

Spontaneous Gastric Perforation in a Neonate Presenting as Gastric Outlet Obstruction

Saeid Aslanabadi¹, Atabak Asvadi Kermani², Davoud Badebarin³, Arash Aslanabadi^{4*}

1. Pediatric Health Research Center, Tabriz University of Medical Sciences, Tabriz, Iran

2. Hematology and Oncology Research Center, Tabriz University of Medical Sciences, Tabriz, Iran

3. Pediatric Health Research Center, Tabriz University of Medical Sciences, Tabriz, Iran

4. Students' Research Committee, Tabriz University of Medical Sciences, Tabriz, Iran

Abstract

Gastric perforation in neonates is a rare but frequently fatal condition which is associated with massive pneumoperitoneum in radiography. Here, we report a case of neonatal spontaneous gastric perforation presenting as gastric outlet obstruction rather than pneumoperitoneum. Physical examination and imaging modalities were indicative of abdominal distension and gastric outlet obstruction. With diagnosis of gastric perforation at laparotomy, subtotal gastric resection was performed and a feeding jejunostomy was placed. The present report highlights that gastric perforation should be of clinical suspicion in neonates with abdominal distension and unusual imaging findings rather than pneumoperitoneum.

Keywords: Spontaneous gastric perforation; gastric outlet obstruction; pneumoperitoneum

1. Introduction

Gastric perforation in neonates is a rare but frequently fatal condition of uncertain etiology. It usually occurs in infants requiring resuscitation or having an episode of hypoxia following birth (1). Although massive pneumoperitoneum is a consistent radiographic characteristic in most of the cases, a few reports have highlighted unusual imaging findings in neonatal gastric perforation (2). Here, we report a case of neonatal spontaneous gastric perforation presenting as gastric outlet obstruction rather than pneumoperitoneum in

imaging modalities.

2. Case presentation

A male infant (weight: 3375g) was born at term following an uneventful elective cesarean delivery. During the first day, he developed respiratory distress and hematemesis. Physical examination revealed a lethargic infant with hypotension, hyporeflexia and tachypnea and a blood pressure of 95/65 mmHg, body temperature of 36.4°C, respiratory rate of 66/min, and pulse rate of 150/min. Subsequently, he received cross-matched packed red blood cells and antibiotics

Corresponding author:

Arash Aslanabadi

Students' Research Committee, Tabriz University of Medical Sciences, Tabriz, Iran

Tel: +989104003601 E-mail: aslanabadi.1991@gmail.com

Received: 01-05-2013, Accepted: 20-05-2013, Published: 07-06-2013

doi:10.7575/aiac.abcm.v.1n.1p.24 URL: <http://dx.doi.org/10.7575/aiac.abcm.v.1n.1p.24>
<http://www.abcm.v.1n.1p.24>



(cefazolin, amikacin, and vancomycin) in the maternity hospital.

On day 10 of life, he was admitted to our university-affiliated hospital with abdominal distention. The patient had normal urination and defecation during first 10 days. On admission, physical examination revealed blood pressure 100/70 mmHg, body temperature 36.7°C, pulse rate 130/min, and respiratory rate 44/minute. Abdominal examination showed marked abdominal distension and decreased bowel sounds. Laboratory findings were as follows: white blood cells $13.3 \times 10^3/\text{mm}^3$, hemoglobin 9.5 g/dL and normal metabolic state (pH=7.34, HCO_3^- =21.1 mmol/L and PCO_2 =39 mmHg). Thoracoabdominal X-ray (Figure 1) and barium radiography illustrated an obstruction in stomach and proximal intestinal tract (Figures 2 and 3). Abdominal sonography showed pyloric muscle thickness (5 mm), pyloric channel length of 14mm, and pyloric channel diameter of 12mm, indicative of gastric outlet obstruction.

Based on the preoperative diagnosis of gastric outlet obstruction, laparotomy through supraumbilical incision was performed on day 4 of admission. At laparotomy, remnant barium leaking from the gastric wall was noted. Further inspection revealed a gastric perforation with necrotic rims. While preoperative radiological diagnosis was indicating of a partial obstruction thereupon the barium aggregation in delayed radiographic observations. Subsequently, subtotal gastric resection was performed and a feeding jejunostomy was placed. He made a good recovery and was discharged from hospital 12 days after admission.

3. Discussion

The present report demonstrates a case of spontaneous gastric perforation, despite manifestations of gastric outlet obstruction in both radiological and ultrasound evaluations. To the best of our knowledge, the present case is the first report of neonatal spontaneous gastric

perforation presenting as gastric outlet obstruction rather than pneumoperitoneum. A bizarre presentation of neonatal spontaneous gastric perforation with hydroperitoneum rather than pneumoperitoneum has been reported by Im et al (2).

Visceral perforation in infants is a rare entity (3). Among these, neonatal gastric perforation often occurs without any apparent precipitating event, after which patients deteriorate rapidly (4). Many theories have been proposed for the pathogenesis of gastric perforation; however the etiology is still unknown. Male gender, hyponatremia, metabolic acidosis, low birth weight and prematurity have been suggested to be poor prognostic factors for survival (5).

There are reports of spontaneous gastric perforations in otherwise healthy infants, usually during the first week of life (6). This clinical entity covers any etiology rather than necrotizing enterocolitis or ischemia, trauma following gastric intubation, obstruction of the distal intestine, or accidental stomach insufflation in the course of assisted ventilation. Although perinatal stress and prematurity are common associations, no predisposing factors can be identified in at least 20% of patients with spontaneous gastric perforations (7). Congenital defects in the gastric wall muscles have been hypothesized as the underlying etiologies of the spontaneous perforations (7,8). Spontaneous gastric perforation often occurs at 3 to 5 days of life with poor activity, abdominal distension and respiratory distress as early manifestations (4). Moreover, signs of hypokalemia and decreased perfusion presenting with tachycardia and lethargy has been reported. In the present case, the patient presented with abdominal distension, respiratory distress and tachycardia.

Debridement and two layer closure of the stomach, without significant gastric resections, are the procedures usually performed in the surgical repair of most perforations. In some



cases, gastrostomy may be indicated (9). In the postoperative period, supportive therapy along with the use of broad spectrum intravenous antibiotics is required (10). Survival of these patients is affected by the duration between the onset of symptoms and the start of definitive therapy, the degree of peritoneal involvement, the prematurity grade and the severity of other asphyxia-related consequences. Mortality rates of gastric perforations are high (45-58%) as sepsis and respiratory failure in premature infants give rise to the complications of this clinical entity (6,11). In conclusion, our case highlights that clinical suspicion of gastric perforation should be

focused on neonates with abdominal distension with unusual imaging findings rather than pneumoperitoneum.

Conflicts of interest

The authors declare that they have no conflict of interest.



Figure 1. Thoracoabdominal radiography illuminating severely dilated stomach

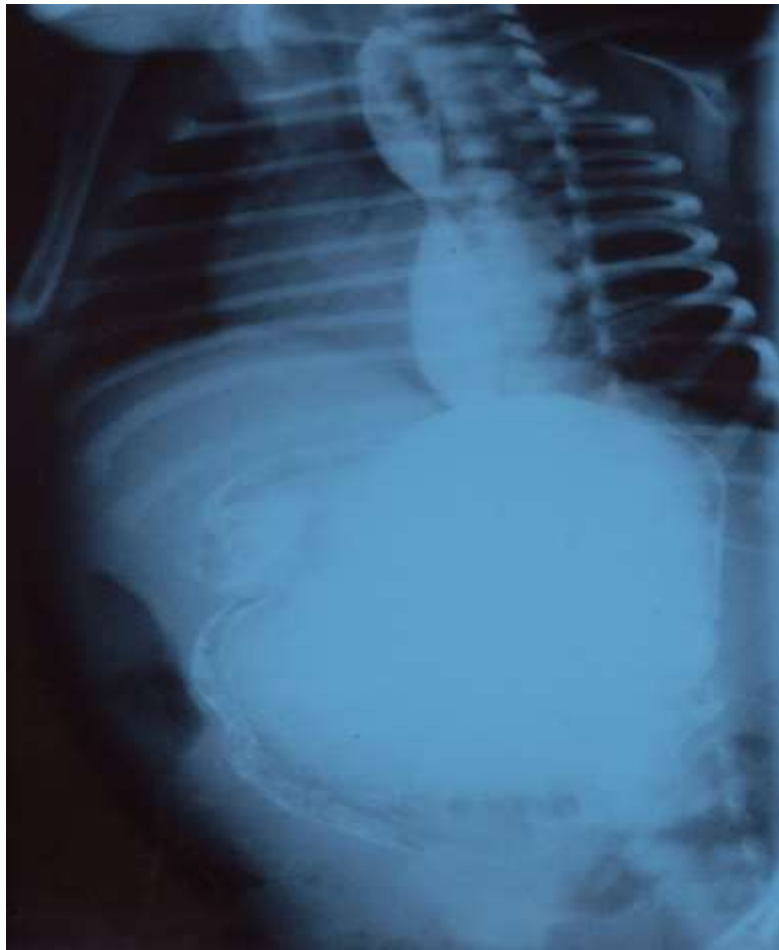


Figure2. Lateral barium radiography indicated of dilation (most probably a dilated stomach)



Figure 3. Delayed radiography. The remaining barium is indicative of a partial obstruction in the gastrointestinal tube

References

1. Kassell NF, Sasaki T, Colohan AR, Nazar G. Cerebral vasospasm following aneurysmal subarachnoid hemorrhage. *Stroke*. 1985;16:562-572.
2. Kassell NF, Torner JC, Haley EC Jr, Jane JA, Adams HP, Kongable GL. The International Cooperative Study on the Timing of Aneurysm Surgery. Part 1: Overall management results. *J Neurosurg*. 1990;73:18-36.
3. Macdonald RL, Weir BK. A review of hemoglobin and the pathogenesis of cerebral vasospasm. *Stroke*. 1991;22:971-982.
4. Sano K, Asano T, Tanishima T, Sasaki T. Lipid peroxidation as a cause of cerebral vasospasm. *Neurol Res*. 1980;2:253-272.
5. Sasaki T, Wakai S, Asano T, Watanabe T, Kirino T, Sano K. The effect of a lipid hydroperoxide of arachidonic acid on the canine basilar artery. An experimental study on cerebral vasospasm. *J Neurosurg*. 1981;54:357-365.
6. Chyatte D. Anti-inflammatory agents and cerebral vasospasm. *Neurosurg Clin N Am*. 1990;1:433-450.
7. Handa Y, Kubota T, Kaneko M, Tsuchida A, Kobayashi H, Kawano H, Kubota T. Expression of intercellular adhesion molecule 1 (ICAM-1) on the cerebral artery following subarachnoid haemorrhage in rats. *Acta Neurochir (Wien)*. 1995;132:92-97.
8. Dorsch NW. Cerebral arterial spasm--a clinical review. *Br J Neurosurg*. 1995;9:403-412.
9. Edvinsson L, Alafaci C, Delgado T, Ekman R, Jansen I, Svendgaard NA, Uddman R. Neuropeptide Y and vasoactive



intestinal peptide in experimental subarachnoid hemorrhage: immunocytochemistry, radioimmunoassay and pharmacology. *Acta Neurol Scand.* 1991;83:103-109.

10. Edwards RM, Stack EJ, Trizna W. Calcitonin gene-related peptide stimulates adenylate cyclase and relaxes intracerebral arterioles. *J Pharmacol Exp Ther.* 1991;257:1020-1024.

11. Faraci FM, Brian JE Jr. Nitric oxide and the cerebral circulation. *Stroke.* 1994;25:692-703.

12. Birk S, Edvinsson L, Olesen J, Kruuse C. Analysis of the effects of phosphodiesterase type 3 and 4 inhibitors in cerebral arteries. *Eur J Pharmacol.* 2004;489:93-100.

13. Pluta RM. Delayed cerebral vasospasm and nitric oxide: review, new hypothesis, and proposed treatment. *Pharmacol Ther.* 2005;105:23-56.

14. Sehba FA, Schwartz AY, Chereshnev I, Bederson JB. Acute decrease in cerebral nitric oxide levels after subarachnoid hemorrhage. *J Cereb Blood Flow Metab.* 2000;20:604-611.

15. Bilginer B, Onal MB, Narin F, Soylemezoglu F, Ziyal IM, Ozgen T. The effects of intravenous cilostazol and nimodipine on cerebral vasospasm after subarachnoid hemorrhage in an experimental rabbit model. *Turk Neurosurg.* 2009;19:374-379.

16. Rybalkin SD, Bornfeldt KE, Sonnenburg WK, Rybalkina IG, Kwak KS, Hanson K, Krebs EG, Beavo JA. Calmodulin-stimulated cyclic nucleotide phosphodiesterase (PDE1C) is induced in human arterial smooth muscle cells of the synthetic, proliferative phenotype. *J Clin Invest.* 1997;100:2611-2621.

17. Sandner P, Kornfeld M, Ruan X, Arendshorst WJ, Kurtz A. Nitric oxide/cAMP interactions in the control of rat renal vascular resistance. *Circ Res.* 1999;84:186-192.

18. Stoclet J, Karavis T, Komar N, Lugnier C. Cyclic nucleotide phosphodiesterases as therapeutic targets in cardiovascular diseases. *Invest Drugs.* 1995;4:1081-1100.

19. Kruuse C, Rybalkin SD, Khurana TS, Jansen-Olesen I, Olesen J, Edvinsson L. The role of cGMP hydrolysing phosphodiesterases 1 and 5 in cerebral artery dilatation. *Eur J Pharmacol.* 2001;420:55-65.

20. Giordano D, De Stefano ME, Citro G, Modica A, Giorgi M. Expression of cGMP-binding cGMP-specific phosphodiesterase (PDE5) in mouse tissues and cell lines using an antibody against the enzyme amino-terminal domain. *Biochim Biophys Acta.* 2001;1539:16-27.

21. Giorgi M, Squitti R, Bonsi P, Paggi P, Toschi G. Activities of 3':5' cyclic nucleotide phosphodiesterases in the superior cervical ganglion of rat: characterization, compartmentalization and observations in young and old animals. *Neurochem Int.* 1994;25:493-500.

22. Lorberboym M, Mena I, Wainstein J, Boaz M, Lampl Y. The effect of sildenafil citrate (Viagra) on cerebral blood flow in patients with cerebrovascular risk factors. *Acta Neurol Scand.* 2010;121:370-376.

23. Zhang R, Wang Y, Zhang L, Zhang Z, Tsang W, Lu M, Zhang L, Chopp M. Sildenafil (Viagra) induces neurogenesis and promotes functional recovery after stroke in rats. *Stroke.* 2002;33:2675-2680.

24. Koktekir E, Erdem Y, Akif Bayar M, Gokcek C, Karatay M, Kilic C. A new approach to the treatment of cerebral vasospasm: the angiographic effects of tadalafil on experimental vasospasm. *Acta Neurochir (Wien).* 2010;152:463-469.

25. Firat MM, Gelebek V, Orer HS, Belen D, Firat AK, Balkanci F. Selective intraarterial nimodipine treatment in an experimental subarachnoid hemorrhage model. *AJNR Am J Neuroradiol.* 2005;26:1357-1362.

26. Hänggi D, Turowski B, Beseoglu K, Yong M, Steiger HJ. Intra-arterial nimodipine for severe cerebral vasospasm after aneurysmal subarachnoid hemorrhage: influence on clinical course and cerebral perfusion. *AJNR Am J Neuroradiol.* 2008;29:1053-1060.

27. Dorhout Mees SM, Rinkel GJ, Feigin VL, Algra A, van den Bergh WM, Vermeulen M, van Gijn J. Calcium antagonists for aneurysmal subarachnoid haemorrhage. *Cochrane Database Syst Rev.* 2007;(3):CD000277.

