

CASE REPORT

HYPOCALCEMIA IN POST CAESAREAN PATIENT PRESENTING AS ACUTE GASTRIC DILATION

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ABSTRACT: Even though Acute Gastric Dilation is rarely encountered complication, if not diagnosed early and treated promptly can lead on to catastrophic events very rapidly. The causal factor for most such cases are benign, hence early diagnosis will help the patient to get conservative management. Here we are describing a post caesarean patient who manifested the features of hypocalcemia and later developed Acute Gastric Dilation. Early establishment of diagnosis in this patient helped to manage the patient conservatively.

KEYWORDS: Acute Gastric Dilation, Hypocalcemia, Caesarean section, Spinal anesthesia.

INTRODUCTION: Acute Gastric Dilation is uncommonly encountered in clinical practice, with varying etiology. Hypocalcemia, anxiety together with post child birth period are all noted to predispose Acute Gastric Dilation. Here we are describing patient who presented with features of Acute Gastric Dilation who was managed successfully with conservative management.

CASE HISTORY: A 27 year old second gravida from poor socio-economic strata was posted for emergency Caesarean section in a peripheral hospital because of previous caesarean section and fetal distress. She gave history of uneventful second pregnancy till date and was booked case. Patient was immunized with two doses of tetanus toxoid and was given nutritional supplements during the pregnancy. Patient underwent hemi-thyroidectomy under general anesthesia 6 years ago uneventfully. She also gave history of previous caesarean section 3 years ago under spinal anesthesia, and surgery was done as there was cephalo pelvic disproportion. No other significant medical or surgical history was given by the patient.

An adult anxious female patient, who was moderately built and nourished, oriented, was found to have pulse of 106 beats per minute with normal character and blood pressure of 136/86 mm of Hg. There was a linear scar of around 7 cm in the neck about 4 cm from the suprasternal notch. Spine and airway were found to be clinically normal. Her uterus was term sized, with no active contractions. No other significant clinical abnormality was found. Hematologic investigations like complete blood count, blood urea and serum creatinine and thyroid stimulating hormone were done and were found to be within normal limits.

Patient was assessed under American Society of Anesthesiologist risk stratification 2E and was taken for surgery under spinal anesthesia. Spinal anesthesia was instituted under aseptic precaution in right lateral decubitus position with 9mg of bupivacaine heavy. Intensity and duration of spinal anesthesia were adequate, after extraction of healthy male baby 20 units of oxytocin was put as infusion. Just after the closure of abdominal musculature, during the inflation of BP cuff patient manifested contractions of hand and Chvostek's sign was also elicited. On

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suspicion of hypocalcemia 500 mg of calcium gluconate was added in the drip. Patient was shifted to post-operative ward, in view of economic background of patient and available resources serum electrolytes with calcium and magnesium assay was advised.

In post-operative ward, 4 hours following surgery, patient had two episodes of vomiting and following which she gave history of burning sensation of the throat later patient gave history of bloated sensation of abdomen and difficulty to breath. Abdominal examination showed a distended abdomen with tenderness in the epigastrium and which was tympanic to percuss over the epigastric region. Chest x-ray was ordered and it showed massive gastric dilation.



Fig. 1: Supine abdominal radiograph showing massively dilated stomach



Fig. 2: Supine abdominal radiograph of same patient after insertion of nasogastric tube

Surgical opinion was sought and nasogastric decompression of stomach was done, the aspirate of around 500 ml of bilious matter was evacuated. Patient was advised to be kept nil per oral for 24 hours and to keep nasogastric tube in-situ, she was asked to get upper gastric endoscopy in higher center.

Serum calcium was found to be 6.2 mg/dl, with normal sodium, potassium and magnesium levels. Patient was put on calcium correction with 1 g of calcium gluconate per day and daily monitoring of serum electrolytes and calcium was done. Nasogastric tube was removed on the first postoperative day, serial abdominal girth chart revealed no further abdominal distension. Oral diet was resumed on the same day, calcium was corrected by oral correction. Patient was subjected to investigations such as vitamin D assay and PTH, she was found to have Vitamin D levels of 18 ng/ml (lab value less than 20 ng/ml needs Vitamin D supplementation). She was given 60,000 IU of Vitamin D twice a week and 500 mg calcium supplementation.

Symptoms of both hypocalcemia and Acute Gastric Dilation improved after correcting hypocalcemia. She was advised for a review after 4 weeks for adequacy and titration of drugs.

DISCUSSION: Acute Gastric Dilation (AGD) was first described by Duplay in 1833.^[1] AGD is a rare but if not treated promptly can have mortality of 80-100%.^[2-5] Patients following surgery

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especially under general anesthesia, debilitated patients, acute infections, emotional stress, trauma, cast syndrome, typhoid, pneumonia, progressive muscular dystrophies, diabetes mellitus, electrolyte disturbances, anorexia nervosa and child birth are few noted predisposing factors for AGD.^[6]

AGD can broadly be classified into obstructive and non-obstructive AGD, obstructive AGD can be caused by obstruction, trauma, and rupture of pulmonary bullae, superior mesenteric artery syndrome, enteric placement of endotracheal tube, and upper endoscopic procedures whereas non-obstructive causes include eating disorders, hemorrhage/trauma resuscitation, medications like mydriatics, electrolyte abnormalities especially potassium and calcium disturbances and infections.

Pathogenesis: It is still not clear about the pathogenesis of AGD many hypothesis have been put forth, "vascular compression of the duodenum" was first theory put forth by Rokitansky in 1842. According to this theory acute gastric dilatation was the result of mechanical compression of the duodenum by the overlying superior mesenteric artery.^[7] But the theory went into disrepute because many evidences suggested that duodenal compression was secondary rather than primary event.

In 1859 Brinton proposed the atonic theory which stated reflex inhibition of the gastric motor nerves and progressive accumulation of secretion and swallowed air as cause of AGD [8]. Moris et al suggested relaxation of superior esophageal spincter following debility or anesthesia as a cause of AGD.^[9] This theory stated that there was continuous suction of gasses during respiration which lead to the AGD. Others suggest the electrolyte disturbances to cause proximal paralytic ileus and AGD. Since there is no substantial evidence supporting any of the above said hypothesis AGD is now thought to be multifactorial. Acute gastric dilatation if not promptly managed can lead on to gastric emphysema, emphysematous gastritis, ischemic necrosis and perforation of stomach especially in the lesser curvature.

Clinical features of AGD were described by Fagge as vomiting in presence of distended abdomen. Some patients also present with abdominal discomfort and inability to vomit. Inability to vomit can be due to compression of fundus on gastroesophageal junction. Patient on examination reveal massively distended abdomen, with doughy feel on palpation and tympanic note on percussion some patients also reveal splash on percussion.

Radiologic investigations like plain X ray abdomen and CT scan can help to clinch the diagnosis. Upper contrast gastrointestinal examination must be performed carefully with a water soluble material to rule out visceral perforation. Endoscopy is essential to find out the health of gastric mucosa and also to rule out mechanical obstructions.

Prime importance in management has to be given to early diagnosis as delay in diagnosis may lead on to life threatening complications. First line of management includes early decompression by nasogastric tube together with fluid resuscitation. Since normal sized nasogastric tube will generally be inefficient, a larger Faucher or Edlich tube can be placed under supervision of anesthesiologist in operating room.

If endoscopy reveals areas of necrosis but no signs of peritonitis are found then conservative management can be done. If conservative management fails then emergency surgical procedure like partial or total gastrectomy should be carried on.^[10]

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In our patient anxiety, post child birth period and hypocalcemia predisposed the patient to AGD. Early decompression of the stomach and correction of underlying hypocalcemia were effective in treating our patient.

CONCLUSION: Early diagnosis and prompt gastric decompression together with close monitoring of the patient can prevent life threatening complication in cases of Acute Gastric Dilatation.

REFERENCES:

1. Todd SR, Marshall GT, Tyroch AH. Acute gastric dilatation revisited. *Am Surg* 2000; 66: 709-710.
2. Powell J. L, Payne J, Meyer C. L, and Moncla P. R. "Gastric necrosis associated with acute gastric dilatation and small bowel obstruction," *Gynecologic Oncology*, 2003; 90(1):200-203.
3. McKelvie P. A. and Fink M. A. "A fatal case of emphysematous gastritis and esophagitis," *Pathology*, 1994; 26(4): 490-492.
4. Luncă S, Rikkers A, and Stănescu A. "Acute massive gastric dilatation: severe ischemia and gastric necrosis without perforation," *Romanian Journal of Gastroenterology*, 2005;14(3): 279-283.
5. Lewis S, Holbrook A, and Hersch P. "An unusual case of massive gastric distension with catastrophic sequelae," *Acta Anaesthesiologica Scandinavica*, 2005;49:95-97.
6. Chaun H et al, Massive Gastric Dilatation of Uncertain Etiology, *Canad. Med. Ass. J.* Feb. 15. 1969; 100: 346-48.
7. Barner H. B. And Sherman C. D. JR: *Int. Abstr. Surg.*, 1963, 117: 103.
8. Bockus, H. L. et al: *Gastroenterology*, vol. 1, 2nd ed., W. B. Saunders company, Philadelphia, 1963, 920.
9. Morris, C. R., Ivy, A. C. and Maddock, W. G.: *Arch. Surg*, 1947; 55: 101.
10. Sorinel Lunca, Andrew Rikkers, Alexandru Stănescu, Acute Massive Gastric Dilatation: Severe Ischemia and Gastric Necrosis without Perforation *Romanian Journal of Gastroenterology*, September 2005; 14(3):279-283.

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