Adenosine treatment of supraventricular tachycardia following epidural test dose: A case study

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This is a case report of supraventricular tachycardia following initiation of epidural analgesia with use of an epinephrine test dose in a parturient during active labor. Vagal stimulatory efforts failed to interrupt the arrhythmia, but treatment with adenosine was successful. Fetal monitoring with a scalp electrode provided evidence of fetal well-being throughout the episode. Adenosine was chosen because of its safety for both the mother and the fetus and its lack of the hypotensive effect often seen with verapamil.

Key words: Adenosine, epinephrine, epidural test dose, supraventricular tachycardia.

Introduction
The administration of a test dose of a small quantity of a local anesthetic solution containing epinephrine is recommended when inserting an epidural to rule out intravascular or subarachnoid placement. This case report presents a parturient who experienced an episode of supraventricular tachycardia (SVT) following an epidural dose of 3 mL of a local anesthetic solution containing epinephrine while having a labor epidural placement for analgesia. Prompt reversal of the tachyarrhythmia was achieved with the drug adenosine, a purine-based nucleoside that depresses sinoatrial node automaticity, slows atrioventricular nodal conduction time and nodal reentry tachycardia, commonly known as SVT.

The purpose of this case report is to inform anesthetists of the possible rapid uptake of enough epinephrine from the test dose solution placed in the epidural space to precipitate this type of arrhythmia in susceptible individuals and to introduce the drug adenosine as an effective and safe treatment for both the mother and the fetus.

Case summary
A 30-year-old primiparous female was admitted to the labor and delivery suite with a 39-week intrauterine pregnancy. She had experienced a spontaneous rupture of membranes and was in early labor, with a cervical dilation of 2 cm. Her husband was present and providing support and contact comfort to the patient. The patient was appropriately informed of the availability of labor epidural analgesia early in her admission sequence.

Approximately 5 hours after admission, when the patient had a cervical dilation of 4 cm and was acutely uncomfortable, the attending obstetric/gynecologic physician requested that the epidural be placed. During the preanesthetic assessment, the patient indicated a totally negative history for medical problems, other than the occurrence of occasional "palpitations" when she was under a great deal of stress. These episodes were infrequent and aborted spontaneously without treatment.

The epidural placement was easily achieved
with the patient in the left lateral decubitus position. A #18 Tuohy-Schliff needle was used with the "loss of resistance" technique, using normal saline. The epidural catheter easily threaded to 3 cm and was negative on aspiration for blood or cerebrospinal fluid. A test dose solution of 3 mL of 1.5% lidocaine with epinephrine 1:200,000 was slowly injected. The total dose of epinephrine was 15 μg. There was no evidence of an intravascular catheter placement, since the heart rate remained unchanged for a full 2 minutes.

After 2 minutes, the patient stated that she felt palpitations beginning. The heart rate increased from 88-144 beats per minute (BPM). The blood pressure remained at 110/70, and the oxygen saturation was 96% on room air. Carotid massage was begun, and the patient was asked to perform Valsalva's maneuver on herself. The heart rate continued to increase to about 170 BPM. Additional vagal stimulatory actions were attempted, including a cold, wet towel applied to the face. Oxygen was started with a nasal cannula at 4 L/min. The patient was experiencing only slight lightheadedness during this entire time. The heart rate continued to increase to 175-180 BPM after about 3-4 minutes. The attending obstetric/gynecologic physician was summoned for consultation.

The epidural catheter was secured, and the patient was turned supine with a rolled wedge at the left hip. The head of the bed was elevated to about 60 degrees. Additional vagal stimulatory actions were attempted, including a cold, wet towel applied to the face. Oxygen was started with a nasal cannula at 4 L/min. The patient was experiencing only slight lightheadedness during this entire time. The heart rate continued to increase to about 170 BPM after about 3-4 minutes. The attending obstetric/gynecologic physician was summoned for consultation.

The emergency room physician was also consulted, and it was decided to administer adenosine. A dose of 6 mg was rapidly administered intravenously, with a normal saline flush following the therapeutic dose. Within 30 seconds, the heart rate slowed to a normal sinus rhythm at a rate of 100 BPM.

The electronic fetal monitor revealed no abnormalities preceding or during this entire process. The fetal heart rate continued at a rate of 130-140 BPM. No evidence of abnormal decelerations was noted.

After a repeat negative aspiration, the epidural catheter was slowly dosed with a bolus of 5 mL of bupivacaine 0.25%. (Incremental doses of the therapeutic drug were selected as an alternative to a repeat test dose.) When a dermatome level of T-8 was obtained, a continuous epidural solution of 0.1% bupivacaine, with the addition of fentanyl 2 μg/mL, was started at a rate of 10 mL/hr. A good level of analgesia was maintained throughout the remainder of the first stage of labor as well as the second stage, and an unassisted spontaneous vaginal delivery occurred 5 hours later. There were no further episodes of arrhythmias.

Discussion

Supraventricular tachycardia is a rapid reentrant rhythm. Heart rates usually range from 140-220 BPM with this rhythm. Pregnant women seem to have increased susceptibility to developing atrial tachycardias, and those with a prior history of SVT tend to have more frequent and severe episodes during pregnancy. SVT can be associated with mitral valve prolapse, Wolff-Parkinson-White syndrome, an idiosyncratic response to endogenous adrenalin, or a sensitivity to epinephrine.

Epinephrine is used routinely in test dose solutions in the amounts of 15-20 μg (3-4 mL of a 1:200,000 solution) as a means of ruling out intravascular injection. This dose, if injected into a blood vessel, should quickly produce a transient increase in the heart rate of 20-30 BPM and usually a slight increase in blood pressure.

Although the original investigation by Moore and Batra tested the intravenous dose of 15 μg in nonobstetric patients, other investigators have noted similar changes in the parturient when an epidural vein was accidentally cannulated and the same dose injected. The time of onset of tachycardia is approximately 20-45 seconds and, even more importantly, the duration is only about 30 seconds.

The goal of treatment of SVT is to improve cardiovascular function by terminating the arrhythmia (especially if the patient is symptomatic) to prevent recurrences and to control the fetal and/or maternal ventricular rate. Since vagal stimulation may have only a very temporary or no effect on patients experiencing SVT, a safe medication for both the mother and the fetus is indicated. Previously, verapamil has been used to correct SVT. However, for a laboring woman and fetus, the resultant hypotension can be as problematic as the tachycardia. An alternative to verapamil is adenosine, which is an endogenous nucleoside—a compound formed by partial hydrolysis of nucleic acid. This nucleoside functions as a mediating metabolite in many physiologic processes, including:

1. Regulation of coronary and systemic vascular tone.
2. Platelet function.
3. Lipolysis in fat cells.
4. Intracardiac conduction.

Adenosine interacts with alpha-1 receptors present on the extracellular surface of myocardial cells, as mediated by guanine nucleotide regulator proteins. It activates potassium channels in a manner similar to the effect of acetylcholine, while depressing calcium channel transference. Activation of the potassium channels shortens the atrial action potential duration, hyperpolarizes membrane po-
tential, and decreases atrial contractility. It is an antagonist to catecholamine-stimulated effects of adenylate cyclase to:

1. Decrease cyclic AMP accumulation.
2. Reduce inward calcium conduction.
3. Reduce pacemaker current in the sinus node cells.

His-Purkinje conduction is not directly effected, and adenosine does not affect conduction in normal accessory pathways. Adenosine is removed from the extracellular space by washout, enzymatic degradation to inosine, phosphorylation to AMP, or reuptake into cells through a nucleoside transport system contained in the vascular endothelium. The recommended dosage of adenosine is 6 mg intravenously given rapidly over 1-2 seconds. Injection at a slower rate reduces the useful effect due to its very short half-life of less than 10 seconds. The 6-mg dosage can be repeated, and a second 12-mg total dose is occasionally required if conversion has not occurred within 1-2 minutes.

Most adverse effects associated with adenosine have been transient and resolve in minutes. The most commonly encountered reactions are dyspnea (12-29%), facial flushing (15-18%) and chest pressure (7%). It may also precipitate premature ventricular contractions, premature atrial contractions, sinus bradycardia, and some degree of atrioventricular block. Occasionally, coughing, nausea, asystole, dizziness, and headaches are observed. Caution should be exercised in patients with chronic obstructive pulmonary disease or asthma, because of bronchoconstriction. These adverse reactions have been attributed to the direct effect on organ adenosine receptors and vascular chemoreceptors.

Studies have not been performed in pregnant women, but because adenosine is a naturally occurring material that is widely dispersed throughout the body, no fetal effects would be anticipated. It was believed that the benefits outweighed the risks in this case.

Conclusion
Absorption of even minute amounts of epinephrine from the epidural space can precipitate SVT. A tachyarrhythmia can be an inconvenience in the nonpregnant state and, in the pregnant patient, it can become a source of fetal oxygenation compromise. Its recognition and reversal through the use of intravenous adenosine offers a safer alternative to the use of verapamil.

REFERENCES

AUTHOR
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