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Supraventricular Tachycardia Associated with Postpartum Metoclopramide Administration

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Metoclopramide has wide clinical applications, both as an anti-emetic agent and for its ability to increase lower esophageal sphincter tone and gastric emptying time.[1,2] These properties are particularly useful for patients at increase risk for aspiration of gastric contents during induction of anesthesia.[3,4] In fact, metoclopramide's efficacy in these settings has been documented,[5–7] and the possibility of a wider scope for its use has been entertained.[8–10] Although metoclopramide has potent central anti-dopaminergic and peripheral cholinergic effects,[1,2] it has few side-effects (*e.g.*, drowsiness, fatigue, dystonic reactions), and, if present, are usually seen following excessive doses in young subjects.[1,2] Despite the fact that it differs from its parent compound, procainamide, by only a 2,5 aryl substitution,[2] metoclopramide has relatively minor and infrequent effects on cardiovascular function.[1] There are, however, instances which document metoclopramide's ability to alter cardiac function,[11,12] but never in the peripartum period. A case of supraventricular tachycardia (SVT) associated with metoclopramide administration in the early postpartum period is presented.

CASE REPORT

A 37-yr-old woman was scheduled for elective laparoscopic tubal ligation 4 h after an uncomplicated spontaneous vaginal delivery. Preoperative evaluation revealed that the patient's only significant past medical history was a total of four spontaneous vaginal deliveries. Review of systems was non-contributory, and the patient denied any untoward effects from drugs. Physical examination was judged to be within normal limits. Postpartum serum electrolytes, hemoglobin, and hematocrit were within normal limits. Chest radiograph and electrocardiogram were not obtained.

After a discussion of anesthetic options, the patient requested spinal anesthesia. She was transferred to the operating room where electrocardiogram leads (modified lead II) and an automated blood pressure cuff were placed. Initial readings showed a heart rate of 85 bpm (sinus rhythm) and an arterial blood pressure of 130/80 mmHg. Metoclopramide, 10 mg, was given iv to facilitate gastric emptying and as an anti-emetic. Within 1 min, the heart rate rose to 170 bpm. She was in no distress, being unaware of any change in her status. Arterial blood pressure remained in the 130–140 mmHg (systolic) range throughout the ensuing period. Various maneuvers (*i.e.*, valsalva, carotid sinus massage, ocular pressure) were employed to increase vagal tone without a decrease in the heart rate. After determining that the PR interval was grossly normal, she received two boluses of verapamil (10 mg iv) over a 10-min period without change in heart rate. She was then given alphaprodine (8 mg iv) and droperidol (1.25 mg iv) for sedation. She then received Digoxin (0.75 mg rapid iv infusion) approximately 20 min after the administration of metoclopramide. This was followed by a gradual decrease in her heart rate to 130 bpm over the next 5 min. The patient was then transferred to a telemetry unit for a 24-h period, where her arterial blood pressure remained stable and her heart rate was noted to be in the 90–110 range. Twelve-lead EKG revealed sinus tachycardia with "non-specific" ST changes. PR, QRS, and QTC intervals were within normal limits. Chest radiograph was also without pathology.

The patient was re-interviewed on several occasions, and continued to deny a history of SVT, palpitations, syncope, or any other cardiac symptomatology. Two days after the initial episode, she underwent successful laparoscopic tubal ligation under spinal anesthesia (lidocaine and epinephrine) and sedation (fentanyl and droperidol).

DISCUSSION

Metoclopramide blocks the cardiac dopamine receptors in rats, and, at higher doses, has transient hypotensive effects in cats.[13,14] In other animal studies, high doses (10 mg/kg) of metoclopramide produced bradycardia, and is as effective an anti-arrhythmic as procainamide, to certain chemical stimuli (barium, chloroform, and adrenalin).[15] However, these cardioactive effects are limited by their transient nature and the extreme doses needed to achieve them.[1,2,12]

In humans, metoclopramide has quinidine-like anti-arrhythmic effects and local anesthetic properties.[12] However, when 11 patients undergoing cardiac catheterization for valvular and ischemic disease were given metoclopramide (20 mg IV), they had no significant changes in left ventricular systolic or diastolic pressure, pulmonary systolic and diastolic pressure, cardiac output, heart rate, left ventricular ejection indices, or intra-cardiac conduction measurements.[12] If metoclopramide is stable in the structurally and functionally abnormal heart, then how can one explain the results seen in this case?

First, there is evidence that metoclopramide can be cardioactive in humans.[11,12] One patient, with an aortic Starr valve in place, developed sinus tachycardia (130 bpm) which lasted 3 min after metoclopramide administration.[12] Also, there has been one case report of a woman who developed extra-systoles after metoclopra-

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mide administration (10 mg iv) lasting 1 h, which recurred with administration of the drug.[11]

Second, this patient probably is in a group at increased risk for developing SVT due to her peripartum state.[16–18] Pregnancy and the immediate postpartum period increase the incidence of SVT in susceptible individuals (with both structurally normal and abnormal hearts), as well as in those free of symptoms before, during, or after pregnancy.[16–18] The incidences of SVT during pregnancy has been estimated to be as high as 2.6%.[19] Successful treatment modalities include verapamil, digoxin, and quinidine,[20] as well as D.C. cardioversion[21] and pacemaker implantation.[20]

The mechanisms by which metoclopramide affects cardiac conduction are likely to be multifactorial in nature. Procainamide, which differs from metoclopramide by a single aryl substitution, is known to prolong conduction time through the AV node and occasionally predispose to re-entrant tachycardias.[15] Metoclopramide also stimulates prolactin secretion,[2] which may be cardioactive in its own right. Further, the significant increase in prolactin secretion seen in the peripartum period,[19] combined with the increase seen with metoclopramide administration, may present a possible explanation for the arrhythmia seen in this patient. The changes in cholinergic tone, seen with both pregnancy[19] and metoclopramide,[1,2] may also play a role in the drug's cardioactive effects. One or a combination of these effects may also explain the resistance of this patient's SVT to conventional therapy. It appears that, in this case and in the one previous report of SVT associated with metoclopramide administration,[11] the patient's rhythm returns to normal only after the dissipation of the original metoclopramide injection (about 1 h).

In summary, a stable postpartum patient without prior history of SVT or evidence of structural heart disorder developed SVT immediately after metoclopramide administration. This may be due to the cardioactive properties of metoclopramide[11,12] or to an underlying pre-disposition from her peripartum state,[16–18] or a combination of factors. Therefore, while metoclopramide remains a useful and generally safe agent in the parturient, it may occasionally cause cardiac rhythm disturbances in patients without evidence of underlying functional or structural cardiac abnormalities.

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