

Symptomatic gastric inlet patches in children treated with argon plasma coagulation: a case series

John Brannon Alberty¹, Ricardo Chanis², Vikram Khoshoo³

¹Division of Pediatric Gastroenterology, Our Lady of the Lake Regional Children's Hospital, Baton Rouge, LA; ²Panama Children's Hospital, Balboa, Panama; ³West Jefferson Medical Center, New Orleans, LA (deceased)

Key words: inlet, patch, heterotopic, argon, pediatric

Abbreviations: mg, milligrams; kgs, kilograms; cm, centimeters; GIP, gastric inlet patch; APC, argon plasma coagulation

Abstract: The pathologic potential of gastric inlet patches is now being recognized. A recent adult study has shown the effectiveness of argon plasma coagulation in ablating the patch and the associated symptoms. There are no reports of ablation in children. We report a case series of successful argon plasma coagulation of gastric inlet patches and their symptoms in 5 children.

Introduction

Gastric inlet patch (GIP) is the presence of heterotopic gastric mucosa in the proximal esophagus. It has been associated with several clinical conditions including dysphagia, globus, vocal cord dysfunction, stricture, web, bleeding and reflux. Histologically, these have been associated with esophagitis, Barrett's esophagus, helicobacter pylori infection and adenocarcinoma of the esophagus.¹⁻¹⁷ Few attempts have been made to treat these lesions.^{18,19} A recent randomized controlled trial in adults with globus associated with GIP showed a significant improvement in symptoms after using argon plasma coagulation (APC) as compared to a sham operation.²⁰ APC has also been shown to be safe and effective for ablation of vascular lesions in children.²¹ We present our experience in five children with GIP ablation using APC.

Case 1

A five year old male presented with a 5 month history of intermittent refusal to eat solids. He reported a fear of choking on solids but tolerated liquids. He also had a sensation of globus pharyngeus. There was no history of chest pain or regurgitation. The patient lost 1.5 kgs over the previous 5 months. The physical and oropharyngeal examination was normal. A barium esophagram did not reveal any structural anomalies. The patient was empirically treated with lansoprazole 15 mg twice daily for 2 weeks with no improvement in symptoms. Esophagogastroduodenoscopy was performed revealing two gastric inlet patches in the proximal esophagus (15 cm from the incisors) measuring approximately 2 cm and 1.5 cm in diameter.

Biopsies of the esophagus showed normal histology. Biopsies of the inlet patches showed normal oxyntic mucosa with no inflammation or organisms. Further 4 weeks of acid suppression therapy did not lead to any improvement in symptoms. The endoscopy was repeated for the purpose of APC ablation (ERBE) of the gastric inlet patches using settings of 0.8 Liters/minute and 20 watts. These low settings were chosen based on the patient's small size. These settings caused an inadequate burn. Adequate ablation was achieved with 30 watts and the entire larger lesion was burned. A side-fire probe was used with the rationale that in such a small space, directional control would be required. The second GIP was not ablated due to concern that the opposing patches might adhere together. Over the first week following the procedure, the parents reported significantly fewer symptoms and after the subsequent week the parents reported complete resolution of symptoms. This result was sustained at 3 month follow up with a weight gain of 2 kgs. The second inlet patch was never ablated. All medicines were discontinued with no further symptoms.

Case 2

A seven year old female presented with an 8 week history of cough without fever, weight loss or wheezing. Treatment with antibiotics, inhaled corticosteroids and cough suppressants did not improve the frequency or character of cough. The cough was incessant (hundreds of episodes per day), including nighttime. The cough was worse after eating. Cetirizine 5 mg gave no relief. She was then treated with lansoprazole 30 mg twice daily with no improvement after four weeks of treatment. A complete blood count, liver function tests, electrolytes, thyroid stimulating hormone, sedimentation rate, and C reactive protein were all normal. An esophagogram reported a small, non-obstructing esophageal web at the level of C-4. Upper endoscopy revealed a normal esophagus except for a 2 cm gastric inlet patch about 15 cm from the incisors. No esophageal web was seen. Based

*Correspondence to: John Brannon Alberty; Email: John.Alberty@ololrhc.com
Submitted: Dec/01/2012; Revised: Mar/08/2012; Accepted: Mar/12/2012
Previously published online: www.landesbioscience.com/journals/jig
DOI: 10.4161/jig.22207

on our previous experience we applied APC to the patch at 0.8 Liters/minute and 30 watts. The side fire probe was again used but this time found the direction of the probe opening difficult to control. Therefore, the straight-fire probe was used with success and found no problems controlling the direction of the arc in this larger child. The entire lesion was ablated. The biopsies of the esophagus were normal and the histology of the GIP showed minimal superficial antral gastritis. Biopsies of the GIP and the antrum were negative for *Helicobacter pylori*. The patient experienced mild congestion and cough for 2 days after the procedure, and then near complete resolution of cough. At six week follow up after the procedure the mother reported that patient now only coughed occasionally during the day. All medicines were discontinued with no further symptoms.

Case 3

A nine year old boy presented with a four month history of progressively worsening dysphagia mostly to solids. He developed an apprehension to eat and had lost 3 kgs. There was no history of regurgitation, cough, recurrent pulmonary symptoms, fever, joint pains or change in the bowel frequency or consistency. The physical examination did not reveal any abnormalities. Blood work including complete blood count with differential count, liver functions, electrolytes, sedimentation rate, antinuclear antibodies, thyroid stimulating hormone, free thyroxin 4, total IgA, total IgE and tissue transglutaminase antibodies were within normal limits. A barium esophagogram did not reveal any abnormalities. A 24 hour multichannel pH/impedance study (done after stopping the antacids for 3 weeks) showed pH<4 in the distal esophagus for 1.8% of the time with good bolus clearance. There were a total of 41 episodes of reflux. The patient had been treated with cetirizine 5 mgs daily and nasal steroids for 8 weeks for presumed post nasal drip syndrome with no improvement. Twelve weeks of lansoprazole 30 mgs a day had not improved any symptoms. An upper endoscopy was performed and revealed a GIP at 17 cms from the incisors and was ablated as in case 2.

The biopsies of the esophagus did not reveal any inflammatory changes or eosinophilia. The antral biopsies were negative for helicobacter pylori. The biopsy of the GIP showed normal oxyntic mucosa without acute inflammation (1 eosinophil per high power field) or organisms. On a subsequent follow up, 3 days after ablation, the parents reported significantly improved tolerance to a soft diet. Seven days after the ablation the patient was consuming a regular diet. All medications were discontinued and the patient has remained completely asymptomatic four months after ablation.

Case 4

A sixteen year old female presented with 3 years of nighttime asthma. She had no daytime symptoms, but awoke nightly from sleep requiring almost nightly use of an albuterol rescue inhaler and multiple emergency room visits. Empiric treatment with 30 mg lansoprazole daily for 3 months gave no relief. Endoscopy revealed a 1.5 cm GIP (**Fig. 1**). This was ablated with APC as in case 2 (**Fig. 2**). The procedure was complicated by 2 days of dysphagia to solids, but the asthma episodes stopped. She has been asymptomatic and not used her albuterol inhaler for 1 year.

Case 5

An eleven year old male presented with a 3 month history of dysphagia to solids, globus and a 3 kg weight loss. Empiric treatment with esomeprazole 40 mg daily and cisapride at 0.2 mg/kg was ineffective. CT of the neck and esophagram were unrevealing. Endoscopy showed an 8 mm GIP which was ablated with APC (Conmed) at settings of 50 watts at 1 Liter/minute. The esomeprazole and soft diet were continued for 1 week. The patients symptoms completely resolved and he returned to a normal diet and weight.

Discussion

Tradition has assigned a non-pathologic value to gastric inlet



Figure 1. GIP before

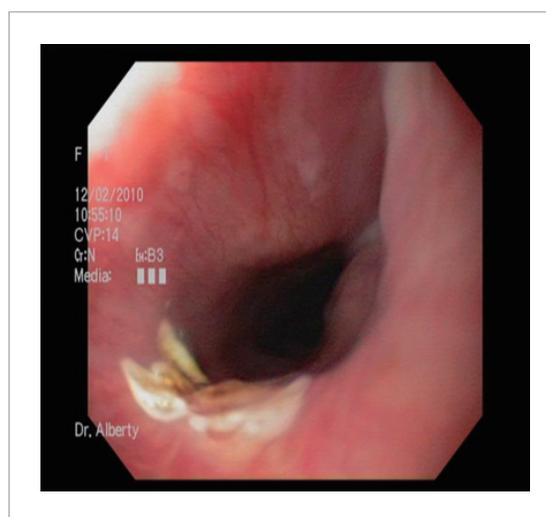


Figure 2. GIP after

patches, but current literature suggests that some patients experience pharyngeal symptoms and experience sustained resolution of after ablation.²⁰ As Bajbouj, et al has shown in adults, our experience in this report also supports the role of APC in resolution of symptoms such as globus, chronic cough, and dysphagia in a small number of children with GIP without any significant adverse events. We chose APC for ablation because the safety in the esophagus is well established in adults and is taking an increasing role in pediatrics especially in the treatment of vascular malformations.

The prevalence of GIP in several large series of adults has ranged from 1.7-3.6%²²⁻²⁴ and in the sole pediatric study it was reported to be 5.9% of 407 endoscopies.⁴ The prevalence of the identification of GIP seems to be linked to the degree of awareness of the endoscopist. Maconi et al reported an 8 fold increase in the prevalence of GIP from 0.29% to 2.27% based on whether the observers were aware versus unaware.²⁵ This prompts us to be more vigilant about identifying these lesions especially during examination of the proximal esophagus. The severity of dysphagia has also been linked to the size of the GIP in adults²⁴ and a higher association of respiratory symptoms has been reported in children with GIP.⁴ Current treatment anecdotally consists of PPI, but no intervention studies have been described for GIP in pediatric patients. Certainly, our data provide a rationale for a controlled trial in children such as the one by Bajbouj et al.²⁰

References

1. Alagozlu H, Ergun M, Cindoruk M, Unal S, Dumlu S, Poyraz A, et al. The rare presentations of a large polyp and an esophageal carcinoma in heterotopic gastric mucosa: a case series. *J Med Case Reports* 2007; 1:127.
2. Alaani A, Jassar P, Warfield AT, Goulesbrough DR, Smith I. Heterotopic gastric mucosa in the cervical oesophagus (inlet patch) and globus pharyngeus--an under-recognised association. *J Laryngol Otol* 2007; 121:885-8.
3. Silvers WS, Levine JS, Poole JA, Naar E, Weber RW. Inlet patch of gastric mucosa in upper esophagus causing chronic cough and vocal cord dysfunction. *Ann Allergy Asthma Immunol* 2006; 96:112-5.
4. Macha S, Reddy S, Rabah R, Thomas R, Tolia V. Inlet patch: heterotopic gastric mucosa-another contributor to supraesophageal symptoms? *J Pediatr* 2005; 147:379-82.
5. Basseri B, Conklin JL, Mertens RB, Lo SK, Bellack GS, Shaye OA. Heterotopic gastric mucosa (inlet patch) in a patient with laryngopharyngeal reflux (LPR) and laryngeal carcinoma: a case report and review of literature. *Dis Esophagus* 2009; 22:E1-5.
6. Satoh S, Nakashima T, Watanabe K, Toda S, Kuratomi Y, Sugihara H, et al.

Hypopharyngeal squamous cell carcinoma bordering ectopic gastric mucosa "inlet patch" of the cervical esophagus. *Auris Nasus Larynx* 2007; 34:135-9.

7. Tang P, McKinley MJ, Sporrer M, Kahn E. Inlet patch: prevalence, histologic type, and association with esophagitis, Barrett esophagus, and antritis. *Arch Pathol Lab Med* 2004; 128:444-7.
8. Abe T, Hosokawa M, Kusumi T, Kusano M, Hokari K, Kagaya H, et al. Adenocarcinoma arising from ectopic gastric mucosa in the cervical esophagus. *Am J Clin Oncol* 2004; 27:644-5.
9. Gutierrez O, Akamatsu T, Cardona H, Graham DY, El-Zimaity HM. Helicobacter pylori and heterotopic gastric mucosa in the upper esophagus (the inlet patch). *Am J Gastroenterol* 2003; 98:1266-70.
10. Malhi-Chowla N, Ringley RK, Wolfsen HC. Gastric metaplasia of the proximal esophagus associated with esophageal adenocarcinoma and Barrett's esophagus: what is the connection? Inlet patch revisited. *Dig Dis* 2000; 18:183-5.
11. Avidan B, Sonnenberg A, Chejfec G, Schnell TG, Sontag SJ. Is there a link between cervical inlet patch and Barrett's esophagus? *Gastrointest Endosc* 2001; 53:717-21.
12. Lauwers GY, Scott GV, Vauthey JN. Adenocarcinoma of the upper esophagus arising in cervical ectopic gastric mucosa: rare evidence of malignant potential of so-called "inlet patch". *Dig Dis Sci* 1998; 43:901-7.
13. Berkelhammer C, Bhagavan M, Templeton A, Raines R, Walloch J. Gastric inlet patch containing submucosally infiltrating adenocarcinoma. *J Clin Gastroenterol* 1997; 25:678-81.
14. Nakajima H, Munakata A, Sasaki Y, Yoshida Y. pH profile of esophagus in patients with inlet patch of heterotopic gastric mucosa after tetragastrin stimulation. An endoscopic approach. *Dig Dis Sci* 1993; 38:1915-9.
15. Yarborough CS, McLane RC. Stricture related to an inlet patch of the esophagus. *Am J Gastroenterol* 1993; 88:275-6.
16. Steadman C, Kerlin P, Teague C, Stephenson P. High esophageal stricture: a complication of "inlet patch" mucosa. *Gastroenterology* 1988; 94:521-4.
17. Waring JP, Wo JM. Cervical esophageal web caused by an inlet patch of gastric mucosa. *South Med J* 1997; 90:554-5.
18. McBride MA, Vanagunas AA, Breshnahan JP, Barch DB. Combined endoscopic thermal electrocoagulation with high dose omeprazole therapy in complicated heterotopic gastric mucosa of the esophagus. *Am J Gastroenterol* 1995; 90:2029-31.
19. Meining A, Bajbouj M, Preeg M, Reichenberger J, Kassem AM, Huber W, et al. Argon plasma ablation of gastric inlet patches in the cervical esophagus may alleviate globus sensation: a pilot trial. *Endoscopy* 2006; 38:566-70.
20. Bajbouj M, Becker V, Eckel F, Miehle S, Pech O, Prinz C, et al. Argon plasma coagulation of cervical heterotopic gastric mucosa as an alternative treatment for globus sensations. *Gastroenterology* 2009; 137:440-4.
21. Khan K, Schwarzenberg SJ, Sharp H, Weisdorf-Schindele S. Argon Plasma Coagulation: Clinical Experience in Pediatrics. *Gastrointestinal Endoscopy* 2003; 57:110-2.
22. Yüksel I, Usküdär O, Köklü S, Başar O, Gültuna S, Unverdi S, et al. Inlet patch: associations with endoscopic findings in the upper gastrointestinal system. *Scand J Gastroenterol* 2008; 43:910-4.
23. Akbayir N, Alkim C, Erdem L, Sökmen HM, Sungun A, Başak T, et al. Heterotopic gastric mucosa in the cervical esophagus (inlet patch): endoscopic prevalence, histological and clinical characteristics. *J Gastroenterol Hepatol* 2004; 19:891-6.
24. Poyrazoglu OK, Bahcecioglu IH, Dagli AF, Ataseven H, Celebi S, Yalniz M. Heterotopic gastric mucosa (inlet patch): endoscopic prevalence, histopathological, demographical and clinical characteristics. *Int J Clin Pract* 2009; 63:287-91.
25. Maconi G, Pace F, Vago L, Carsana L, Bargiggia S, Bianchi Porro G. Prevalence and clinical features of heterotopic gastric mucosa in the upper oesophagus (inlet patch). *Eur J Gastroenterol Hepatol* 2000; 12:745-9.