Superior Mesenteric Artery Syndrome Following Laparoscopic Gastric Banding Procedure

Mary B. White, MD and Kelli Horton, RD

While gastric banding typically is associated with more gradual weight loss and fewer complications than other types of bariatric surgery, this case demonstrates the importance of maintaining vigilance for troublesome signs.

Superior mesenteric artery (SMA) syndrome—also called Wilkie's syndrome or cast syndrome—is a relatively rare disorder involving compression of the duodenum. Under normal conditions, the third part of the duodenum passes unhindered through a 10- to 20-mm space created by the SMA neurovascular bundle and the abdominal aorta, situated at an angle of 25° to 60°. SMA syndrome occurs when narrowing of the aortomesenteric space to less than 8 mm or reduction of the aortomesenteric angle to less than 25° results in compression of the duodenum by the SMA anteriorly and the aorta posteriorly (Figure 1).[1,2]

SMA syndrome can present as a chronic, intermittent, acute, partial, or complete duodenal obstruction.[3] If not diagnosed expeditiously, it can result in severe electrolyte disturbances (hypokalemia), malnutrition, dehydration, oliguria with acute renal failure, gastric rupture, and death.[1,3,4]

In this article, we report the case of a patient who developed SMA syndrome three years after undergoing gastric banding for morbid obesity. In this case, subsequent cosmetic surgery and complications thereof appear to have contributed to the conditions that produced the syndrome and its manifestations. Following the case presentation, we discuss the etiology, symptoms, diagnosis, and treatment of SMA syndrome.

INITIAL EXAM

In August 2007, a 42-year-old woman presented to the emergency department (ED) with intractable nausea and vomiting, abdominal pain, and abdominal distension. Her medical history included fibromyalgia, morbid obesity, depression, irritable bowel syndrome, migraines, and sleep apnea.

The patient had undergone a laparoscopic gastric banding procedure for her morbid obesity in February 2004, at which time her weight was 209% of her ideal body weight (IBW). Over the two years following this procedure, she had lost 100 lb at a steady rate. This weight loss was accompanied by cutis laxa, which was treated with abdominoplasty followed by a thigh and buttock lift in February 2006. The buttock lift was complicated by a surgical wound infection with *Enterobacter* species and methicillin-resistant *Staphylococcus aureus*, as well as by the concomitant development of severe bouts of abdominal pain and postprandial, nonbloody vomiting of undigested food up to 10 times per day. As a result, the patient was hospitalized and experienced further weight loss of about 25 lb over a two-month period.

The patient's pain was intermittent, sharp, and located in the epigastrium and the right upper quadrant of her abdomen. It lasted for hours, occasionally radiated to her back, and worsened with meals. She rated the severity of her pain, at its worst, as 10 on a scale of 1 to 10. The pain and vomiting persisted despite treatment for reflux and for possible *Helicobacter pylori* infection, use of gastric motility agents, and adjustment of oral intake to smaller and more frequent meals. Overall, the patient lost a total of 140 lb—57% of her previ-

Continued on page 29

Dr. White is the transitional care unit director in the department of medicine at the West Los Angeles VA Medical Center (WLA VAMC), Los Angeles, CA and an associate professor of medicine at the David Geffen School of Medicine at the University of California, Los Angeles. **Ms. Horton** is a nutrition support dietitian and the nutrition and food service clinical section chief at the WLA VAMC.

Continued from page 26

ous weight—between February 2004 and August 2007. During this period, her body mass index dropped from 45 to 19.

Upon initial examination in the ED, the patient's vital signs were significant for tachycardia (heart rate, 108 beats/min) and hypertension (blood pressure, 159/120 mm Hg). She had dry mucous membranes, and an abdominal examination indicated minimal bowel sounds and tenderness in all four quadrants. Significant results of laboratory studies were a low serum potassium level of 3.07 mmol/L (reference range, 3.5 to 5.3 mmol/L), a normal blood urea nitrogen concentration of 18 mg/dL (reference range, 5 to 25 mg/dL), a high normal serum creatinine level of 1.3 mg/dL (reference range, 0.5 to 1.4 mg/dL), normal serum hemoglobin and hematocrit values, and an elevated white blood cell count of 22,000 cells/μL (reference range, 4,500 to 11,000 cells/μL).

An abdominal computerized tomography (CT) scan revealed the patient's stomach to be massively distended with fluid to a size of 30 cm x 20 cm x 20 cm (a normal, empty stomach is approximately 7 cm x 4 cm x 4 cm) (Figure 2). The adjustable gastric band was still in place. The distance between the SMA and the aorta was only 4 mm, and sagittal reconstructed CT images showed an aortomesenteric angle of approximately 10° (Figure 3). There was marked dilatation of the transverse duodenum to the right of the aorta. The small bowel distal to the transverse duodenum was decompressed.

TREATMENT COURSE

In the ED, the patient was given fluid resuscitation with normal saline 2L IV and an IV infusion of potassium. Attempts at placing a nasogastric (NG) tube to drain and decompress her stomach were unsuccessful.

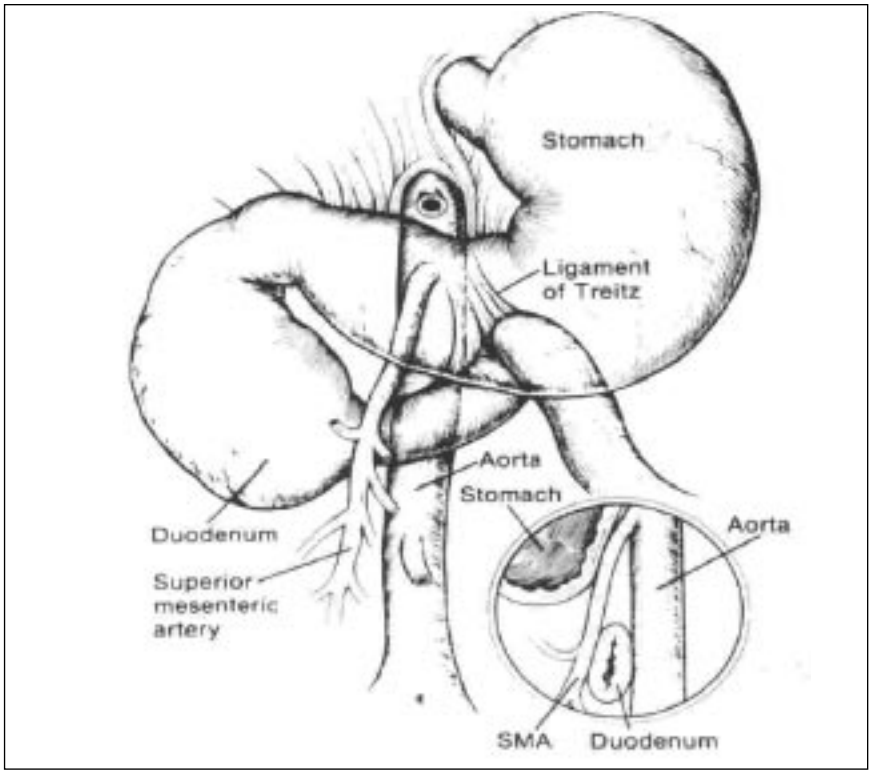

Figure 1. Diagram showing the anatomic relationship between the duodenum, aorta, and superior mesenteric artery (SMA) in SMA syndrome. The lateral view demonstrates compression of the duodenum between the vascular structures. Figure reprinted with permission from: Cohen LB, Field SP, Sachar DB. The superior mesenteric artery syndrome. The disease that isn't, or is it? *J Clin Gastroenterol*. 1985;7(2):113–116.[2] Copyright © 1985 Lippincott Williams & Wilkins.

After urgent gastroenterology and general surgical consultations, the patient was taken to the operating room and given general anesthesia for airway protection. She underwent NG tube placement, which resulted in the drainage of approximately 5 L of fluid from her stomach. An emergent esophagogastroduodenoscopy (EGD) showed an edematous, hemorrhagic, and friable gastric mucosa, primarily along the greater curvature, with some sparing of the lesser curvature (Figure 4). There was no evidence of ulceration or perforation. At this point, the decision was made to try conservative treatment—further fluid resuscitation and monitoring—before considering surgical intervention.

Parenteral nutrition was initiated through a dedicated port of a peripherally inserted central catheter (PICC) and kept at a maintenance rate due to alterations in liver enzymes with higher rates of dextrose. After the approximately three weeks it took the patient's gastric mucosa to heal, a nasogastrojejunal (NGJ) feeding tube was placed under endoscopic guidance to allow continued gastric decompression and jejunal nutrition. Parenteral nutrition was discontinued after jejunal feeds of a semielemental nutritional formula were tolerated, providing 44 kcal/kg/day.

Continued on next page

Continued from previous page

The patient's weight increased rapidly once additional caloric intake and adequate fluid replacement were achieved. She gained 21 lb within three weeks of initiating enteral nutrition. At that time, her gastric output through the NGJ tube suddenly decreased from a daily average of 1,800 mL/day to less than 100 mL/day. The NGJ tube was removed and a clear liquid diet was initiated. The patient's diet was advanced slowly with her continued tolerance of solid foods. A repeat abdominal CT scan showed an appropriately small gastric pouch with no evidence of bowel dilation to support obstruction. She was discharged after a total hospital stay of two and a half months, with her gastric band still intact.

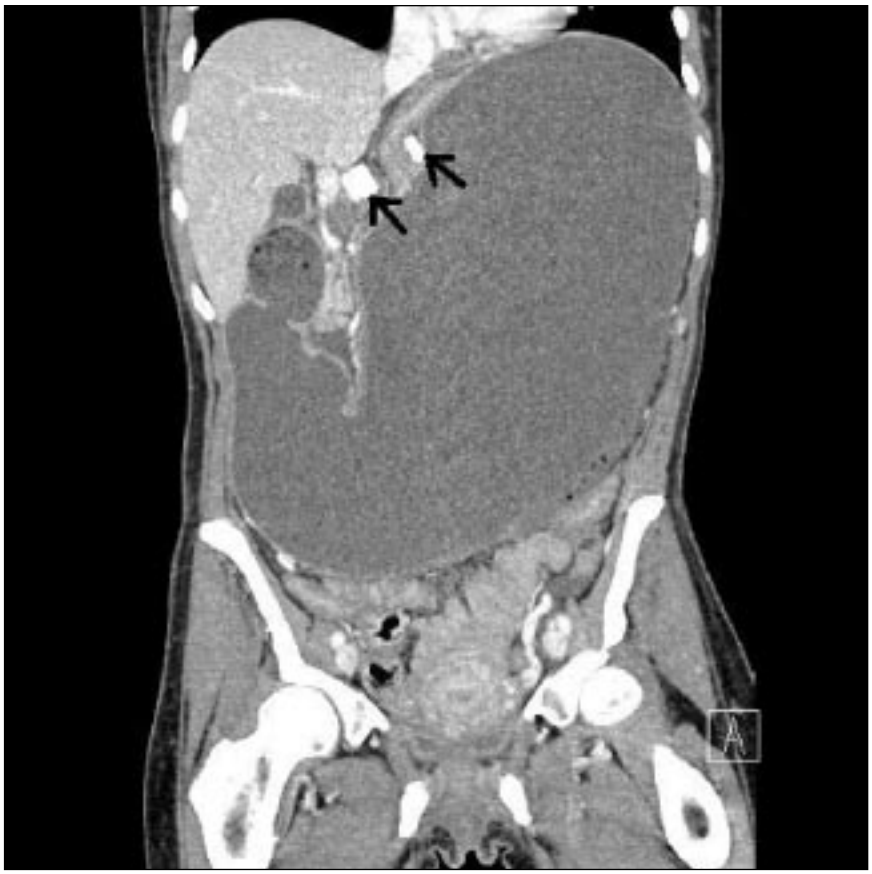

Figure 2. Coronal computerized tomography scan of the patient's abdomen, showing a massively distended stomach with gastric band in place at the upper aspect of the lesser curvature (arrows).

ABOUT THE CONDITION

Rapid, dramatic weight loss—as is often seen with bariatric surgery, catabolic conditions, anorexia nervosa, and bulimia nervosa—is a major cause of SMA syndrome. Such weight loss can deplete mesenteric fat stores, which help to maintain the normal aortomesenteric angle and distance. Other causes of SMA syndrome include spinal elongation surgery, such as for scoliosis; exaggerated lumbar lordosis; rapid linear growth without weight gain; and anatomic abnormalities, including high insertion of the duodenum at the ligament of Treitz and an SMA originating in a lower position off the abdominal aorta.[3,5,6]

Predisposing factors for SMA syndrome include female sex, thin body habitus, and abdominal wall laxity.[3,6] As a thin woman who had experienced dramatic weight loss following laparoscopic gastric banding, which resulted in abdominal wall laxity, our patient clearly fit this profile.

Although there have been reported cases of SMA syndrome associated with rapid weight loss after laparoscopic Roux-en-Y gastric bypass surgery for morbid obesity,[7] our patient's case appears to be the first in which massive gastric distension and SMA syndrome followed a gastric banding procedure. Often, the laparoscopic gastric banding is preferred over either laparoscopic or open Roux-en-Y because it produces more gradual weight loss and is simpler, with fewer complications and the lowest mortality rate of all bariatric procedures. Furthermore, it offers the advantages of adjustability and reversibility.[8]

Over the two years after her gastric banding procedure, our patient lost 100 lb—a gradual loss that is unlikely to have resulted, by itself, in SMA syndrome. It is notable that her symptoms did not begin until after the February 2006 procedures, used to treat cutis laxa, from which infections complications caused her to lose an additional 25 lb (16% of her body weight) over a two-month period. We hypothesize that this additional, severe weight loss was sufficient to deplete her mesenteric fat stores and result in SMA syndrome.

We further speculate that the patient's extreme gastric dilatation may have prevented her from being able to expel the accumulated fluid through vomiting. With massive gastric distension, the intragastric pressure may exceed the gastric venous pressure,

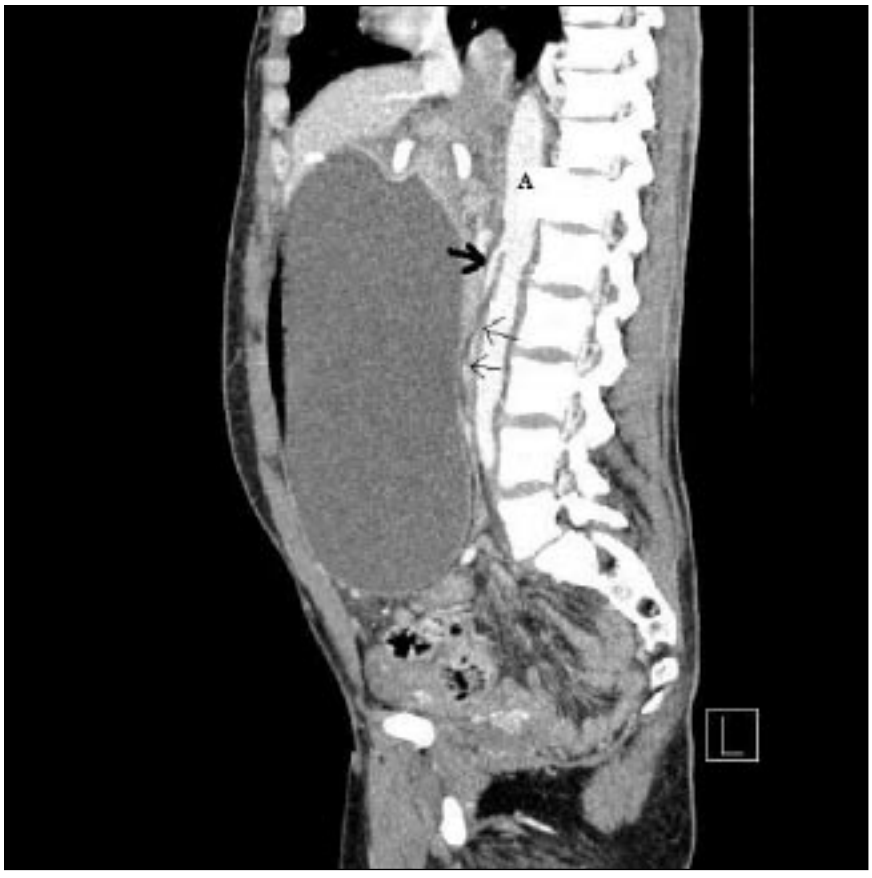

Figure 3. Sagittal computerized tomography scan of the patient's abdomen, showing the narrowed aortomesenteric angle and distance (bold arrow) and compressed portion of the duodenum (fine arrows). (The "A" indicates the aorta.)

resulting in gastric atony, ischemia, necrosis, and possible perforation.[9] Moreover, obstruction of the gastroesophageal junction by the distended gastric fundus, which angulates the esophagus against the fibers of the right diaphragm, may produce a one-way valve that inhibits vomiting of the stomach contents.[10]

Based on the patient's history of episodic abdominal pain and recurrent vomiting of undigested food, it is likely that she experienced intermittent obstruction of the duodenum for several months leading up to her acute presentation. The patient was hypokalemic and dehydrated on presentation, and the abdominal CT scan showed narrowing of both the aortomesenteric angle and the aortomesenteric space.

Diagnosis

Clinicians should consider the diagnosis of SMA syndrome for any patient who presents with nausea, postprandial or bilious vomiting, epigastric pain, abdominal distension, and weight loss.[11,12] Gastric dilatation is seen in 72% of cases with acute presentations, compared with 11% of chronic cases. Symptoms are exacerbated in the supine position but often are alleviated by prone, knee-to-chest, or left lateral decubitus positions,[3,4] which increase the aortomesenteric angle and distance and reduce the duodenal compression.[5] On examination, 80% of patients with SMA syndrome are thin. Patients also may have a distended abdomen, high pitched bowel sounds, and succussion splash.[3,4]

The diagnostic criteria for SMA syndrome are a dilated duodenum, an aortomesenteric angle of less than 25°, and compression of the third part of the duodenum by the SMA. Diagnosis of SMA syndrome can be made by upper gastrointestinal series in which barium is observed in the dilated first and second portions of the duodenum, with an abrupt linear cutoff in the transverse portion. Fluoroscopy may demonstrate an antiperistaltic flow, producing a to-and-fro motion of barium in the dilated duodenum proximal to the obstruction; delayed transit through the gastroduodenal region of about four to six hours; and relief of the obstruction in the left lateral decubitus position.[3,6] Abdominal CT scanning can demonstrate a dilated duodenum and is useful for measuring the aortomesenteric angle and distance on sagittally reconstructed images,[3,11] as well as assessing intraperitoneal fat stores.[4] Frequently, EGD is performed to assess the gastric mucosa and rule out other causes of obstruction, such as tumors, webs, and peptic ulcer disease.[10]

Treatment

Treatment involves fluid resuscitation, correction of electrolyte abnormalities, decompression of the stomach and duodenum with an NG tube, proper positioning of the patient after eating, and identification and treatment of the underlying cause of SMA.[3,8] A conservative approach was chosen for our patient, with placement of a double lumen NGJ tube for feeding and continued decompression. If the patient cannot tolerate

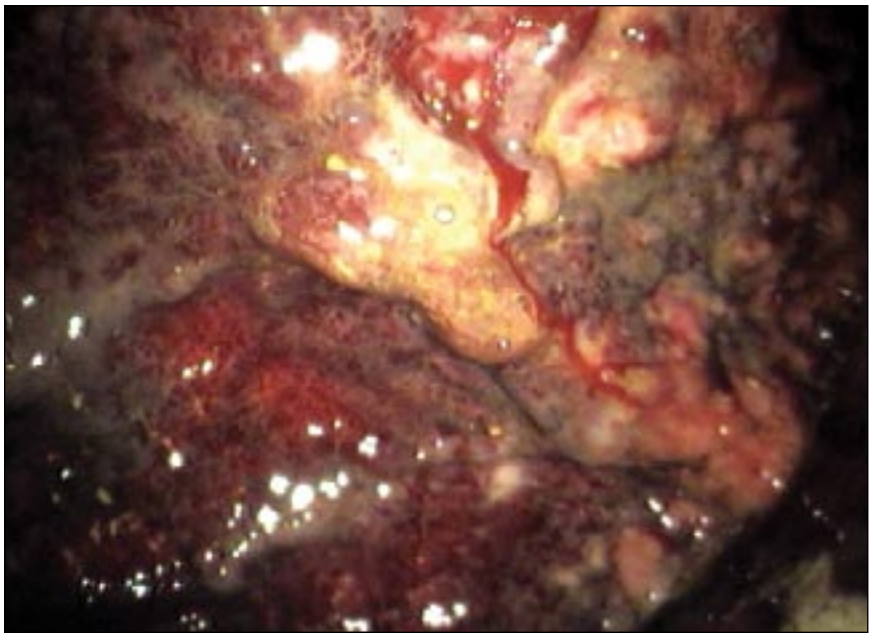

Figure 4. The patient's edematous and hemorrhagic gastric mucosa.

enteral feeding through an NGJ tube (for example, if the gastric mucosa has been damaged, as in the case of our patient), parenteral nutrition should be given. Nutritional support is essential for restoring a positive nitrogen balance and increasing mesenteric fat stores, thereby increasing the aortomesenteric angle and relieving duodenal compression.[7]

If conservative strategies for managing SMA syndrome are unsuccessful, surgical options to consider include laparoscopic or open duodenojejunostomy and laparoscopic severing of the ligament of Treitz and mobilization of the duodenum.[3,11,13]

SUMMING UP

SMA syndrome is a rare but serious disorder requiring timely diagnosis and treatment. With the soaring rate of obesity in our society and increasing frequency of bariatric surgery, clinicians might expect to see an increase in the number of cases of SMA syndrome.

Author disclosures

The authors report no actual or potential conflicts of interest with regard to this article.

Disclaimer

The opinions expressed herein are those of the authors and do not necessarily reflect those of Federal Practitioner, Quadrant HealthCom Inc., the U.S. government, or any of its agencies. This article may discuss unlabeled or investigational use of certain drugs. Please review complete prescribing information for specific drugs or drug combinations—including indications, contraindications, warnings, and adverse effects—before administering pharmacologic therapy to patients.

REFERENCES

1. Adson DE, Mitchell JE, Trenkner SW. The superior mesenteric artery syndrome and acute gastric dilatation and eating disorders: A report of two cases and a review of the literature. *Int J Eat Disord.* 1997;21(2):103–114.
2. Cohen LB, Field SP, Sachar DB. The superior mesenteric artery syndrome. The disease that isn't, or is it? *J Clin Gastroenterol.* 1985;7(2):113–116.
3. Shetty A, Hill ID. Superior mesenteric artery syndrome. eMedicine web site. http://www.emedicine.com/ped/TOPIC2175.HTM. Updated July 16, 2006. Accessed August 5, 2008.
4. Scovell S, Hamdan A. Superior mesenteric artery syndrome. UpToDate Online. Version 15.2. http://uptodateonline.com/utd/content/topic.do?topicKey=gi_dis31570&selectedTitle=1~44&source=search_result. Updated September 11, 2006. Accessed October 24, 2007.
5. Kingham TP, Shen R, Ren C. Laparoscopic treatment of superior mesenteric artery syndrome. *JSLS.* 2004;8(4):376–379.
6. Plesa A, Constantinescu C, Crumpei F, Cotea E. Superior mesenteric artery syndrome: An unusual cause of intestinal obstruction. *J Gastrointestin Liver Dis.* 2006;15(1):69–72.
7. Schroeppel TJ, Cholcote WS, Lara MD, Kothari SN. Superior mesenteric artery syndrome after laparoscopic Roux-en-Y gastric bypass. *Surgery.* 2005;137(3):383–385.
8. Mun EC, Tavakkolizadeh A. Surgical management of severe obesity. UpToDate Online. Version 15.3. http://uptodateonline.com/utd/content/topic.do?topicKey=obesity6916&selectedTitle=4~40&source=search_result. Updated September 17, 2007. Accessed October 26, 2007.
9. Lo DY, Yen JL, Jones MP. Massive gastric dilation and necrosis in anorexia nervosa: Cause or effect? *Nutr Clin Pract.* 2004;19(4):409–412.
10. Lunca S, Rikkers A, Stanescu A. Acute massive gastric dilatation: Severe ischemia and gastric necrosis without perforation. *Rom J Gastroenterol.* 2005;14(3):279–283.
11. Richardson WS, Surowiec WJ. Laparoscopic repair of superior mesenteric artery syndrome. *Am J Surg.* 2001;181(4):377–378.
12. Lippl F, Hannig C, Weiss W, Allescher H, Classen M, Kurjak M. Superior mesenteric artery syndrome: Diagnosis and treatment from the gastroenterologist's view. *J Gastroenterol.* 2002;37(8):640–643.
13. Kim IY, Cho NC, Kim DS, Rhoe BS. Laparoscopic duodenojejunostomy for management of superior mesenteric artery syndrome: Two case reports and a review of the literature. *Yonsei Med J.* 2003;44(3):526–529.