

axis or increases in total serum cortisol and cortisol-binding protein during pregnancy^{3,9}.

Ultrasound images of the adrenal gland, a suppression test with high-dose dexamethasone, and the measurement of ACTH levels are recommended for initial diagnosis. If the patient has borderline or low ACTH levels or no suppression after the dexamethasone test, the condition may be adrenal in origin. Ultrasonography identifies adrenal tumors in 73% of cases⁹. MRI is a second-line test for localization diagnosis¹.

Therapeutic alternatives for adrenal adenoma include unilateral adrenalectomy during pregnancy or medical treatment followed by surgery after delivery¹⁰. No ideal time for performing adrenalectomy has been reported, but most obstetricians and surgeons think that the second trimester is the best time for surgery⁸. A trend to less neonatal complications has been reported in women undergoing surgery during pregnancy, provided maternal conditions are not affected¹. Blood pressure, glucose and potassium levels, and water balance should be strictly monitored after surgery.

Successful results have been reported after treating hypercorticism with metyrapone¹⁰. Metyrapone, an 11-beta-hydroxylase inhibitor, decreases plasma cortisol levels. Although metyrapone may cross the placenta and affect adrenal corticosteroid synthesis, no adverse effects attributable to this treatment have been reported. Other drugs such as ketoconazole, aminoglutethimide, cyproheptadine, or mitotane are contraindicated in pregnancy because of their known teratogenic potential⁸.

References

1. Lo K, Lau T. Cushing's syndrome in pregnancy secondary to adrenal adenoma. A case report and literature review. *Gynecol Obstet Invest.* 1998;45:209-12.

2. Buescher M, McClamrock H, Adashi E. Cushing syndrome in pregnancy. *Obstet Gynecol.* 1992;79:130-7.
3. López S, Fernández L, Castillo M, Payá A, Basil C, Miret M, et al. Síndrome de Cushing en el embarazo. *Prog Obstet Ginecol.* 2006;49:205-9.
4. Torres O, Felpeto M, Rodríguez A, Hernández A. Síndrome de Cushing y gestación. Presentación de una paciente. *Rev Cuba Endocrinol.* 1996;7:125-30.
5. Zubillaga I, Alcubilla O, Marcano A, Zapata L, Adrian O, Padua A, et al. Síndrome de Cushing y embarazo. *Rev Obstet Ginecol Venez.* 1981;41:55-60.
6. Delibasi T, Ustun I, Aydin Y, Berker D, Erol H, Gul K, et al. Early severe preeclamptic findings in a patient with Cushing's syndrome. *Gynecol Endocrinol.* 2006;22:710-2.
7. Buhimschi C, Turan O, Funai E, Azpurua H, Bahtiyar M, Turan S, et al. Fetal adrenal gland volume and cortisol/dehydroepiandrosterone sulfate ratio in inflammation associated preterm birth. *Obstet Gynecol.* 2008;111:715-22.
8. Kita M, Sakalidou M, Saratzis A, Ioannis S, Avramidis A. Cushing's syndrome in pregnancy: report of a case and review of the literature. *Hormones (Athens).* 2007;6:242-6.
9. Lindsay J, Jonklaas J, Oldfield E, Nieman L. Cushing's syndrome during pregnancy: personal experience and review of the literature. *J Clin Endocrinol Metab.* 2005;90:3077-83.
10. Polli N, Pecori Giraldi F, Cavagnini F. Cushing's syndrome in pregnancy. *J Endocrinol Invest.* 2003;26:1045-50.

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Pharmacobezoar induced by enteral nutrition

Farmacobezoar por nutrición enteral

A pharmacobezoar is a collection of drugs or drug excipients that accumulate in the gastrointestinal tract and may induce intestinal obstruction, among other conditions. There are usually predisposing factors (postoperative anatomical changes, gastroparesis, dehydration, or the use of anticholinergic or opiate drugs¹) that contribute to its formation. Very few cases of pharmacobezoar caused by enteral nutrition formulas have been reported, and the condition mainly occurs in the esophagus, probably due to gastric acid reflux.

The case of a patient with an esophageal tumor who required enteral nutritional support for gastrostomy and

experienced intestinal obstruction secondary to such nutrition is reported below. The patient was a 56-year-old male with a history of alcohol-induced liver disease, chronic pancreatitis, and pancreoprivic diabetes mellitus. He had been diagnosed at the age of 55 with a stenosing tumor in the middle esophageal third (T4N1M0), and percutaneous endoscopic gastrostomy had been performed before starting treatment with chemotherapy and radiotherapy. The patient was on an oral diet alone for 7 months, and enteral nutrition (750 mL/day of a normal protein polymeric formula with fiber: 17% protein, 33.2% carbohydrates, 49.8% lipids, and 10.8 g insoluble fat) was started 15 days before hospitalization was required. The patient was admitted to the gastroenterology department for abdominal pain and leakage of nutritional contents around the catheter for approximately the preceding two

days. He was then receiving 2,000 mL of glucosaline with 40 mEq of potassium chloride/24 h, morphine chloride (1 ampoule/24 h), midazolam 15 mg (twice daily), nystatin solution (5 mL/8 h), insulin glargine (8 units/24 h), pantoprazole (40 mg/24 h), and the abovementioned enteral nutrition formula.

Physical examination revealed a poor general condition, skin and mucosal pallor, and wasting. Measurement of vital signs showed blood pressure of 93/70 mmHg, a heart rate of 108 beats per minute, and an axillary temperature of 36°C. Cardiopulmonary auscultation was normal. Diffuse tenderness with no peritoneal signs was found on abdominal palpation, with leakage of nutritional contents around the gastrostomy catheter. Laboratory tests showed leukocytosis ($23.4 \times 10^3/\mu\text{L}$ [normal range {NR}: 3-10]) with neutrophilia (89.9% (NR: 30-70)), creatinine 1.29 mg/dL, urea 106 mg/dL, glucose 170 mg/dL, sodium 133 mmol/L, potassium 4 mmol/L, chloride 93 mmol/L, amylase 16 U/L, and alanine aminotransferase 26 U/L. The gastrostomy catheter was replaced by one of a bigger size (24 Fr), which decreased leakage around the catheter but resulted in more severe pain and abdominal distention. Ultrasound examination and computed tomography of the abdomen and pelvis (Fig. 1a and b) showed obvious signs of mechanical obstruction in the small bowel but did not reveal the cause of obstruction. In view of these findings, urgent laparotomy was performed, which disclosed a significant amount of intra-abdominal free serous fluid and widely dilated small bowel loops from Treitz arch to the ileocecal valve. On palpation, the intraluminal contents became increasingly harder in the distal direction, and were virtually solid at the terminal ileum. Enterotomy was performed at approximately 2 m of the first jejunal loop, and a large amount of semisolid fecal matter was removed.

Appendicostomy was performed and removal of ileal contents through a Foley catheter (no. 30) was attempted, but was not possible because of their virtually solid consistency. A new enterotomy was therefore performed at approximately 50 cm of the ileocecal valve through which material was removed by squeezing the bowel. In total, 6 L of semisolid contents and fecaloid material were removed. A sample was analyzed and was found to be enteral nutrition.

During the postoperative period, the patient required support with total parenteral nutrition because of paralytic ileus. Thirteen days after surgery, enteral nutrition was restarted (fiber-free polymeric formula) as a continuous infusion through the gastrostomy catheter with good tolerability. The patient died 15 days after surgery from infection and respiratory failure.

Bezoars are collections of undigested material which accumulate in the gastrointestinal tract. Three types of bezoar have been described, based on their composition: phytobezoar (vegetal fiber), trichobezoar (hair), and pharmacobezoar (drugs)². Several drugs have been implicated in the formation of pharmacobezoars, including antacids (aluminum hydroxide gel, sucralfate), cholestyramine, psyllum preparations, nifedipine, enteric-coated aspirin, and enteral formulas^{1,2}. Enteral nutrition formulas rich in insoluble fiber promote bezoar formation. The most common clinical signs of bezoar include epigastric pain, nausea, vomiting, and postprandial fullness. A bezoar may cause various complications such as intestinal obstruction, gastrointestinal bleeding, and even gastric or intestinal perforation³. A gastrointestinal bezoar induced by enteral nutrition is a rare cause of intestinal obstruction, and very few cases have been reported in the literature⁴.



Figure 1 A and B Abdominal computed tomography of the patient showing the gastrostomy catheter and distention of small bowel loops.

Despite its low prevalence, it should be included in differential diagnosis because early treatment allows for a good outcome. In a patient receiving enteral nutrition through a gastrostomy catheter who starts to experience heavy peristomal leakage and associated abdominal pain, distal intestinal obstruction should be included in differential diagnosis. In addition, the slowing of gastrointestinal tract motility was promoted in this patient by concomitant administration of opiate drugs and possible gastroparesis secondary to his diabetes.

Conflict of interest

The authors state that they have no conflict of interest.

References

1. O'Neil HK, Hibbeln JF, Resnick DJ, Bass EM, Aizenstein RI. Intestinal obstruction by a bezoar from tube feedings. *AJR*. 1996;167:1477-8.

2. Byrne WJ. Foreign bodies, bezoars, and caustic ingestion. *Gastrointest Endosc Clin N Am*. 1994;4:99-119.
3. Scaife CL, Saffle JR, Jeffrey R, Morris SE. Intestinal Obstruction Secondary to enteral feedings in burn trauma patients. *J Trauma*. 1999;47:859-63.
4. Dedes KJ, Schiesser M, Schäfer M, Clavien PA. Postoperative bezoar ileus after early enteral feeding. *J Gastrointest Surg*. 2006;10:123-7.

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