Thoracoabdominal Aortic Graft Infection Presenting As Failure To Thrive

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Citation

Abstract
A case of prosthetic aortic graft infection is presented here followed by a review of prosthetic vascular infections highlighting the diverse clinical presentations and complex management issues.

CASE
A 63 yr old woman was admitted directly to hospital from the cardiothoracic surgery clinic for further evaluation in September 2006. She had thoracoabdominal aortic aneurysm repair in May 2006 for recurrent renal embolization complicated by renal infarction. Since surgery, patient developed anorexia, 40lb weight loss in 4 months and chronic abdominal pain. She was assessed as failure to thrive. She also complained of generalized fatigue, malaise and thirst. She denied fevers but admitted to chills. An infectious disease consult was requested to assist the primary team in further evaluation and management of the patient.

Systemic review revealed cough productive of clear sputum for two months but otherwise as in history of present illness. Additional past medical and surgical history include depression, pulmonary embolism, hypothyroidism, total abdominal hysterectomy and bilateral salpingo-oophorectomy and hyperlipidemia. She had no known allergy to any medications. She had a family history of coronary artery disease and hypertension. She was a current smoker and had a 40 pack-year smoking history. She had a distant history of alcohol abuse. Her current medications were empiric intravenous antibiotics (Vancomycin and piperacillin-tazobactam) and her home medications (esomeprazole, metoprolol, warfarin, levothyroxine and escitalopram).

Physical examination revealed a chronically ill-looking middle aged woman who was not toxic. She was lethargic but easily aroused. She was partially oriented to time. Her vitals signs were BP 101/59 mmHg, pulse 90 bpm, respiratory rate 20 bpm, temperature 36.6 °C, weight 114 pounds and height of 5ft 2in. Pertinent findings included a left paramedial abdominal scar with a non-healing superior aspect but without any signs of inflammation and tenderness over lower thoracolumbar spine and left paraspinal muscles.

Significant laboratory results include a serum creatinine of 2.8 mg/dl, serum sodium of 127mEq/L, white blood count (WBC) of 18,800 cells/mm$^3$ with 78% neutrophils and 6% band forms. Hemoglobin was 10.0 g/dL. Liver functions tests were within normal limits except for serum alkaline phosphatase of 254 units/L. Computed tomography (CT) scan of abdomen and pelvis revealed air around the aortic graft and a left retroperitoneal mass.

An assessment of a thoracoabdominal aortic graft infection and a non healing abdominal surgical wound was made. Repeat blood culture was taken and empiric antibiotics continued. Surgical debridement of vascular graft and sending appropriate operative cultures was planned. Magnetic resonance imaging/angiography (MRI/MRA) of chest and abdomen showed gas within the proximal portion of the thoracoabdominal aorta graft concerning for fistulization with the distal esophagus, left complex renal abscess and areas of infarction in the right kidney.

An upper gastrointestinal series showed no esophageal perforation or fistula. On hospital day 2, patient developed a fever of 38.9°C. The only positive culture was a wound culture that grew extended spectrum beta lactamase (ESBL) producing Enterobacter cloacae. Piperacillin-tazobactam was discontinued and meropenem started.

On hospital day 6, exploration, irrigation and debridement and washout of the thoracoabdominal aortic graft was done.
Also performed was retroperitoneal debridement and washout, left nephrectomy. Patient was extubated the following day and had an uneventful postoperative course. Operating room cultures grew ESBL Enterobacter cloacae. Vancomycin was stopped. Intravenous Meropenem and Ciprofloxacin were continued for 2 weeks postoperative then oral ciprofloxacin for 6 weeks then lifelong oral trimethoprim/sulfamethoxazole. Patient was discharged home 4 weeks after surgery.

Figure 1
Figure 1: MRA of the thorax showing air around the thoracic aortic graft.

Figure 2
Figure 2: MRA of the abdomen showing air around abdominal aortic graft and left renal complex abscess.

DISCUSSION
Prosthetic vascular graft (PVG) infection is an uncommon cause of failure to thrive and fever of unknown origin especially when it presents as an intrabdominal abscess as demonstrated with the case above. PVG infection is defined in one the following three ways: perigraft infection or abscess formation, exposed graft due to disruption of overlying soft tissue and graft erosion or fistula formation involving a mucosal surface. The incidence varies according to the site of infection ranging from <1% in aortic grafts to about 6% in infrainguinal grafts [1,2]. Ducasse et al showed a male predominance (M:F= 4:1) and immunodeficiency in 23% of patients [2]. Soetevent et al found no specific risk factors for graft infections [3].

The most common route of contamination is thought to be microbial seeding at the time of surgery. Other routes include spread from adjacent infection and secondary graft infection from a remote site seeding the bloodstream [1]. The microbiological profile is a varied one. The most commonly implicated organism is Staphylococcus aureus in about 43-54% of cases, usually runs a virulent course and usually occurs early (less than 3 months after surgery) [2,4]. Less virulent organisms cause delayed infections e.g. coagulase negative staphylococci, corynebacterium spp and propionibacterium acnes. Enterococci and anaerobes usually are seen in the setting of a polymicrobial infection (14%). Still less commonly seen are candida spp. In 30% of cases, no isolates are found [4].

In early infections, the usual clinical presentation is systemic signs of sepsis with fever, chills, leukocytosis and bacteremia or fungemia. Local inflammatory features may also be prominent such as abscess, sinus tract formation, graft occlusion, pseudoaneurysm, graft exposure and poor tissue incorporation. Late infections have less systemic toxicity. The local stigmata as mentioned above predominate in the clinical presentation [1-4]. For some patients, there are no systemic or local signs but the culture of the explanted graft material is positive. Much less commonly, patients complain of local pain without a palpable mass. They may also present with life-threatening hemorrhage as a result of graft-enteric fistula. Distal ischemia with pain due to graft thrombosis or embolization is also seen [1-4].

CT scan is vital for evaluating a patient for possible PVG infection with sensitivity and specificity approaching 100%. Findings on CT scan that suggests a complicating graft infection include the presence of perigraft fluid not attributable to recent (< 3 months) graft implantation, increasing perigraft fluid in the early post-graft implantation setting, perigraft fluid with fat stranding of gas bubbles, lack
of fat plane between graft and bowel, and anastomotic aneurysms. The perigraft findings on CT scan may be minimal to nonexistent for less virulent pathogens. Image-guided percutaneous aspiration of the perigraft fluid should be considered at the time of initial CT scan if no pseudoaneurysm is seen and fluid sent for cytologic and microbiologic analysis. MRI may show subtler perigraft inflammatory changes than CT scan. Other investigative modalities include indium-labeled WBC scan, ultrasonography, sinography and gastrointestinal endoscopy may also be employed. Obtaining operative cultures of explanted graft tissue and perigraft aspirate is paramount to achieving a microbiologic diagnosis. Blood cultures are often negative especially in late onset. Recovery of organisms may be enhanced by broth culture of graft, sonification of graft and molecular methods such as polymerase chain recovery.

The principles of management are fourfold. Firstly, the infected graft must be excised. Secondly, complete debridement of all infected perigraft tissues is necessary. Thirdly, targeted parenteral antimicrobial therapy over about 6 weeks is important. Lifelong suppressive antibiotics must be undertaken if graft excision is not possible. Lastly, revascularization is important for survival of distal tissues and is undertaken by extra-anatomic or in-situ bypass.

Complications of PVG infections include stump blowout, limb ischemia or mortality. Mortality is seen in up to 18% of cases. Mortality is worse with conservative vs. surgical management (36.4% vs. 14%), worse with extra-anatomic vs. in-situ bypass (16% vs. 5.8%) and worse with early vs. late infection (80% vs. 30%).

References
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