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Pulmonary Aspiration Syndrome after Inhalation of Gastric Fluid Containing Antacids

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Oral administration of antacids to increase gastric-fluid *p*H of pregnant patients at term has gained wide acceptance. It is assumed that aspiration of gastric contents will be less likely to produce chemical pneumonitis when the gastric-fluid *p*H has been elevated by prior administration of antacids. This report describes pulmonary edema and impaired oxygenation in a patient treated with antacids who aspirated gastric fluid with a *p*H of 6.4 during anesthetic induction for cesarean section.

REPORT OF A CASE

A 31-year-old, 78-kg woman, gravida 3, para 1, abortus 1, was admitted at term in early labor. The prenatal course had been uncomplicated, and there was no history of cardiac or pulmonary disease. A lumbar epidural catheter was inserted and analgesia provided with 0.25 per cent bupivacaine. Labor did not progress, and the analgesia was allowed to dissipate. Thirteen hours following admission it was decided to proceed with cesarean section. Reinjection of the epidural catheter did not produce sensory loss, and preparations for general endotracheal anesthesia were initiated. An antacid, Riopan® (magnesium and aluminum hydroxide as a single molecule, 80 mg/ml), 15 ml, had been administered orally every two hours during labor (total 90 ml), with the last dose an hour prior to anesthetic induction.

Anesthetic induction (1150) was achieved with thiamylal, 250 mg, and succinylcholine, 80 mg, preceded by preoxygenation and metocurine, 2 mg. Cricoid pressure was started shortly after thiamylal administration. Fasciculations did not occur. On initiation of direct laryngoscopy the pharynx was found to contain green fluid. An estimated 50 ml of this fluid were suctioned from the pharynx before the glottic opening could be visualized for tracheal intubation. Aspiration through the tracheal tube yielded a "small amount" of similar fluid. An oral gastric tube was placed and 300 ml of green fluid were obtained. The *p*H values of the pharyngeal and gastric aspirates, determined separately (Corning *p*H meter and Beckman *p*H electrode), were 6.4. Ventilation was controlled with 60 per cent nitrous oxide in oxygen until delivery of a male infant (1- and 5-min Apgar scores 7 and 9) at 1210. The first blood-gas analysis, at 1215, revealed Pa_{O_2} 115 torr, Pa_{CO_2} 36 torr, and *p*H 7.40 during controlled ventilation with 50 per cent nitrous oxide in oxygen. Wheezing and decreased compliance were seen at 1300, and pink frothy fluid appeared in the endotracheal tube at 1330 as the surgical procedure was completed. Positive end-expiratory pressure (PEEP, 5 cm H_2O) was applied and a subsequent blood-gas analysis during breathing of 100 per cent oxygen revealed a large A-aD_{O_2} (table 1). At this point, intraoperative fluids consisted of lactated Ringer's solution, 550 ml. Blood pressure and heart rate remained near awake levels throughout the operation. Urinary output since the beginning of the operation was 50 ml.

Arterial oxygenation remained impaired, necessitating controlled ventilation via the tracheal tube with high oxygen concentrations and 5–10 cm H_2O PEEP for the following 72 hours (table 1). The patient's trachea was extubated on the fourth postoperative day, and satisfactory oxygenation was maintained with supplemental oxygen via nasal prongs. Roentgenograms of the chest initially showed bilateral pulmonary infiltrates, which cleared gradually over the following seven days (table 1). Analysis of the gastric aspirate revealed a magnesium concentration of 40 mEq/l.

DISCUSSION

Inhalation of 0.3–0.4 ml/kg gastric fluid with a *p*H below 2.5 is considered likely to produce acid pneumonitis in man.[1] Nevertheless, this critical volume and *p*H remain unproven. The *p*H value of 2.5 has been questioned in a case report describing pulmonary aspiration syndrome following aspiration of gastric fluid with a *p*H of 3.5.[2] In the present case, pulmonary edema and impaired oxygenation occurred following aspiration of an undetermined volume of gastric fluid of *p*H 6.4.

Aspiration is considered acid when *p*H is less than 2.5 and "nonacid" or neutral when *p*H is more than 2.5.[3] Nonacid aspiration can cause either transient or sustained pulmonary damage. The nature and extent of this damage depend not only on the volume aspirated but also the composition of the aspirate, especially its tonicity and the presence of large particles or food material. The presence of digestive enzymes or

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TABLE 1. Summary of Clinical Data

Time	Pa_{O_2} (torr)	Pa_{CO_2} (torr)	*p*H	$F_{I_{O_2}}$	PEEP (cm H_2O)	Radiographic Findings
1215	115	36	7.40	.5	0	Bilateral pulmonary infiltrates consistent with aspiration or pulmonary edema
1340	63	34	7.38	1.0	5	
1500	53	43	7.34	1.0	10	
1900	80	41	7.33	.9	10	
Postoperative day 1	64	32	7.40	.5	10	Bilateral pulmonary infiltrates unchanged; appearance consistent with adult respiratory distress syndrome
Postoperative day 2	53	36	7.48	.6	5	
Postoperative day 4	68	38	7.47	Nasal oxygen	0	Patchy bilateral pulmonary infiltrates
Postoperative day 7						Decreased extent of patchy bilateral pulmonary infiltrates
Postoperative day 14	79	39	7.48	.21	0	Pleural thickening; bilateral pulmonary infiltrates no longer visible

bile in the aspirate is of limited importance in the development of aspiration pneumonitis.[3] The actual volume aspirated by our patient could not be accurately determined, but food particles were not present. However, the aspirate did contain the antacid, as confirmed by the presence of 40 mEq/l magnesium. The effect of this magnesium on the tonicity of the gastric aspirate and subsequent pulmonary effects as manifested by pulmonary edema and impaired oxygenation cannot be determined. In addition, the direct effects of this or other commercially available antacid preparations on the tracheobronchial tree remain unknown.

Pulmonary aspiration is a recognized hazard in pregnant patients, and the logic of prophylactic antacid therapy to increase gastric-fluid *p*H is widely accepted. Recent reports also advocate antacid administration to nonpregnant patients considered to be at risk from pulmonary aspiration.[4] However, the efficacy of this prophylaxis, the potential harmful effects of antacids on tonicity of inhaled fluid, and possible adverse effects of antacids directly on the tracheobronchial tree remain unknown. We do not believe this case report should deter the use of antacids for high-risk patients. However, the use of glycopyrrolate[5] and/or a histamine H_2-receptor antagonist such as cimetidine[6] to increase gastric-fluid *p*H should also be considered in management of high-risk patients. These drugs, particularly cimetidine, might effectively neutralize gastric-fluid *p*H without introducing potential harmful effects from aspiration of gastric fluid containing antacids.

REFERENCES

1. Roberts RB, Shirley MA: Reducing the risk of acid aspiration during cesarean section. Anesth Analg (Cleve) 53:859–868, 1974
2. Taylor G: Acid pulmonary aspiration syndrome after antacids. Br J Anaesth 47:615–617, 1975
3. Wynne JW, Modell JH: Respiratory aspiration of stomach contents. Ann Intern Med 87:466–474, 1977
4. White FA, Clark RB, Thompson DS: Preoperative oral antacid therapy for patients requiring emergency surgery. South Med J 71:177–179, 1978
5. Baraka A, Saab M, Salem MR, et al: Control of gastric acidity by glycopyrrolate premedication in the parturient. Anesth Analg (Cleve) 56:642–645, 1977
6. Stoelting RK: Gastric fluid *p*H in patients receiving cimetidine. Anesth Analg (Cleve) 57:675–677, 1978