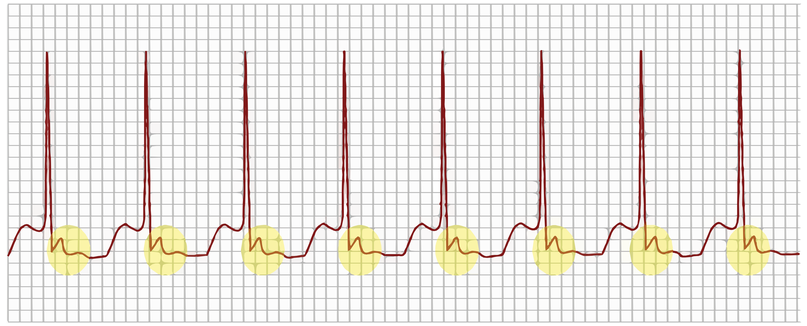

Figure 1:AVNRT is evidenced by the yellow P wave that immediately follows the QRS complex.

Approximately five years following her primary CD, on the day of surgery for her secondary CD, the patient informed the staff that she was not taking her metoprolol; as it was discontinued by her cardiologist 3 months after the initial event. She denied any palpitations since then. Vital signs were: BP 106/55, HR 64, temp 36.6 °C, and RR 20.

Spinal anesthesia was planned for her secondary CD. Shortly after the placement of the spinal anesthetic, which consisted of bupivacaine 0.75% 1.5 ml, fentanyl 15 mcg, and morphine PF 150 mcg, the patient developed a heart rate in the 180s and her EKG showed a narrow complex tachycardia. Despite giving phenylephrine 80 mcg IV, the heart rate did no slow down. She was then given esmolol 10 mg IV, which broke the SVT, and her heart rate returned to 80 bpm. Throughout this episode the fetal heart rate was stable at 145 bpm. The patient did not have any more episodes of AVNRT during her admission. Cardiology was consulted and the patient was started on metoprolol XL 25 mg PO daily. She was also advised to follow up as an outpatient at the cardiology clinic.

Discussion

After reviewing the previous anesthetic record, we originally thought that her first episode of SVT may have been triggered by a partially intravascular epidural catheter; as her EKG changes occurred almost immediately following the initial epidural bolus and the subsequent analgesia was patchy. Since we experienced the same EKG changes shortly after the spinal, it is difficult to conclude that the first episode of SVT was, indeed, triggered by epinephrine.

CONCLUSION

PSVT occurs approximately at a rate of 2.6% in pregnant individuals; mostly as AVNRT.[2] Our patient had an episode of AVNRT during each CD after neuraxial anesthesia, but did not have any underlying cardiac pathology. The dilation of the cardiac chambers, which increases the length of a re-entrant circuit, and a decreased refractory period, are some of the cardiovascular changes in pregnancy that can facilitate AVNRT.[3] Our patient might be particularly sensitive to these conduction changes during pregnancy. Fortunately, the majority of the dysrhythmias, occurring during pregnancy, are non-lethal and resolve as the conduction changes return to their normal non-pregnant physiological state.

REFERENCES

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