Letter from the Editor

Dear Reader:

We are proud to provide this publication, which has brought a sense of achievement to many generations of Albany Medical College Internal Medicine residents. Our residents can share their academic work and discoveries with other medical professionals by publishing it in the Albany Medical Review.

What we publish today as case reports and interesting findings, may become common knowledge in near future… What we view as a rare occurrence today, may help identify and explain complex concepts… Together, we can share our knowledge and learn from each other.

It is our honor to present the Albany Medical Review. We appreciate the guidance and support provided by Michelle Snavely, Kiat Mok, M.D. and Raymond P. Smith, M.D. in preparing this issue.

 Truly, 
Jaspreet Arora, M.D.

OSTEOPOROSIS AND VERTEBRAL FRACTURES IN ANKYLOSING SPONDYLITIS

Anupam Batra, MD, Resident, Internal Medicine, Albany Medical College

Case Presentation:
A 61 year old man recently diagnosed with ankylosing spondylitis presented with back pain that began after a fall one week prior. The low back pain was reported to be persistent, non-radiating, 7/10 in intensity, dull and ach- ing in nature, relieved by lying supine, and attenuated by Ibuprofen twice daily. The pain was worse in the morn- ing, but improved as the day progressed. He denied any history of psoriatic plaques, uveitis, inflammatory bowel disease, or any systemic inflammatory immune-mediated rheumatic disease. He also denied any significant peripheral joint pain or stiffness. His medical history was remark- able for hypertension, diabetes mellitus type II, hypercholesterolemia, and erectile dysfunction. There was no history of spondyloarthritides in his family. He lived with his wife at home, denied tobacco or illicit drug use, and consumed alcohol on social occasions.

On examination his vital signs were unremarkable. Cardi- opulmonary and abdominal examinations were also unre- markable. There was tenderness over the spinous process of T10. Loss of lumbar lordosis and hyperkyphosis was noted. No active synovitis of the peripheral joints was detected. No neurologic deficits were appreciated. Complete blood count and basic metabolic panel were normal. Erythrocyte sedimentation rate (ESR) was slightly elevat- ed and C-reactive protein (CRP) level was normal. HLA B27 was positive. Vitamin D insufficiency was noted. Radiograph of the spine showed severe kyphosis of the cervical and thoracic divisions, a T10 vertebral body fracture, and ‘bamboo’ appearance of the spine with fusion of the sacroiliac joints. A dual photon x-ray absorptiometry showed bone mineral density (BMD) T-scores of 2.1 of the lumbar spine, -3.3 at the right femoral neck, and -2.7 at the right femoral neck.

He was evaluated by the Neurosurgery service and his thoracic vertebral fracture was deemed inoperable. He was treated conservatively with Salsalate and advised to avoid vigorous activity and heavy lifting. He returned for a follow up visit and the primary care physician initiated a discussion regarding the role of bisphosphonates and vitamin D supplementation in ankylosing spondylitis in the setting of known vertebral fracture and underlying osteoporosis. He did not wish to start bisphosphonates due to their side effect profile but was agreeable to initiate vitamin D supplementation. In terms of analgesia and functional status, the patient wished to control his pain with Ibuprofen, use a TLSO brace, and perform a gentle daily exercise regimen.

Discussion:
In ankylosing spondylitis (AS), chronic inflammation primarily affects the axial skeleton leading to new bone forma- tion with resulting syndesmophyte formation as...
Osteoporosis and Vertebral Fractures in Ankylosing Spondylitis (CONTINUED)

well as ankylosis of the spine and sacroiliac joints (1). Changes in bone remodeling and structure contribute to the increased risk of vertebral fractures in AS (2). Osteoporosis has been documented more often in the spine than in the hip of patients with AS early in the disease course, whereas in patients with long-standing disease, osteoporosis was frequent in the hip (2, 3). Bone loss has been reported in the hip and is associated with vertebral fractures in AS patients (4, 5). In a recent report, the prevalence of vertebral fractures (grades 2-3) was found to be 29.6% using the World Health Organization (WHO) criteria for BMD at the total hip and 33.3% using the WHO criteria for osteoporosis (6). The odds ratio of clinical vertebral fractures was 7.7 in a retrospective population-based study (7) and 3.3 in a primary care-based nested case-control study (8). The risk of vertebral fractures is multifactorial and independent of and superimposed on other clinical risk factors (9). Vertebral fracture risk was found to be greater in men than in women and was associated with low BMD, disease activity, and syndesmophyte extent (6, 10). Vertebral fractures causing irreversible hyperpyrexia characterize advanced disease with extensive syndesmophyte formation (bamboo spine) (11, 12). The greatest difficulty in the diagnosis of vertebral fractures is that only one third of morphometric vertebral fractures come to clinical attention. Many such acute fractures are likely overlooked and attributed to an acute inflammatory flare of back pain characteristic of AS. Since conventional radiographs of the lumbar spine are not specific for vertebral fractures, additional imaging such as CT, MRI, and bone scintigraphy may improve diagnostic yield (2) (Figure 1).

BMD T-scores can be misleading. In the current case, the patient’s lumbar spine BMD T-score was 2.7, which is above the young adult normal range. This is an artificial increase and represents aberrant, dense, and brittle bone formation. T-scores at the femoral neck will often fall within the osteoporotic range and better correlate with overall morbidity from osteoporosis in AS.

In a cross-sectional study in AS patients with active disease, the bone formation marker osteocalcin (OC) Z-scores and the bone resorption marker serum C-telopeptides of type I collagen (CTX) were strong markers to detect bone loss in AS (1). Furthermore, it was noted that increased bone turnover played a significant role in the development of osteoporosis in AS patients (1). The authors reported that low vitamin D levels played a role in the development of AS-related osteoporosis. Aemento and coworkers reported that vitamin D is an endogenous modulator of the immune response, which may slow down the inflammatory process by suppressing active T cells and cell proliferation (13). Therefore, vitamin D supplementation in AS patients with vitamin D insufficiency may have an anti-inflammatory effect leading to decreased vertebral fracture risk.

Effective therapy should target inflammation, bone formation, and bone resorption. The General Practice Research Database notes that the use of NSAIDs is associated with a 30% decrease in the risk of clinical vertebral fractures, though there is still a lack of prospective studies to substantiate this claim (3,8). Data from several studies showed that bisphosphonates inhibit inflammation in AS (14). Furthermore, bisphosphonates may be used in the treatment of osteoporosis in high risk patients (15). TNF-inhibitors have shown to decrease osteitis, prevent bone loss, and decrease inflammatory markers such as CRP and IL-6 (16,17).

In patients with AS presenting with an acute flare of back pain, persistent inflammation, prolonged disease duration, hyperpyrexia, and femoral neck BMD T-scores within the osteoporotic range, it is prudent for the clinician to include vertebral fracture in the differential diagnosis.

Figure 1. Displaced vertebral body fracture at T10. The sagittal computed tomography scan above highlights the following features of the current case: a displaced posterior vertebral body fracture at T10; fusion of the anterior longitudinal ligament; severe kyphosis of the thoracic spine; and virtual fusion of the thoracic spine at all levels.

*Contributions from Prashant Kaushik, MD.


Left Ventricular Assist Device Percutaneous Driveline Infection Causing Klebsiella Bacteremia

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Case Presentation:

A 45 year old man presented with a four day history of fevers, chills, rigors and general malaise. The patient’s past medical history was significant for idiopathic cardiomyopathy. In 2009, the patient received a left ventricular assist device (LVAD). Twelve months postoperatively he developed a driveline infection secondary to Klebsiella pneumoniae and Proteus mirabilis and was treated with chronic suppressive therapy with ciprofloxacin. The patient was lost to follow-up until 2011, two months prior to the present admission, when he was treated for sepsis secondary to a driveline infection and a left anterior abdominal wall abscess. Klebsiella pneumoniae and alpha hemolytic streptococcus were isolated from the abdominal wall abscess, which was incised, drained and treated with chronic suppressive therapy with amoxicillin-clavulanate. Approximately one month later he presented with septic shock due to Klebsiella pneumoniae bacteremia, presumably from the chronic driveline infection. The patient was not a candidate for a heart transplant because of expected non-compliance with therapy.
Left Ventricular Assist Device Percutaneous Driveline Infection Causing Klebsiella Bacteremia (continued)

and he was treated conservatively with antibiotics alone. After two
weeks of treatment, given his lack of improvement and poor prognosis,
his death was inevitable. He succumbed to the infection shortly thereafter.

Discussion:
A left ventricular assist device (LVAD) is a mechanical pump used to
augment or supplant left ventricular function in patients suffering from
end-stage heart failure either as a bridge to transplant or, increasingly, as
an alternative to heart transplantation. Despite technical improvements,
infection is a common complication that is associated with 18% - 60%
of cases (1-4). Infection is more common during the first three months
post-implantation, however, the cumulative risk increases the longer the
device remains implanted. Known risk factors include size, turbulence
(i.e., pulsatile vs. continuous flow designs), and available entry routes.
Environmental risk factors include duration of hospital stay, indwelling
lines and catheters, and the administration of total parenteral nutrition.
Patient risk factors include severity of heart failure symptoms, diabetes
mellitus, obesity, alcohol use, end-stage renal disease, increasing age,
and poor nutritional status (1).

Infection can involve the surgical site, driveline, or pocket pump.
Driveline infections are most common. The driveline contains both the
power and ventilation source and is covered with velour to promote
adherence to tissue. The pocket pump is located in the abdominal cavity
although newer devices are being placed within the pericardium (1, 7-
8). LVAD infections can either be primary or secondary. Primary
infection occurs when the internal portions of the LVAD are infected
first. Secondary infection occurs when there is a bacteremia from a
remote source (e.g., urinary, pulmonary) that contaminates the device
(1).

Clinical presentation is often insidious. Patient complaints include
fever, drenching night sweats, weight loss, and wound related signs such
as drainage, necrosis, erythema, and pain at the percutaneous driveline
site. Other symptoms include chest pain caused by of LVAD malfunc-
tion as well as stroke-like symptoms as a result of embolic phenomena.
When infection is suspected in a patient with a LVAD, the American
Heart Association recommends a basic infection workup including
Gram stain and culture of every potential infection source as well as
ultrasoundography for pocket site infection and a transesophageal echocar-
diogram (1-2). A bacterial cause accounts for 87% of LVAD-associated
infections and commonly involves skin flora pathogens including Staph-
ylococcus aureus and coagulase-negative Staphylococcus, although
gram negative organisms including Pseudomonas, Klebsiella, and Enterobacter
are not uncommon. Other causative pathogens include fungi (9%), viruses
(1%), and protozoa (0.3%) (1).

Definitive treatment requires removal of the LVAD. However, in many cases
this is impossible without concurrent transplantation. When the LVAD cannot
be removed, treatment involves antimicrobial therapy and possibly fluid drain-
age, although, patients often relapse. Toda et al. investigated the effect of
bloodstream infections (BSI) on survival of patients receiving LVAD support.
They found that there was decreased survival two years post-implantation
among patients who had developed a BSI (68%) compared to those individuals
who had not (84%) (3). Although LVAD infections increase mortality, there is
data to suggest that survival may be comparable if the patient subsequently has
a successful transplantation. Schulman et al. performed a retrospective study
investigating the effect on LVAD infections on post-transplant outcomes.
They found that patients who developed a BSI were less likely to be success-
fully transplanted: 31.8% vs. 81.1%. However, if a patient was successfully
transplanted there was no significant survival difference between those patients
who had developed a BSI (85.7%) compared to those who had not (82.5%) (5).

Like all device-related infections, prevention is key. Preventative strategies
include a combination of MRSA nasal decontamination in carriers, sterile
management of the exit site, and perioperative antimicrobial prophylaxis cover-
ing gram positive, negative and fungal organisms (6). Ideally the best strategy
to prevent percutaneous driveline infections would be to develop a fully im-
plantable LVAD. A miniaturized LVAD is currently the subject of clinical
trials as bridge-to-transplant and destination therapy. Unlike previous models
it is implanted in entirety next to the left ventricular apex, which would pre-
sumably decrease infection rates (7-8).

*Contributions from Raymond Smith, MD.


Abdominal Pain and Vomiting
Mehul Pragani, MD, Resident, Internal Medicine, Albany Medical College

Case Presentation:
A 20-year-old woman with a past medical history significant for long-
standing headaches and mental retardation was admitted to the hospital
because of six days of worsening right upper quadrant abdominal pain,
suprapubic pain and intractable vomiting. Four days prior to presenta-
tion she was diagnosed with an E. coli urinary tract infection by her
PCP and was prescribed trimethoprim/sulfamethoxazole. Unfortunately,
his symptoms gradually worsened over the next few days and she decid-
ed to come to the ED. Upon presentation, she was afibrile, her blood
pressure ranged from 122-175 mmHg systolic and 83-103 mmHg dia-
tolic, and she was tachycardic between 91-112 beats per minute. On
abdominal exam, she had right upper quadrant and suprapubic tender-
ness to deep palpation without guarding or rebound tenderness. She did
not have leukocytosis. Her primary laboratory abnormality was trace
blood on her urinalysis.

A CT scan of the abdomen revealed a right adrenal mass of 6.0 x 5 x 4.9
centimeters. Within the first 24 hours of hospitalization, she had intermittent
hypertension with blood pressures that ranged between 122-175 mmHg
systolic and 72-103 mmHg diastolic. She was started on a calcium-channel
blocker. A 24-hour urine collection showed that her metanephrine level was
1195 mcg and her normetanephrine was 6680 mcg; both significantly elevat-
ed. The managing team decided that excision of the adrenal mass would be the
most appropriate treatment. Eleven days prior to surgery, phenoxybenzamine
was started and the calcium-channel blocker was tapered off over four days.
Her blood pressure remained stable during this time and her headaches were
well controlled with medication. A beta-blocker, propranolol, was added to the
patient’s regimen eight days prior to her procedure. She successfully under-
went laparoscopic right adrenalectomy twenty days after admission.

Discussion:
Pheochromocytomas are rare, catecholamine-secreting tumors, arising from
cromaffin cells. Ninety percent occur in the adrenal glands. Extra-adrenal
tumors are found mostly in the organ of Zuckerkandl (1). The annual inci-
dence of this tumor is 2-8 per million people and there is a higher frequency
between the fourth and sixth decades of life (1, 2, 3). Its prevalence in hyper-
tensive patients who have undergone workup for secondary causes of hyper-
tension is 0.3% (4). Excess catecholamine circulation is the primary culprit in
the signs and symptoms seen with pheochromocytoma. The most common finding is hypertension seen in 60-70% of patients (1, 5). Sustained hypertension is seen in 50% of cases, while 45% may present with only paroxysmal hypertension. If the tumor secretes predominantly epinephrine, hypertension may be seen as well (6). Common symptoms include headaches (50%), truncal diaphoresis (50%), and palpitations (50-60%), while rarely, chest pain, abdominal pain, nausea and vomiting may be present as well (1, 2, 7). The sudden and episodic pattern of catecholamine release causes patients to experience bouts of symptoms. The frequency of these episodes can vary between patients and may range from once every few months to several times daily. Precipitating factors include palpation of the tumor, postural changes, exertion, anxiety, trauma, pain, ingestion of tyramine-containing foods, medications (histamine, glucagon, tyramine, phenothiazine, metoclopramide, adrenocorticotropic hormone), intubation, induction of anesthesia, and chemotherapy (6).

Diagnosing the tumor involves imaging and testing for specific biochemical markers. Currently, the most accurate screening procedure for the disease is a 24-hour measurement of urinary fractionated metanephrines, normetanephrine and norepinephrine, with a sensitivity of 99% and specificity of 45% in sporadic cases. Following lab data, imaging should be done to localize the tumor. A CT scan can identify 95% of adrenal pheochromocytomas 1 cm or larger and extra-adrenal tumors > 2 cm. MRI has greater sensitivity and specificity when compared to CT. Further investigation may be done with the radiopharmaceutical agent I-metaiodobenzylguanidine (MIBG) scan. It involves tumor uptake of the agent and allows the investigator to identify adrenal medullary hyperplasia, extra-adrenal pheochromocytoma, and metastases. It has a specificity of 95-100% (6).

In this case, the patient’s adrenal mass was incidentally found on abdominal CT. In these cases, MRI and MIBG may not always be the next appropriate step. A urinary measurement of fractionated metanephrines is essential in diagnosing this tumor. A survey conducted in 2000 looked at 1004 adrenal incidentalomas and it was found that that 4.2% are pheochromocytomas (8).

A CASE OF REFRACTORY HYPOTENSION

**Kerry M. Barba, MD, Resident, Internal Medicine, Albany Medical College**

**Case Presentation:**
A 23-year-old Caucasian man with a history of childhood asthma presented to an outside hospital with two days of fever, chills, dyspnea and cough productive of green sputum. This was preceded by 2 weeks of wheezing. He was presumed to have community-acquired pneumonia after an admission chest radiograph suggested a left sided infiltrate. He was treated with moxifloxacin, supplemental oxygen and a bronchodilator regimen. Sputum cultures were negative.

The patient was transferred to our facility for a higher level of care upon which he developed refractory hypotension and required mechanical ventilation upon arrival to our facility. Physical exam was unremarkable except for tachycardia and coarse breath sounds throughout all lung fields. Laboratory evaluation revealed leukocytosis (WBC 13.2). An ABG showed significant hypoxia despite 100% FiO2 (PaO2/FiO2 58) therapy. The patient was admitted to the medical ICU, and treatment of acute hypoxic respiratory failure secondary to ARDS was continued with full ventilatory support via ARDS net protocol. Review of the CT angiography from the outside hospital confirmed absence of the right pulmonary artery (Figure 1). A transesophageal echocardiogram showed right ventricular systolic dysfunction and pulmonary hypertension.

He was sedated and pharmacologically paralyzed to optimize ventilatory compliance. Nitric oxide therapy was started with subsequent improvement in oxygenation and hemodynamics. Sequential dose-related responses were initially observed. Despite improvements in oxygenation, the patient eventually became refractory to nitric oxide therapy. He went into asystole and succumbed to his illness six days after admission.

The mainstay of therapy for this tumor is surgical excision. Appropriate preoperative management must be carried out before the patient is taken to the operating room. As mentioned earlier, precipitants of excess catecholamine release include induction of anesthesia or tumor palpation. As seen in our case, alpha-adrenergic blockade with phenoxybenzamine should be started at least 10-14 days prior to surgery. Beta-blockade may be started preoperatively, after alpha-blockade, to control supraventricular arrhythmias and tachycardias. This was done with our patient eight days prior to her procedure with propranolol (9).

This case is an example that shows some of the classic signs and symptoms of pheochromocytoma. It may have gone undiagnosed for many years given that the patient did not readily reveal her symptoms due to her mental retardation. Postoperative follow-up should include evaluation of plasma metanephrine levels at 6 weeks and again at 6 months after surgery. The recurrence rate in familial pheochromocytoma is fairly high and annual follow-up is recommended. Imaging should only be done based on test results.

**References:**
A CASE OF REFRACTORY HYPOXIA

contrast to other conditions, the hypoluent lung is the site of abnor-
mality in IUAPA; the compensatory hyperinflation in the contralateral
normal lung gives a false impression of unilateral hyperlucency. Ab-
sence of air trapping on expiratory film can distinguish IUAPA from
SINS.

Once IUAPA is suspected, an echocardiogram can exclude other car-
diovascular abnormalities and demonstrate pulmonary hypertension.
Anatomic details and the presence of bronchectasis can be visualized by
CT or MRI (Figure 2).

Treatment of IUAPA depends upon clinical presentation with varying
therapeutic approaches described in the literature. Surgery, including
pneumonectomy or lobectomy, has been performed in patients with
recurrent hemoptysis or intractable pulmonary infections. Emboliza-
tion of collateral arteries is another possible approach to hemoptysis.
In the setting of significant pulmonary hypertension, revascularization
of ipsilateral hilar arteries demonstrated by pulmonary venous wedge
angiography may significantly improve outcomes, especially if hilar
arteries are large (2). If revascularization is not possible or unsuccess-
ful, medical management similar to that for primary pulmonary hyper-
tension has been postulated to be helpful. For example, inhaled nitric
oxide can be used for selective pulmonary vasodilatation and enhance-
ment of ventilation-perfusion matching (5).

EOSINOPHILIC GASTROENTERITIS

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Case Presentation:

A 42-year-old Taiwanese woman presented to our hospital with recur-
rent abdominal pain and swelling. She denied fever, chills or any other
symptoms except for 1 to 2 weeks of non-bloody diarrhea. She also
denied any sick contacts, animal contact or any pets. She stated that she
did consume raw seafood occasionally, and had done so within the few
weeks prior to admission.

One month prior to presentation, she was also admitted to an outside
hospital (OSH) with similar symptoms. At that time she had a paracen-
tesis with removal of 3.5 liters of ascites fluid. Fluid analysis showed a
4,356 white blood cells (WBC) per high powered field with 97% seg-
mented neutrophils, and 3% monocytes. The fluid cytology was normal
and cultures of the ascites fluid were without any bacterial growth.
Peripheral complete blood count (CBC) revealed significant leukocyto-
sis of 19,900/mm³ with 53% eosinophils. Hepatitis panel was negative
including hepatitis A antibody, hepatitis B core antibody, hepatitis B
surface antigen, and hepatitis C antibody. CA-125 was elevated at 66.8.

Screening for ova and parasites, giardia and cryptosporidium were nega-
tive. Strongyloides IgG and IgM were both negative. CT of the abdo-
men and pelvis with and without contrast showed diffuse simple ab-
dominal ascites, and thickened edematous proximal and mid jejunal
bowel walls. An EGD at the OSH showed an irregular Z-line, non-
bleeding erosive gastropathy, no gross lesions of duodenum, and normal
jejunal mucosa. Biopsy of the jejunum revealed benign, small intestinal
mucosa. The patient was started on albendazole for presumptive parasit-
ic infection and discharged from the OSH.

On review at our hospital, she had a significant past history of seasonal
allergies, allergy to pet dander and gastric reflux. She had allergies to
ibuprofen and cephalaxin, which caused her to have peri orbital swelling.
Her only home medication was omeprazole. She denied any current or
prior smoking, drinking or illicit drug use. She was a housewife, living
with her husband. Her prior work included teaching in Taiwan for 17
years. She is native to Taiwan, and has been in the United States 4
months prior to presentation. Family history was only significant for her
father who had diabetes. Her exam was only significant for moderate
ascites with mild diffuse abdominal pain on palpation. Laboratory studies
including liver function tests were normal, except for a slightly low albumin.
Stool culture, ova, parasitology, and clostridium difficile testing were negative.
Erythrocyte sedimentation rate, CRP, anti-nuclear antibody and anti-
mitochondrial antibody were unremarkable.

Paracentesis with removal of 1.3 liters of ascites fluid showed WBC of 10,816/
mm³, with 97% eosinophils and RBC of 7,000/mm³. Serum ascites albumin
gradient was less than 1.5. Fluid cytology, culture, adenosine deaminase, acid-
fast bacilli smear were all unremarkable. Tryptase, Quantiferon Gold, Chic-2
genetic test for 4q12 deletion, and repeat IgG and IgM for Strongyloides were
negative. Repeat CT scan showed a non-urgent paraduodenal hernia, non-
specific swirling appearance to the superior mesenteric vein around the superi-
or mesenteric artery, and marked ascites. Repeat EGD with multiple biopsies
up to the jejunum, all showed normal mucosa. Subsequent colonoscopy with
biopsies was also normal. Bone marrow biopsy results are limited, but showed
eosinophilia with normal morphology.

Infectious Disease, Hematology, and Gastroenterology services were involved
during this hospitalization. Peritoneal biopsy was considered, but deemed too
invasive and with too many complications. She continued to have a peripheral
leukocytosis with eosinophilia at greater than 60%. Her ascites re-accumulated
rapidly, and she required repeat paracentesis for symptomatic relief 8 days
after the first. With close follow up appointments, within one week, she was
discharged home with a prednisone taper for presumptive eosinophilic gastro-
enteritis.

Discussion:

Eosinophilic Gastroenteritis (EG), first described in 1937, is a rare disorder
with multiple gastrointestinal manifestations. Although the pathogenesis and
cause of the disease are not fully understood, hypersensitivity is thought to
play a major role. Many patients that are affected have a predisposing history
of eczema, atopy, asthma, seasonal allergies, food sensitivities, and increased
levels of serum IgE (1, 2). Studies have shown a major role played by eosino-
phils and related chemokines, such as eotaxin, which is specific for eosinophils.
Cytokines mediated by Th-2 cells such as IL-3, IL-5, and IL-13 are also in-
volved. Most cases occur in whites, but it has also been noted in asians. Most

*Contributions from Brianne Navetta, Michael Schuster, Anthony L. Malanga, & Scott Beegle, MD.

in persons without the right pulmonary artery. N Engl J Med 1980; 303:1070-1073. 4) Van BBW, Le FF, Gong T, Ooi CS. -
Eosinophilic Gastroenteritis (CONTINUED)

of those affected are men in their 30’s to 50’s. EG clinically manifests depending on the affected site of the gastrointestinal tract. There are three main subtypes: mucosal (the most common), muscularis, and subserosal. Mucosal EG may present with anemia secondary to gastrointestinal blood loss, and weight loss due to malabsorption or protein losing enteropathy. Muscularis EG can present with small bowel or gastric outlet obstruction. Lastly, subserosal EG may present as eosinophilic ascites, as seen in our patient. The literature shows that many have extra-intestinal manifestations such as eosinophilic cystitis, splenitis, or hepatitis (1). Lab testing for intestinal parasite infection should be done, as these can mimic EG (3). Diagnosis of EG depends on eosinophilic infiltrate on biopsy. Biopsy of gastric and small intestinal mucosa is diagnostic in up to 80%, and often multiple biopsies are required as the infiltration can be patchy. Peripheral eosinophilia may or may not be present. If the peripheral eosinophilia is greater than 1,500 cells per microliter for greater than 6 months, then hypereosinophilia syndrome, an idiopathic disease, must be considered. Treatment is primarily corticosteroids, although anti-histamine, mast cell stabilizing and, leukotriene inhibiting drugs and empiric anti-helminthic therapy have been trialed (1, 3, 4).

*Contributions from Mandeep Singh MD and Xinjun Zhu MD.


AMR Image Challenge

A 58 year old female presented to Albany Medical Center with worsening anemia and heme positive stools with a concern for chronic GI blood loss. An EGD was performed and the above image in the pylorus is what was found.

Question: What is the diagnosis?

Answer: Gastric Antral Vascular Ectasia (GAVE), or also referred to as Watermelon Stomach for the long red streaks present in the mucosa.