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Case Report

Acquired non-hypertrophic and non-peptic primary pyloric functional obstruction (Jodhpur disease) first time described in African children

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ABSTRACT

The occurrence of Jodhpur Disease (JD) in two African children is reported. It is a rare, acquired, primary gastric outlet obstruction typical of Asian patients, mainly from India, not related to anatomic lesions, muscular hypertrophy, or peptic ulcer of the pylorus. It typically presents at a mean age of 2.9 years, predominantly in males. Two males, 2 and 12 years old, presented with non-bilious vomiting in the last three weeks and two months, with electrolyte imbalance and malnutrition. Helicobacter pylori infection was excluded. Ultrasound, Gastrointestinal Radiology and Gastroscopy concurred to identify the features of JD. Pyloroplasty reestablished regular transit. First described in 1997, JD appears to be confined to the Asian context. Its etiology is undefined. Occurrence in previously healthy children has been attributed to a possible neuromuscular enzymatic functional pathology. Nutritional, environmental or genetic causes have been speculated. JD disease responds to a simple pyloroplasty, followed by patient recovery. JD in two African children denies the assumption that it is exclusive to Asian individuals. It should be considered in other contexts, particularly in the presence of persistent, unexplained vomiting and malnutrition. If recognized with adequate expertise, it can be resolved with a simple procedure.

Keywords: Jodhpur disease, Gastric outlet obstruction, Child

INTRODUCTION

Jodhpur disease (JD) is a rare condition that typically occurs in children. It was first described in Jodhpur, India, in 1997 as an acquired gastric outlet obstruction (GOO) not secondary to peptic lesions and without the pyloric muscular hypertrophy that commonly occurs in the first months of life. JD is associated with recurrent non-bilious vomiting, progressive weight loss, abdominal pain, and distension.[1]

The reported incidence is 1 in 100,000, with a slight predilection for males. The mean age of diagnosis is 2.9 years, with a wide range from one month to sixteen years.[2] The rare occurrence of JD, with progressive malnutrition and growth retardation, far from the age for typical pyloric hypertrophic stenosis, can lead to delayed recognition and treatment. The prevalent Asian origin of the reported cases makes the occurrence of JD unexpected in other parts of the World, challenging the identification of the disease. Authors present the first two African cases with the features of JD.

CASE REPORTS

Case 1

A two-year-old male was admitted for non-bilious vomiting after every meal in the last three weeks, with moderate weight loss and without abdominal pain, fever or other symptoms. At physical examination, signs of dehydration were noted, with distension of the epigastric region. Normal blood count was recorded with abnormal serum electrolytes (Na+ 143 mmol/l, K+ 2.6 mmol/l) and acid-base status (HC03-act, r 27.0 mmol/l, HC03-std, r 28.1 mmol/l, BE (ECF), r 3.9 mmol/l, BB(B), r 52.0 mmol/l). The *Helicobacter pylori* test was negative. A plain abdominal X-ray revealed a distended, fluid-filled

stomach with poor intestinal air content (Figure 1). The pyloric canal appeared normal on ultrasound. Upper endoscopy revealed an abundant gastric content with normal fundic and antral mucosa. A stenotic pyloric ring was associated with diffused oedema of the gastric outlet mucosa (Figure 1b). At laparotomy, the stomach was distended, and the small bowel and colon were normal. The incision for a Finney pyloroplasty did not reveal signs of wall muscular hypertrophy or scarring (Figure 1c). The pathologist confirmed these features on the intraoperative specimen. Postoperative recovery was rapid, and normal oral feeding could be resumed within one week. Normal weight for age was regained in one month.

Case 2

A 12-year-old male was referred with projectile non-bilious vomit for two months, associated with weight loss and recurrent epigastric pain, no fever. Blood count was normal, but a hyponatremia with hypokalemia was registered. The abdomen was scaphoid with a positive succussion splash test in the epigastric region. The pyloric channel resulted in normal at ultrasonography, and the Upper gastrointestinal series documented a distended stomach with a delayed pyloric transit (Figure 2). The *Helicobacter pylori* test was negative. Endoscopy described a pyloric stenotic ring with diffuse mucosal gastric hyperemia. A Heineke-Mikulicz procedure was performed, and the pyloric wall was found to be free from scarring or muscular hypertrophy, as later confirmed by the pathology report. The postoperative course was uneventful, and regular oral feeding was restored within a week. Body weight for age was regained in a short time.

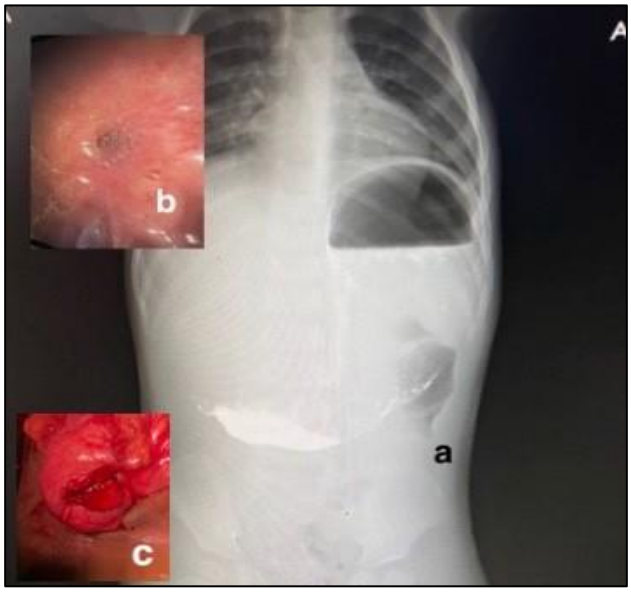

Figure 1: Distended fluid-filled stomach (a) Endoscopic appearance of pyloric stenosis; (b) open view of the pyloric channel; and (c) during Finney's procedure.

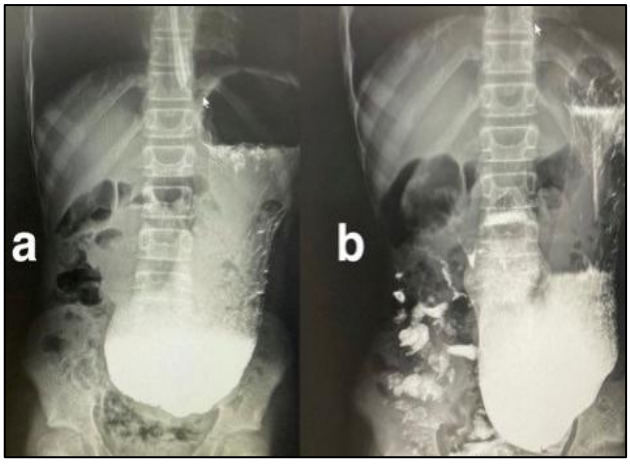

Figure 2: Upper GI series showing a massively distended stomach with delayed pyloric transit of contrast. (a) Start (b) After 5 hours.

DISCUSSION

Sharma et al described the first case of JD in five children aged 3 months to 6 years, all from Jodhpur in North India. A review was published by the same Author in 2008 and other cases were reported from different Countries, all gathered under the same definition from the place where the first cases were reported.[1-7] All had in common a history of non-bilious vomiting after feeding, abdominal distension and/or abdominal pain, a normal length of the pyloric channel without ultrasonographic signs of hypertrophy, gastric distension and delayed emptying at radiology, and no endoscopic findings of peptic lesions or inflammation.[3,10] Tests for *Helicobacter pylori* were negative whenever done. These strict diagnostic criteria were recently confirmed.[8,9]

Universal benefit from Heineke-Mikulicz's procedure suggests a pyloric ring abnormality in terms of function. This operation involves making a longitudinal incision along the pylorus and then suturing it transversely to widen the gastric outlet. Finney pyloroplasty can also be adopted. The histopathology pattern of JD is typical, excluding fibroproliferative, neoplastic or inflammatory features.[3] A less invasive alternative to surgery that has been explored is pneumatic dilation of the pyloric channel with a balloon via an upper endoscope. This method has been successful in only one case treated without surgery.[11] All reported patients satisfying the criteria for JD are from Asian countries. For three more cases from France, identifiable as JD, the ethnicity was not specified.[12] No cases from Africa have been reported to date.

It has been suggested that a dietary factor may contribute to the condition, as the staple diet in the Jodhpur region, an area characterized by a dry, hot climate with very little rainfall, is the cereal Bajra (millet).[3] Nevertheless, the disease has been encountered in other parts of Asia with varied weather and climate, and some cases have been

reported in patients born of consanguineous marriages, indicating a possible genetic linkage.[13] However, the genetic factors contributing to the development of JD are not well understood, and further research is needed to identify these factors and understand their role.

The etiology of JD remains poorly understood. Several theories have been proposed, including neuromuscular incoordination from an unspecified agent leading to permanent changes in pyloric function or a lack of nitric oxide synthase, failing the pyloric smooth muscles to relax.[3,11,12] Abnormality of the interstitial cells of Cajal, acting as pacemakers for the gastrointestinal smooth muscle cells, could contribute to the condition. More research is needed on the etiology of JD through advanced studies in immunohistochemistry and neuromuscular enzymatic pathophysiology.

The interest of our cases lies in the absence of any previously published report of JD among African children. Diagnosis of JD in our patients adhered to accepted criteria, and the response to pyloroplasty was rapid and complete, as observed in other reported cases. Although JD appears to be exclusive to Asian children due to unexplored genetic or environmental reasons, it cannot be excluded that in large, underserved African rural areas, cases of malnutrition associated with persistent vomiting could hide a possible JD. Recognition and simple, resolutive treatment could be missed due to limited access to expert medical attention and diagnostic services.

CONCLUSION

Although JD appears to be exclusive to Asian children due to unexplored genetic or environmental reasons, it cannot be excluded that in large, underserved African rural areas, cases of malnutrition associated with persistent vomiting could hide a possible JD. Recognition and simple, resolutive treatment could be missed due to limited access to expert medical attention and diagnostic services.

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