Intra-Arterial Embolization as a Treatment Option in a Case of GAVE Syndrome

Soumil Singhal1, Mangerira C Uthappa1

1Department of Intervention Radiology and Intervention Oncology, BGS Gleneagles Global Hospital Bangalore, Bangalore, Karnataka, India

Address for correspondence Soumil Singhal, MD, Department of Intervention Radiology and Intervention Oncology, BGS Gleneagles Global Hospital, 67, Uttarahalli Main Road, Sunkalpalya, Kengeri, Bangalore 560060, India (e-mail: drsoumilsinghal75@gmail.com).

J Clin Interv Radiol ISVIR

Abstract

Gastric antral vascular ectasia (GAVE) syndrome is a rare condition affecting the stomach. It is associated with upper gastrointestinal bleeding. The exact pathogenesis of the condition is poorly understood. Watermelon stomach is synonymous with GAVE syndrome due to its endoscopic appearance. Here we report a unique experience of performing an intra-arterial embolization in a case of GAVE presenting with uncontrolled bleeding.

Introduction

Gastric antral vascular ectasia syndrome or GAVE syndrome is a rare condition of the stomach, which is associated with severe acute or chronic upper gastrointestinal bleed. The exact pathogenesis of the condition is poorly understood; however, the condition has been linked to various medical conditions.

This condition was first described by Rider et al in 1953.1 Wheeler et al2 reported its characteristic endoscopic features. Based on the distinctive endoscopic features, the term watermelon stomach was coined by Jabbari et al.3 Watermelon stomach endoscopically resembles a pattern similar to the watermelon's exterior, the prominent erythematous stripe-like pattern radiates from the pylorus to the antrum resembling the similar design. GAVE syndrome is often misdiagnosed as either antral gastritis or portal hypertensive gastropathy.4 Treatment of the condition can be challenging and mainly includes three options including: (1) medical management, (2) endoscopic management, and (3) surgical management. Scanty case reports of the use of various pharmacologic therapies in the management of the condition have been reported. Surgical resection is the definitive option available, but it comes at the cost of significant morbidity and mortality rates.4 Endoscopic management is the main conservative treatment option available.5 Complete eradication of the condition requires two to four sessions based on the site, extent, and number of lesions. Up to 40% of postprocedure recurrence has been reported in some studies.6 We report a unique experience of performing an intra-arterial embolization in a case of GAVE presenting with uncontrolled bleeding even after multiple rounds of endoscopic management.

Case Report

A 63-year-old male patient, who is a known case of chronic liver disease (etiology: cryptogenic) since 2011, presented to the emergency department with complaints of two episodes of hematemesis and melena. The patient had several such incidents in the past for which esophageal variceal ligation and argon plasma coagulation (APC) was performed on several occasions.

On examination, patient's vitals were stable with evidence of pallor, pedal edema, and ascites. The patient had a Child–Pugh score B with a MELD (Model for End-Stage Liver Disease) score of 15. Supportive measures were taken, and an emergency upper gastrointestinal endoscopy was performed considering bleeding varices as the cause for the patient's symptoms. Endoscopy showed eradicated esophageal varices, mild portal gastropathy in fundus and body, nodular erythematous streaks in the antrum extending to the proximal body, and lesion in the prepyloric region and the antrum with active ooze. Duodenum showed features of erythematous duodenopathy with no active ooze. A diagnosis of GAVE syndrome was made. Forced argon plasma coagulation (APC) was performed (40 W, 2 L/min) to settle down the active ooze. The patient was symptomatically normal for 24 hours following which he redeveloped melena with a drop...
in hemoglobin concentration (drop from 9 to 8.3 g%). Due to the sudden drop, again an emergency repeat endoscopy was performed, which showed blood at the former site of APC. A definite bleeding point could not be found due to blood in the field of view. APC was abandoned, and computed tomography (CT) was planned. A CT was performed after a multidisciplinary meeting between the hepatologist, hepatic surgeon, and the interventional radiology team.

Computed tomography abdominal angiography was performed (16 slice, GE Healthcare). The scan was performed in the supine position in suspended respiration. The contrast was injected using a pressure injector through an 18 gauge cannula sited in the left upper limb vein. Omnipaque (Iohexol 350, General Electric Healthcare) was used as intravenous contrast media. Patient underwent two angiographic phases with the first phase acquisition taken at 6 seconds and the second phase at 45 seconds following a bolus trigger. Technical parameters were set as pitch (1.2), collimation (arterial: 16 × 0.6 mm and enous: 24 ×1.2 mm), rotational time (0.6 s), kV (120), and mA (120). CT angiography showed the patient to have features of chronic liver disease with features of portal hypertension and ascites. There was a focal mucosal thickening seen at the antral region of the stomach with multiple prominent vessels supplying this area with mildly increased vascularity best appreciated on the arterial phase (Figs. 1, 2). No active contrast extravasation was
demonstrated on any phase of the CT angiography. Due to
recurrent fall in patients hemoglobin concentration, estab-
lished the diagnosis of GAVE syndrome and failed APC, a
unique and alternative approach was planned for the man-
agement of the condition. An empirical intra-arterial emboli-
zation was proposed based on the CT angiographic finding of
multiple arterial feeders to the thickened antral region.

A super selective empirical intra-arterial embolization
was planned and performed under mild sedation. Right fem-
oral arterial access was achieved under ultrasound guidance,
and a 5F sheath was inserted. Celiac artery and common
hepatic artery were cannulated using a 5F cobra catheter,
and angiographic run was acquired. A normal celiac artery
anatomy was noted with the common hepatic artery diving
into hepatic artery proper and gastroduodenal artery. Super
selectively catheterization of the gastroduodenal artery
was then performed using a 2.7F microcatheter (Progreat,
Terumo). An angiographic phase was acquired, which
demonstrated multiple small vessels supplying the area of
focus in the antral region with these small gastric feeding
vessels arising mainly from the right gastroepiploic artery
with early contrast runoff (Fig. 3). Empirical embolization
was performed using gel foam slurry, which was made by
mixing small gelatin sponge pledgets with contrast using
the Tessari technique. Gel foam slurry was first injected
super-selectively into these multiple small feeding vessels.
Check angiography showed stasis of flow across these small
feeding vessels. Following this, coil embolization was per-
formed using three Nester micro-coils (2–70 × 4 mm and
1–140 × 4 mm), which was placed in the right gastroepi-
plioic artery and the superior pancreaticoduodenal artery,
respectively (Fig. 4). Cross circulation from the superior
mesenteric artery was blocked using gel foam slurry injec-
tion into the inferior pancreaticoduodenal artery, and this
was confirmed on the successive angiographic run. Other
sources of cross circulation were checked by confirming
angiographic series from the proper hepatic artery, gastric
artery, splenic artery, and colic arteries. The patient was
hemodynamically stable postprocedure with no further drop
in hemoglobin concentration or any complaints of melena.
The patient was shifted to ICU on day 5 due to develop-
ment of oliguria, increase lactate, and breathlessness. The patient
developed multi-organ failure and bacterial peritonitis by
day 7 and eventually succumbed due to cardiopulmonary
arrest. No follow-up imaging or endoscopy was performed
due to deterioration in the patients’ condition.

Discussion

GAVE syndrome or watermelon stomach is a rare condition
of the stomach, which can be misinterpreted as either antral
gastritis or portal hypertensive gastropathy. The condition
is common in elderly patients with a female preponderance.
The mean age at presentation is about 73 years in women
and 68 in men, with up to 71% of patients being women.
Ninety percent of patients present with nonresponsive iron
deficiency anemia and occult bleed. The exact patho-
genesis is not well understood; however, the condition has
shown a link to various conditions like liver cirrhosis, dia-
betes, end-stage renal disease, hypothyroidism, connective
tissue disorder, and cardiac disease. Cirrhosis is one of
the most common causes of the condition, with up to 30%
of patients having preexisting cirrhosis. Patients with
GAVE present with chronic iron-deficiency anemia, acute
GI bleed, unexplainable abdominal pain, and rarely as gas-
tric outlet obstruction. Up to 60% of patients with connec-
tive tissue disorder have skin telangiectasias. Diagnosis
of the condition can be using endoscopy, enteroscopy, and
sometimes using red blood cell scans or capsule endoscopy.
GAVE syndrome, classically present as a red lesion which is
often hemorrhagic, which can be either in a linear fashion or
diffusely spread out. These are predominantly located within
the gastric antrum. Histologically, the underlying region
shows hyperplastic antral mucosa, thrombosed dilated capil-
ary channels, and hypertrophy of the fibromuscular lamina
propria without inflammatory signs. The histological find-
ings are also appreciated on endoscopic ultrasound. Some
studies have found a 5-year latency period for the diagnosis
of the condition. Due to its features, GAVE can be misin-
terpreted as portal hypertensive gastropathy; however,
there are specific features which help in differentiating the
conditions. Portal hypertensive gastropathy involves the cor-
pus and the fundal region of the stomach, whereas, GAVE is
most limited mainly to the antral region. GAVE has a classi-
cal endoscopic finding. Procedures such as β-blocker therapy
or transjugular intrahepatic shunt are not helpful in case of
GAVE syndrome.

Treatment of the condition can be challenging and mainly
includes three options including, (1) medical management,
transcytoplasmic diffusion by a direct exposure of the
vascular endothelium to the tumor cells. In this study, we
demonstrated that vascular endothelial growth factor,
a cytokine associated with angiogenesis, was expressed
in normal intestinal polyps and in polyps of patients
with Gardner syndrome. Although our study was not
designed to determine if vascular endothelial growth
growth factor is involved in angiogenesis in intestinal
polyps, these findings suggest that vascular endothelial
growth factor may play a role in the growth of normal
intestinal polyps and in polyps of patients with Gardner
syndrome.

Techniques of experimentally induced angiogenesis
in rat intestine

We evaluated the effect of phosphodiesterase inhibitors
in the rat intestine. In the control experiment, intestinal
segments were exposed to a vehicle alone. In one
experiment, the segments were exposed to a vehicle
alone and then to an inhibitor of phosphodiesterase
for 15 minutes. A control experiment was performed in
each case.

In two experiments, the intestinal segments were
exposed to an inhibitor of phosphodiesterase for 15
minutes. The inhibitors used were dipyridylate or
dipyridamole. The segments were then exposed to a
vehicle alone and then to either an inhibitor of
phosphodiesterase or to a vehicle alone. The inhibitors
used were dipyridylate or dipyridamole. The segments
were then exposed to a vehicle alone and then to either
an inhibitor of phosphodiesterase or to a vehicle alone.

In one experiment, the intestinal segments were
exposed to a vehicle alone and then to an inhibitor of
phosphodiesterase for 15 minutes. The inhibitors used
were dipyridylate or dipyridamole. The segments were
then exposed to a vehicle alone and then to either an
inhibitor of phosphodiesterase or to a vehicle alone.

In one experiment, the intestinal segments were
exposed to an inhibitor of phosphodiesterase for 15
minutes. The inhibitors used were dipyridylate or
dipyridamole. The segments were then exposed to a
vehicle alone and then to either an inhibitor of
phosphodiesterase or to a vehicle alone.

In one experiment, the intestinal segments were
exposed to an inhibitor of phosphodiesterase for 15
minutes. The inhibitors used were dipyridylate or
dipyridamole. The segments were then exposed to a
vehicle alone and then to either an inhibitor of
phosphodiesterase or to a vehicle alone.

In this experiment, the intestinal segments were
exposed to an inhibitor of phosphodiesterase for 15
minutes. The inhibitors used were dipyridylate or