

Risk continues: Very late manifestation of helicobacter pylori at gastric augmented bladder

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Abstract

Helicobacter Pylori was previously demonstrated at gastric patch after gastrocystoplasty and a possible relationship with acid-haematuria syndrome was established after symptomatic relief by medical treatment. We present the long term outcome of a male bladder exstrophy patient after gastrocystoplasty. There was past history of bladder perforation, acid haematuria syndrome and treatment of HP and recurrent urinary tract infections, noncompliance on regular follow-up and cadaveric renal transplantation. At the preoperative evaluation for renal transplantation HP was present in the biopsy samples collected during gastroscopy and cystoscopy. The significance of persistent HP after gastrocystoplasty in the long term follow-up was discussed.

Keywords: Cancer, Gastrocystoplasty, Helicobacter pylori, Peptic ulcer, Renal transplantation.

Introduction

Gastrocystoplasty may be considered in patients with renal insufficiency and short length of bowel. The advantages include aciduria, less mucous secretion, less urinary tract infections and stone.¹ Acid-haematuria syndrome, hyponatremichy pochlormal kalosis, hypergastrinaemia, perforation, development of peptic ulcer and malignancy should be encountered in long term follow-up. Helicobacter Pylori infection is involved in the pathogenesis of several disease such as peptic ulcer disease, mucosa associated lymphoid tissue lymphomas (MALTomas), intestinal metaplasia and gastric adenocarcinoma.²⁻⁵ We also demonstrated the presence of Helicobacter Pylori at gastric patch in patients with acid-haematuria syndrome^{6,7} and the symptoms disappeared after medical treatment. Here, we review the follow-up of a patient with gastrocystoplasty and emphasize the importance of HP infection in the long term.

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Case Report

The medical records of a 27 years old male bladder exstrophy patient was reviewed retrospectively. Transureteroureterostomy, ureteroneocystostomy, gastrocystoplasty and epispadias repair was performed at 4 years of age after failed repairs. He was discharged on clean intermittent catheterisation (CIC) and prophylaxis. Acute abdomen secondary to bladder perforation was diagnosed six months later. Conservative treatment failed and exploration was indicated. A perforation side was not identified and effective drainage of urine extravasation by ureter stent and suction drains were provided. The importance of CIC, prophylaxis, haematuria, febrile urinary tract infections and regular follow-up was again established.

We documented Helicobacter Pylori at the gastric part of bladder in patients suffering from haematuria-dysuria syndrome after gastrocystoplasty. Subsequently, this patient was re-evaluated for this relationship at 9 years of age. The blood Elisa test for Helicobacter Pylori was (+). At cystoscopy Helicobacter Pylori was absent on tissue samples from gastric patch of the bladder. Haematuria disappeared after medical treatment with antibiotics and proton pump inhibitor (Amoxicillin 40 mg/kg/day, Metronidazole 25 mg/kg/day, Bismuth subsalicylate 8 mg/kg/day) for 2 weeks.

He had suffered from intermittent haematuria, pain, problems related to CIC and hydronephrosis for 12 years. He was hospitalized for recurrent UTI at different centers repeatedly and was treated by wide spectrum antibiotics. The follow-up was not regular even at the same medical center. End stage renal disease had developed at 20 years of age. He was finally scheduled for renal transplantation after haemodialysis. The blood Elisa test for Helicobacter Pylori was positive. Upper gastrointestinal endoscopy and cystoscopy revealed the presence of Helicobacter Pylori at stomach and gastric patch in the bladder (Figure). He was treated with antibiotics and proton pump inhibitor for 2 weeks (Amoxicillin 40 mg/kg/day, Clarithromycin 2 X 500 mg /day, Lansoprazole 2 X 30 mg/ day). The serology of EBV was also positive. At that time, successful cadaveric renal transplantation was performed. He is on CIC for

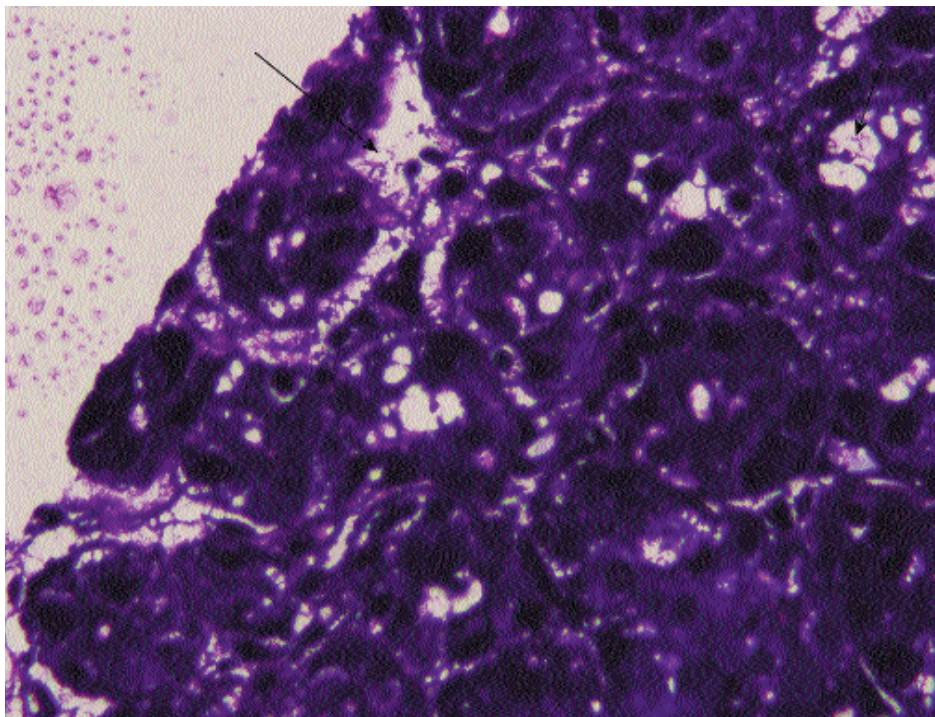


Figure: Few *Helicobacter pylori* organisms in surface mucus (arrows) (Modified Giemsa stain X 1000).

every 3 h/day and on medications after transplantation. There is still self limiting intermittent haematuria for a few days for every 3 months.

Discussion

Acid haematuria syndrome is a common complication after gastrectomy. We established a relationship between *Helicobacter Pylori* infection and acid haematuria syndrome by demonstration of *Helicobacter Pylori* in the gastric patch and disappearance of symptoms after medical treatment.¹ Since, *Helicobacter Pylori* is common in general population, we recommend routine *Helicobacter Pylori* investigation before gastrectomy and medical treatment if presents. *Helicobacter Pylori* mainly infects the gastric mucosa and the possible contamination way of gastric patch is still obscure.^{2,5}

We observed the persistence of *Helicobacter Pylori* in the gastric patch in the long term follow up of a patient with gastrectomy who had a past history of severe intermittent acid haematuria syndrome responding to medical treatment and bladder perforation.^{6,7} In our clinical practice, once we achieve the disappearance of symptoms we do not perform a redo biopsy of gastric patch to detect *Helicobacter Pylori*. In this case, before the renal transplantation upper gastrointestinal endoscopy

and cystoscopy revealed the presence of *Helicobacter Pylori*. Although the patient was treated for *Helicobacter Pylori* infection in the past, the presence of *Helicobacter Pylori* may be a consequence or continuing colonization of the primary *Helicobacter Pylori* infection, antibiotic resistance and/or a possible new *Helicobacter Pylori* reinfection.⁸

Atrophic gastritis was found in biopsy samples after gastrectomy.⁹ Malignant transformation may develop after gastrectomy in the long term follow-up.⁹⁻¹¹ The presence of *Helicobacter Pylori* in the gastric patch may be important as, a predisposing factor for gastric atrophy. Bladder cancer was diagnosed in immunocompromised patients with post-transplant CMV, BK or EBV infections.^{10,12,13} The risk of

gastric cancer is also higher after renal transplantation and the relationship between *Helicobacter Pylori* infection is questionable.¹⁴

We determined the presence of *Helicobacter Pylori* in the stomach and gastric patch, with immunosuppressive therapy after renal transplantation, and EBV positivity being the potential risk factors for the development of a malignancy in the stomach and bladder, following gastrectomy in this particular patient.

Persistent *Helicobacter Pylori* in the gastrectomy is a hidden danger for highly potential risks of acid haematuria syndrome, perforation and malignancy. *Helicobacter Pylori* infection risk continues throughout life even after medical treatment. The prevalence of *Helicobacter Pylori* infection is high in paediatric candidates for renal transplantation and most of the patients are asymptomatic.¹⁵ We recommend investigation of *Helicobacter Pylori* infection in gastrectomy patients before and after the augmentation and following renal transplantation.

Conflict of Interest: The authors declare no conflict of interest.

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