Gastric Antral Vascular Ectasia (GAVE) Syndrome

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Abstract

Gastric antral vascular ectasia (GAVE) syndrome is an uncommon cause of chronic gastrointestinal bleeding and iron deficiency anaemia. We describe two cases of GAVE, one pernicious anaemia related and the other portal hypertension related. In both the cases, progressive mucosal changes, which lead to development of GAVE, were documented. Those changes were progression of multiple antral erythematous spots into linear configuration and lastly to watermelon stomach. One of the cases was treated with tranexamic acid with good response. ©

Introduction

In 1953, Rider *et al* described gastric antral vascular abnormalities, which were given the name of watermelon stomach by Jabbari *et al* in 1984. GAVE presents clinically as chronic gastrointestinal bleeding and endoscopically as appearance resembling watermelon. Little is known about its pathogenesis.[1] Although surgical antrectomy is curative, which may not be possible in diffuse disease or in presence of portal hypertensive collaterals, there is a role of medical as well as endoscopic treatment.

Till date to our knowledge, no report in the world literature describes progressive mucosal appearances that lead to development of GAVE. Here, we have documented progressive changes in endoscopic appearances in two cases, in which typical endoscopic picture of GAVE developed later.

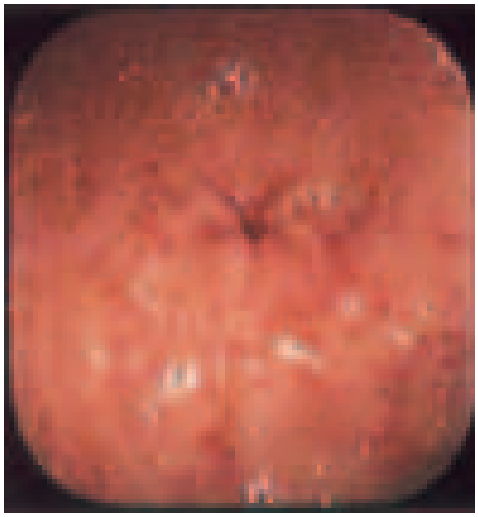

Fig. 1 : Multiple erythematous spots in the antrum

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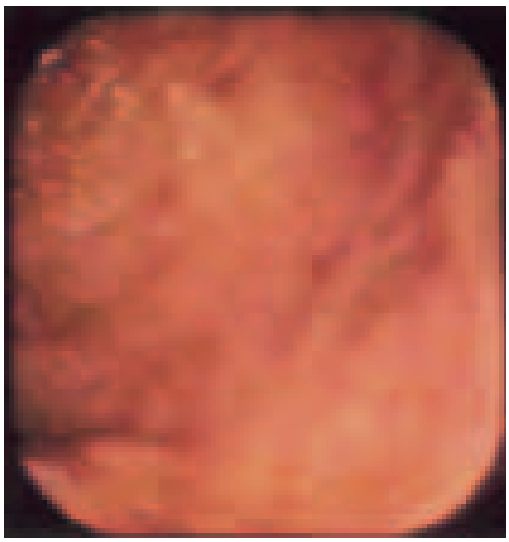

Fig. 2 : Linear configuration of erythematous spots

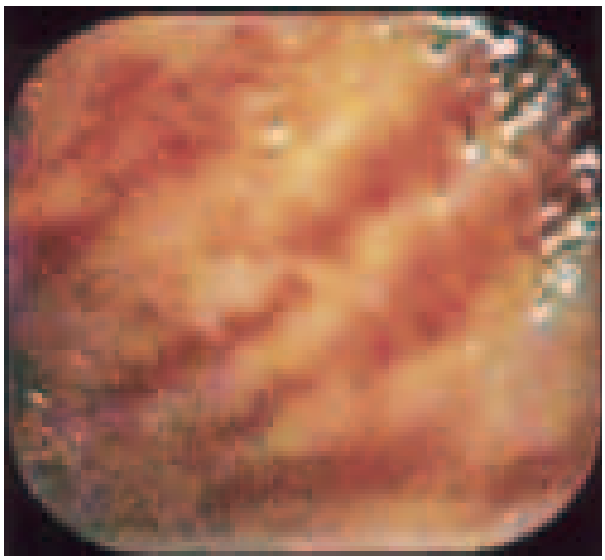

Fig. 3 : Watermelon stomach

Table 1 : Hematological parameters and upper GI endoscopy findings of both the patients

No.	Year	Hb gm/dl	MCV fl	Fe mg	TIBC	% sat	Indication for Upper GI scopy	Upper GI scopy findings
1.	1982	9.0	101	98	297	33	Pernicious anaemia	Atrophic gastritis
	1999	10.5	92	23	430	5	Iron deficiency anemia, stool OB+	Multiple red spots in antrum
	2001	13.0	91	50	342	15	Follow up, stool OB+	Few linear red stripes
	2002	8.8	93	33	408	8	Iron deficiency anaemia, stool OB+	Watermelon
2.	1999	4.7	91	20	454	6	Iron deficiency, stool OB+	Multiple red spots in antrum
	2001	6.0	92	53	387	12	Persistent iron deficiency anaemia	Watermelon
	2002	10.0	89	78	280	35	Follow up endoscopy	Watermelon

Case Reports

Case 1

A 69 years male presented 20 years ago, with pallor and weakness of six months duration. He was investigated as a case of anaemia and diagnosed to have pernicious anaemia on basis of megaloblastic type of anaemia, positive anti-parietal cell antibodies, positive intrinsic factor antibodies, low vitamin B12 levels, achlorhydria and atrophic gastritis on upper GI scopy. He was put on lifelong parenteral vitamin B12 supplementation and was doing well (hemoglobin ~ 12 gm/dl) till last 4 years. Four years back, his Hb dropped to 10 gm/dl and was found to have iron deficiency anaemia with positive occult blood test in stools. Colonoscopy and small bowel barium series were normal. His upper GI scopy at that time (1999) revealed atrophic gastritis with presence of multiple small red spots in whole of the antrum (Table 1, Fig. 1). Follow up upper GI scopy in 2001 revealed linearly arranged multiple red sports at few sites in antrum and atrophic gastritis (Table 1, Fig. 2). Upper GI scopy in 2002 revealed classical watermelon appearance in antrum in addition to gastric atrophy (Table 1, Fig. 3). Patient was put on iron supplementation in addition to vitamin B12 therapy and is doing well.

Case 2

A 71 years female was investigated for iron deficiency anaemia of four years duration, for which she required continuous iron supplementation and multiple blood transfusions. She had two hospital admissions for complaints of easy fatigability, weakness and dyspnoea on exertion (in 1999 and 2001). Clinical examination revealed pallor. She had iron deficiency anaemia with normal liver and renal function tests. Cardiac work up, including ECG, chest X-ray and 2 D-echocardiography was normal. Her stool occult blood test was positive. Colonoscopy and small bowel barium series were normal. Upper GI scopy in 1999 revealed multiple tiny red spots in whole of the antrum (Table 1). Imaging studies revealed (USG and CT scan-abdomen) presence of spontaneous lienorenal shunt, dilated tortuous collaterals at splenic hilum, mild splenomegaly and normal liver. Again in 2001, her upper GI scopy revealed classical watermelon appearance in antrum without evidence of varices or portal hypertensive gastropathy (Table 1). Her hepatic and portal venogram revealed presence of portal hypertension, large lienorenal shunt and small portoazygous shunts. All the tests for viral and autoimmune etiology, lipid profile and glucose tolerance test were normal. She refused to undergo liver biopsy. She was put on Tranexamic acid 500 mg tablets three times a day and is maintaining hemoglobin ~ 10 gm/dl (2002) and is symptom-free for last one year (Table 1).

Discussion

Gastric antral vascular ectasia syndrome is a rare entity. It is present in 3% of cirrhotics, whereas 30-40% of GAVE patients have portal hypertension. Classic noncirrhotic patient is middle-aged female with autoimmune disease or atrophic gastritis.[1] Its association with scleroderma, pernicious anaemia, chronic renal failure, aortic stenosis, gastric carcinoma, bone marrow transplantation, Addison's disease and antral mucosal prolapse is reported in the world literature.[2-4]

It is characterized by chronic gastrointestinal blood loss, iron deficiency anaemia and transfusion dependency. Very rarely, it may present as acute bleeding. Endoscopic appearances are classically described as watermelon stomach-prominent longitudinal red stripes on antral mucosa radiating out from pylorus. Each stripe represents multiple flat red mucosal lesions of 1 to 5 mm size, lying in close proximity.[1]

Differentiation from portal hypertensive gastropathy is by 1) antrum as predominant site, 2) lack of snake-skin-like appearance, 3) dilated capillaries with thrombi, fibrohylinosis and spindle cell proliferation in biopsy, 4) no relation to degree of portal hypertension and 5) no response to beta-blockers and portosystemic shunts. Pathogenesis of GAVE is unclear - mucosal atrophy, high gastrin levels, hypertrophy of neuroendocrine cells in antrum (5-HT and VIP), mechanical stress and portal hypertension are implicated.[2]

Treatment of GAVE is difficult. Medical treatments like steroids, estrogen-progesterone combination and tranexamic acid are tried with inconsistent results. Endoscopic treatment with laser photocoagulation (Nd-Yag or APC) or heat probe diathermy is increasingly reported to be successful and can delay surgical antrectomy, which is definite curative treatment.[3,5,6]

None of the reports reviewed gradual development of GAVE. In this report, we describe natural course of development of watermelon stomach. It starts as localized erythematous spots and then, develops into linear lesions and finally, into classic watermelon stomach.

Acknowledgements

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Book Review

A Hand Book on Diabetes Mellitus

The second edition of "Hand Book on Diabetes Mellitus by Dr. V Seshiah (Chief Editor)" is a simple, practical, clinical update on a most important contemporary challenging disease. Published by All India Publishers and Distributors, Chennai, New Delhi. This book contains 22 chapters and has updated information on almost every aspect of diabetes. One of the major strengths of the book is its colour charts, tables, flow diagrams and simple language. In about 300 pages all the vital information has been presented. The basic chapters on Insulin secretion and action, is a concise presentation of insulin biosynthesis, secretion and action. The classification and diagnosis of diabetes has a very important table on differentiating points between type 1 diabetes, MODY and type 2 diabetes in children. The etio-pathogenesis of diabetes has very well defined illustrations and flow charts. Similarly the chapter on medical nutrition therapy is replete with very important information on glycemic index cholesterol content, fatty acid compositions and n6/n3 values of oils. This information is vital and often required by the physicians. The chapter on OHA and insulin are two excellent chapters as also the one on diabetes and pregnancy. The most important feature of these chapters is the unique style of presentation of clinical information in a very practical manner. The emergencies in diabetes namely the hyperglycemic and the hypoglycemic acute emergencies are very well written. The biochemical mechanisms, pathophysiology and the correlation of metabolic abnormalities with clinical features in these chapters provide easy understanding. The micro and macrovascular complications and the chapter on pathogenesis of complications contain all the important information and provide very useful tips of management and referral. The associated conditions such as infections and surgery in a diabetic and lipids and hypertension covers the important issues related to associated conditions. In addition diabetes foot and sexual functions have been also covered extremely well.

May be separate chapters on diabetes in the elderly, type 2 diabetes mellitus in children and one on prevention of diabetes will find place in the next edition.

However these points are covered elsewhere in the book.

On the whole this is a very useful, simple and practical companion for persons caring for diabetes.

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