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 aryI 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-year-old woman was scheduled for elective laparoscopic tubal ligation 4 h after an uncomplicated spontaneous vaginal delivery. Pre-operative 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 150-140 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 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 (150 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-
mide administration (10 mg iv) lasting 1 h, which re-
curred with administration of the drug.11

Second, this patient probably is in a group at in-
creased risk for developing SVT due to her peripartum
state.16–18 Pregnancy and the immediate postpartum pe-
riod increase the incidence of SVT in susceptible indi-
viduals (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 verap-
amil, digoxin, and quinidine,20 as well as D.C. cardio-
version21 and pacemaker implantation.20

The mechanisms by which metoprololende affects
cardiac conduction are likely to be multifactorial in
nature. Procainamide, which differs from metoprol-
olene by a single aryl substitution, is known to pro-
long conduction time through the AV node and oc-
casionally predispose to re-entrant tachycardias.18 Meto-
prolomide also stimulates prolactin secretion,4 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 meto-
prolomide administration, may present a possible ex-
planation for the arrhythmias seen in this patient. The
changes in cholinergic tone, seen with both pregnancy19
and metoprolomide,1,2 may also play a role in the
drug’s cardioactive effects. One or a combina-
tion of these effects may also explain the resti 
ance of this patient’s SVT to conventional therapy. It appears that, in
this case and in the one previous report of SVT asso-
ciated with metoprolomide administration,11 the pa-
ient’s rhythm returns to normal only after the dissipa-
tion of the original metoprolomide 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 meto-
prolomide administration. This may be due to the car-
dioactive properties of metoprolomide11,12 or to an
underlying pre-disposition from her peripartum
state,16–18 or a combination of factors. Therefore, while
metoprolomide remains a useful and generally safe
agent in the parturient, it may occasionally cause car-
diac rhythm disturbances in patients without evidence of
underlying functional or structural cardiac abnor-
malities.

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