Introduction:
Medications that inhibit tumor necrosis factor alpha (TNFα) are used to treat autoimmune inflammatory diseases such as rheumatoid arthritis and psoriasis. Their use has been associated with an increased risk of mycobacterial and fungal infection.1 Recent studies have suggested that serious infections caused by a wide range of organisms are more frequent among patients taking anti-TNFα medications.2,3 This case describes a life-threatening infection in a patient taking etanercept.

Case Presentation:
A 42 year old man presented to hospital with a 24 hour history of headache, dental pain, nausea, vomiting, fever and left sided weakness. Two weeks prior he had experienced an URI type illness with some mild confusion but symptoms had resolved spontaneously within 48 hours and he did not seek medical evaluation. Past medical history was significant for psoriatic arthritis treated for the last 18 months with etanercept. On examination he was febrile to 101°F, tachycardic and delirious. His dentition was poor and there was a spastic paresis of the left arm and a left facial droop. Laboratory studies showed WBC 14.0 with 95% neutrophils. A CT scan (Figure 1A and Figure 1B) showed a large right frontal mass, surrounding edema and effacement of the right ventricle with midline shift. The patient was empirically treated with intravenous ceftriaxone, metronidazole and vancomycin before being taken to the operating room. In the OR an encapsulated purulent mass was removed which later grew Streptococcus constellatus of presumed odontogenic origin. The patient made a good recovery over the next week but his left sided paresis persisted and he was discharged to a rehabilitation facility.
Large streptococcal brain abscess in a patient taking anti-TNF medication (continued)

Conclusion: As the use of anti TNF α medications increases it is important to consider the potential adverse effects including life threatening infections that are not limited to mycobacterial or fungal etiologies. Any infectious symptoms should be evaluated thoroughly in this high risk group.

Discussion:
• Brain abscess accounts for 1-2% of all intracranial masses in the developed world.
• They may develop via contiguous spread of infection from adjacent structures such as otitis media and sinusitis, or via hematogenous spread from a distant site of infection e.g. endocarditis with septic emboli.
• Causative organism depends on the mechanism of infection and the immune status of the host. Many pyogenic brain abscesses are polymicrobial. Otogenic and odontogenic infections may be caused by anaerobes, rhinogenic infections by streptococcus and post surgical infections by staphylococcus. In immunocompromised patients consider other organisms such as listeria, pseudomonas, mycobacterium tuberculosis, toxoplasma, nocardia and fungi.
• Antibiotics should be based on clinical suspicion of the causative organism but an empiric regimen to cover local invasion and hematogenous spread might include metronidazole, a third generation cephalosporin and vancomycin.
• Neurosurgical evaluation should be obtained immediately. Intervention may be diagnostic, to obtain a microbiological diagnosis and narrow the spectrum of antibiotics, or therapeutic.
• The use of steroids and anticonvulsant therapy will depend on the clinical presentation and amount of edema surrounding the abscess.
• Meta-analysis suggests that there is an increased risk of serious infections in patients taking anti TNF α medications.
• Anti TNF α medications should be discontinued in the setting of severe infection or sepsis
• Anti TNF α medications are relatively contraindicated in the setting of chronic or recurrent infection. A US “boxed” warning for etanercept states: “Caution should be exercised when considering the use in patients with chronic infection, history of recurrent infection, or predisposition to infection (eg, poorly-controlled diabetes or residence/travel from areas of endemic mycoses). Do not give to patients with an active chronic or localized infection. Patients should be educated about the symptoms of infection and closely monitored for signs and symptoms while undergoing treatment.”
• Patients should be screened for latent tuberculosis before anti TNF α medication is initiated.

References:

Crohn’s colitis complicated by a “secondary” Entamoeba histolytica infection: A case report and review of the literature

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Introduction: Amebiasis, caused by Entamoeba histolytica, is a disease of worldwide importance and estimated to result in 40,000 to 100,000 deaths annually (1). Because the primary mode of transmission is through ingestion of fecally contaminated food and water, its prevalence is greatest in areas with inadequate sanitation (2). In developed countries, amebiasis primarily affects migrants from and travelers to endemic regions, men who have sex with men, and immunosuppressed or institutionalized individuals. E. histolytica infection is asymptomatic in the majority of individuals, however symptoms may progress from mild diarrhea to dysentery and crampy abdominal pain (3). The clinical features can closely mimic ulcerative colitis or Crohn’s colitis. Because the treatment of Inflammatory Bowel Disease (IBD) is different from that of amebiasis, it is important to recognize amebiasis and treat it appropriately. Moreover, the coexistence and superimposition of amebic colitis with IBD may have serious clinical implications. There have been very few reports in the literature presenting the superimposition of amebiasis on...
Crohn’s disease. This report is a rare case of Crohn’s colitis with superimposed *Entamoeba histolytica* infection.

**Case Presentation:**
A 46-year-old Army veteran with a past medical history significant for Crohn’s disease presented to the hospital complaining of abdominal pain, intermittent bright red blood per rectum and worsening diarrhea. The patient was diagnosed with Crohn’s disease in 1993 and stated that he had a 17 year history of chronic diarrhea, which at times was blood streaked. The diarrhea was associated with vague abdominal pain and occurred often in the mornings. Previous evaluation for his chronic diarrhea included sigmoidoscopies in 2000, 2004, and 2008 all of which had biopsy findings consistent with Crohn’s disease. The patient was started on Mesalamine 800 mg TID and Azathioprine 50 mg daily in 2000. Due to an inadequate clinical response, the patient was transitioned to Infliximab in 2006. Despite symptom improvement, he stopped taking Infliximab in 2009 because of insurance issues. The patient reported no other past medical history and denied taking other medications. He denied any weight loss, low-grade fevers, and oral or skin ulcers. The patient denied any recent antibiotic use, hospitalizations, travel, or sick contacts. Over the past seven years, his travel included the East Coast of the United States, Texas, and Southern California. While in the army, he spent significant time in Germany, France, Italy, and Austria.

The patient was re-started on Mesalamine. Stool studies were negative for culture, ova/parasite, *C. difficile* toxin, *Cryptosporidium* and *Giardia*. Surrogate markers for Crohn’s disease including C-ANCA, P-ANCA, and Saccharomyces cerevisiae were all negative. A colonoscopy revealed flask shaped ulcers within the sigmoid colon and mild cecal inflammatory changes. The biopsies from the sigmoid revealed trophozoites, most consistent with *E. histolytica*. (Figure 1) None of the biopsies within the colon or terminal ileum were consistent with Crohn’s disease. The patient’s immune hemagglutination assay for *E. histolytica* was found to be positive at 2.783 (positive > 0.4). The patient was treated with metronidazole 500 mg 3 times daily for 10 days, followed by paromomycin 30 mg/kg PO daily for 7 days. His Mesalamine was also continued. After a week the patient reported that he was having one to two non-bloody bowel movements a day. After completion of metronidazole and paromomycin, he had complete resolution of his abdominal pain, hematochasia, and diarrhea.

**Discussion:**
The clinical characteristics and the colonoscopic findings of amebic colitis can resemble either ulcerative colitis or Crohn’s disease (4, 5). Although recognition of most cases of amebiasis is usually straightforward, the coexistence of Crohn’s disease with a superimposed *E. histolytica* infection can be difficult to diagnose and can lead to complications if not properly identified and treated. We present an unusual case of an individual with Crohn’s disease with a superimposed *E. histolytica* infection. A search of the literature revealed few similar cases, especially in North America.

Most reported cases of Crohn’s disease with superimposed *E. histolytica* have occurred in areas endemic for amebiasis. In the majority of cases, the diagnosis of amebic colitis was often mistaken for IBD and amebic colitis was not diagnosed until surgical intervention was required. In 1991, a patient with a 7 year history of Crohn’s colitis developed invasive amebic colitis during steroid and 6-mercaptopurine treatment for active disease. The patient’s stool specimens, mucosal biopsies, and serological studies were negative for *E. histolytica* and the diagnosis was established on pathological examination of the surgically resected bowel (6). In 2006, Ozdogan et al described a patient who had Crohn’s colitis with superimposed *E. histolytica* infection resulting in colonic perforation and an intra-abdominal abscess. Post operative pathology revealed numerous trophozoites of *E. histolytica* in the subserosa and muscularis propria.

The literature has shown that patients with IBD have a greater prevalence of amebiasis as compared to the normal population (8). Factors leading to the increased development of *E. histolytica* infection in patients with IBD are likely related to conditions favoring the proliferation and invasion of the protozoan parasite. Ingestion of *E. histolytica* initiates infection. Excystation in the intestinal lumen produces trophozoites that use the galactose and N-acetyl-D-galactosamine specific lectin to adhere to colonic mucins and thereby colonize the large intestine. Chadee and others have demonstrated the *E. histolytica* adherence lectin (Gal-Lectin) stimulates TNF-alpha by macrophages (9). The TNF-alpha attracts *E. histolytica* trophozoites through chemotaxis, which may aid the parasite in the process of tissue invasion (1, 10). Peterson et al found that higher levels of TNF-alpha were associated with increased risk of first and recurrent *E. histolytica* related diarrheal episodes. Several studies have also shown increased TNF-alpha levels in the serum and intestinal mucosa of individuals with IBD (11, 12). Other conditions favoring *E. histolytica* proliferation include alcohol use, malignancy, malnutrition, and steroid use. The administration of corticosteroids or other immunosuppressive drugs should be avoided in order to prevent precipitating fulminant amebic colitis with toxic megacolon or perforation. Yamamoto et al reported a patient who developed toxic megacolon after being treated with steroids for suspected ulcerative colitis (13).
The colonoscopic findings of amebic colitis can mimic IBD (4, 5). The presence of discrete ulcers with normal intervening mucosa is more likely to be mistaken for Crohn’s disease than ulcerative colitis (4, 5). Endoscopy in amebiasis often shows ulcers in the sigmoid, rectum, and the cecum (14, 15). Early lesions of amebic colitis may show only erosions of the mucosa, however as the disease progresses, flask shaped ulcers along the long axis of the colon may be seen (16). A yellow slough, which contains the trophozoites, often covers the ulcers. The slough must be included in the biopsy along with the edges of the ulcers for a high diagnostic yield (17).

Diagnosis can often be challenging as patients with E. histolytica often present with non specific symptoms such as diarrhea and abdominal pain. Examination of stool is simple and inexpensive and may reveal trophozoites of E. histolytica (14). However antibiotics can interfere with results and multiple specimens may need to be collected to detect the organism (18, 19). Pai evaluated biopsies of 11 patients with histopathologically diagnosed amebic colitis, who were initially suspected to have tuberculosis or IBD. Amebae were not detected in the single patient in his series who had a stool examination. The patients in the series of Marcus et al and Blumencranz et al were found to have negative stool examinations as well (15). Recent studies have shown that microscopic examination of a single stool specimen may be positive in only one third to one half of the cases, therefore three or more exams may be required to identify the parasite (19). The diagnosis can be further complicated by the fact that E. histolytica is morphologically similar to E. dispar, a non pathogenic organism (14, 15). Ustun et al found that the trichome staining method is more effective for the detection of E. histolytica/E. dispar as compared to the wet mount + Lugol’s iodine staining and modified formol ethyl acetate methods. Diagnosis may also be made using both the serum and indirect hemagglutination antibody and the enzyme linked immunosorbent assay, which are highly sensitive (90-100%) (20). They often become positive within 7-10 days of infection, however they are unable to distinguish between active or prior invasive infection (20).

Treatment of amebiasis is aimed at eliminating the invading trophozoites and eradicating intraluminal encysted organisms. Metronidazole 500 to 750 mg PO three times daily for 7 to 10 days in adults, is the treatment of choice for invasive colitis. It has been reported that E. histolytica can persist in the intestine in up to 40 to 60 percent of patients who receive a nitroimidazole (21). Therefore, it is recommended that treatment be followed with paromomycin to eradicate luminal infection (21).

We have presented a patient with Crohn’s disease with a superimposed E. histolytica infection who does not live in an area endemic for amebiasis. It has been previously suggested that patients who live in areas endemic for amebiasis be screened for E. histolytica; however this practice is not common in North America. E. histolytica/E. dispar should be included in the differential diagnosis of all patients with suspected IBD because of the detrimental results that can occur with a misdiagnosis. While most cases involve patients who have ulcerative colitis superimposition with E. histolytica in areas endemic for amebiasis, our case demonstrates an E. histolytica superimposition in a patient with Crohn’s disease here in North America.

References:
CROHN’S COLITIS COMPlicated BY A “SECONDARY” ENTAMOEBA HISTolyTICA INFECTION: A CASE REPORT AND REVIEW OF THE LITERATURE (CONTINUED)


A 52 YEAR OLD FEMALE WITH ELEVATED METHEMOGLOBIN FOLLOWING A TRANSESOPHAGEAL ECHOCARDIOGRAM: A CASE REPORT

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Case Presentation:
A 52 year old lady with a past medical history significant for mitral valve prolapse presented to an outside hospital complaining of a two day history of progressive full body myalgias, low back pain, fatigue, anorexia, constipation, and delirium. The patient had undergone a root canal procedure one week prior to admission. In the emergency room, she was intubated for airway protection and blood, CSF, and synovial fluid from her right wrist were sent for analysis. CT and MRI of the spine revealed an epidural abscess stretching from C2 to C6 and osteomyelitis involving C5 and C6. Blood, CSF, and synovial fluid samples grew methicillin sensitive Staphylococcus aureus (MSSA). In the interim, the patient was transferred to Albany Medical Center for neurosurgical and orthopedic evaluation. The patient underwent epidural abscess resection as well drainage of the wrist. Because of the MSSA bacteremia, a transesophageal echocardiogram (TEE) was performed to rule out infective endocarditis.

Topical benzocaine was administered to the oropharynx prior to passing the probe to the gastric fundus. No vegetations were visualized and the patient tolerated the procedure well. Following the procedure, the patient became increasingly hypoxic by pulse oximetry. An arterial blood gas sample was obtained and was significant for its dark brown colour, elevated oxygen partial pressure, decreased oxyhemoglobin, and elevated methemoglobin. The patient was given intravenous methylene blue 65mg, followed by a repeat arterial blood gas one hour following administration by which time oxygen partial pressure, oxyhemoglobin, and methemoglobin levels had normalized.

Discussion:
One of the complications of benzocaine administration, a frequently used topical anaesthetic agent used in various endoscopic procedures, is acquired methemoglobinemia. Methemoglobinemia arises when hemoglobin is oxidized from its ferrous iron state (Fe²⁺) to its ferric iron state (Fe³⁺). When hemoglobin is in its ferric iron state, it no longer can bind oxygen and it further interferes with oxygen delivery to tissue by “shifting” the oxygen dissociation curve to the left [1]. Benzocaine accelerates the conversion of ferrous to ferric iron. Typical features of methemoglobinemia include cyanosis, dark brown appearance of arterial blood, low pulse oximetry readings, and normal PaO₂. Pulse oximeters estimate arterial oxygen saturation by measuring the absorbance of two wave-lengths of light, which detect oxyhemoglobin and reduced hemoglobin. This accounts for the dissociation of pulse oximetry findings and PaO₂ seen in methemoglobinemia [2]. The goal of treatment is to restore oxygen delivery by promoting reduction of hemoglobin back to its ferrous iron state. In general, methemoglobinemia is treated with methylene blue if: (1) methemoglobin is greater than 30% or, (2) if there are signs of tissue hypoxia, central nervous system depression, or hemodynamic instability. Alternative therapies for refractory cases include exchange transfusions and hyperbaric oxygen [1]. If the patient suffers from glucose-6-phosphate dehydrogenase deficiency (G6PD), methylene blue should not be administered, since G6PD is
required in the overall conversion of methylene blue into its active metabolite. Treating this population with methylene blue is not only ineffective but also can worsen oxidative stress [2]. In these patients, ascorbic acid (300-1000mg per day) is recommended [3]. Failure to recognize and treat methemoglobinemia can result in death from cardiopulmonary failure [3].

Acquired methemoglobinemia secondary to benzocaine is extremely rare. In a study of 28, 478 TEEs performed at the Mayo Clinic using topical 20% benzocaine, 19 patients developed methemoglobinemia for an incidence of 1 case per 1499. Risk factors include high oxidative stress states such as sepsis, systemic inflammation, and malignancy. During these stress states, cytochrome b5 reductase is thought to be saturated, thereby, impairing reduction of methemoglobin. Other potential risk factors include pharyngeal mucosal inflammation causing increased absorption of benzocaine and concurrent use of various medications (e.g., dapsone). Lastly, although anemia does not directly affect methemoglobin production, in the Mayo Clinic study, most patients who developed methemoglobinemia had concurrent anemia. The decreased hemoglobin led to greater clinical manifestations of methemoglobinemia [3].

Despite being a known complication, benzocaine-related methemoglobinemia is poorly understood. Guay reviewed 242 documented episodes of acquired methemoglobinemia secondary to local anesthetics notably: prilocaine, benzocaine, lidocaine, and tetracaine. She concluded that some patients seem to be susceptible to develop benzocaine-related methemoglobinemia and some people are not. Some patients have developed the condition after a one second spray and some patients have failed to develop the syndrome after receiving large doses. More importantly, it is impossible to predict who will develop the syndrome and who will not. For this reason, she argues that the use of benzocaine in the clinical setting should be abandoned [4].

Conclusion:
Acquired methemoglobinemia secondary to topical benzocaine use during TEE is a rare adverse reaction with a potential of causing serious hypoxic tissue injury and possibly death. If methemoglobin is greater than 30% or if there are signs of tissue hypoxia, central nervous system depression, or hemodynamic instability, the patient should promptly be treated with methylene blue unless the patient suffers from G6PD deficiency. Because it is not possible to predict which individuals will suffer from this potential mortal condition, we should consider abandoning use of topical benzocaine in clinical settings.

References:

Staphylococcus lugdunensis causing Tricuspid valve and AICD intracardiac endocarditis
Neil Yager, DO, Resident, Internal Medicine, Albany Medical College; Cynthia Carlyn, MD, Section of Infectious Disease, Stratton VA Medical Center; Jessica Chapman, MD, Division of Internal Medicine, Albany Medical College; Sujata Balulad, MD, Division of Cardiology, Albany Medical College

Introduction:
Automatic implantable cardioverter defibrillators (AICD) and pacemakers are being utilized in patients more than ever. Infections of these devices is one of many potential complications. We report a rare case of Staphylococcus lugdunensis AICD and tricuspid valve endocarditis.

Case Presentation:
A 58-year-old male presented to the Emergency Department by Emergency Medical Services with four days of fevers, chills and generalized malaise. The majority of the history was taken through the family because the patient was very confused and disoriented during the interview. He was experiencing ‘flu like’ symptoms, diarrhea and worsening confusion. He had been talking nonsensically and not answering questions appropriately.

His past medical and surgical history is significant for
Staphylococcus lugdunensis causing Tricuspid Valve and AICD Intracardiac Endocarditis (continued)

for obesity, hypercholesterolemia, chronic renal insufficiency, obstructive sleep apnea (CPAP at night), severe chronic obstructive pulmonary disease, congestive heart failure, a biventricular Medtronic Concerto ICD placement several years prior, and coronary artery bypass surgery one year prior. He smoked for over 40 years. He lived alone and there was no known alcohol or recreational drug abuse.

On physical exam his vitals were temperature 99.9 degrees Fahrenheit, heart rate 102 beats per minute, blood pressure 73/62 mmHg, respiratory rate 20 breaths per minute, and pulse oximetry 93% on 2L nasal cannula. In general he was an obese male, awake and alert but disoriented to place and time. His answers to questions were inappropriate. He spoke in full sentences. His head was normocephalic and atraumatic, pupils were round and equally reactive to light bilaterally. Oropharynx was normal and mucus membranes moist. The neck showed no jugular venous distention, no bruits and was non-tender. Cardiovascular exam was remarkable for diminished heart sounds (thought due to body habitus) no third or fourth heart sound and no murmurs, rubs or gallops. He had a well-healed midline incision on his chest. His AICD site was without signs of infection. The lung fields had diffuse expiratory wheezes throughout. The abdominal and genital and rectal exams were normal. Lower extremities were cool, with chronic venostasis changes bilaterally and delayed capillary refill. On neurologic examination the patient followed some commands but was rather agitated. His muscle strength was 5/5 throughout and he had no focal neurological deficits.

EKG and chest x-ray were non-revealing. White blood cell count was 21.6 x10^3 cells per cubic millimeter of blood with 24% bands. Metabolic profile was remarkable for a potassium of 6.5 mEq/l and a creatinine of 7 mg/dl. His liver function tests, lactic acid and ammonia were normal. Troponin was 0.11 ng/ml, CK-MB 12.9 ng/ml, % MB 7. He was admitted to the Intensive Care Unit (ICU) and received emergent hemodialysis.

Two separate blood cultures on admission 20 minutes apart were positive for S. lugdunensis. Transesophageal echocardiogram demonstrated tricuspid valve vegetations and thrombi and vegetations on the intracardiac AICD leads. He received two weeks of IV oxacillin before cardiothoracic surgery explanted the entire AICD device and wires. Pacer pocket and left chest tissue cultures were negative. Blood cultures post operatively remained negative. The patient received three more weeks of appropriate antibiotic therapy. Unfortunately due to his underlying co-morbidities and multi-organ damage from ongoing sepsis, the patient expired approximately one month after removal of the infected device. The cause of death was attributed to S. lugdunensis endocarditis in the setting of his multiple co-morbidities.

Discussion:

S. lugdunensis is a virulent coagulase negative staphylococcus causing a variety of infectious diseases including meningitis, abscesses and endocarditis. It tends to be more virulent than other coagulase negative staphylococci and has been compared to Staphylococcus aureus in that respect. 1 The organism forms biofilm allowing it to stick to devices and limit antibiotic penetration. 2 The clinical spectrum of disease due to S. lugdunensis is shown below in Figure 2.
**STAPHYLOCOCCUS LUGDUNENSIS CAUSING TRICUSPID VALVE AND AICD INTRACARDIAC ENDOCARDITIS** (CONTINUED)

*S. lugdunensis* tricuspid valve endocarditis has been reported infrequently in the literature. Most reported cases of *S. lugdunensis* endocarditis have been in the setting of prosthetic aortic valves and mitral valves. It remains unclear why the tricuspid valve was affected in our patient but it may have to do with the generalized virulence of the organism. There are also few reported cases of *S. lugdunensis* pacemaker lead infections, and even fewer cases of *S. lugdunensis* AICD lead infections. The first case of AICD lead endocarditis due to this organism was reported in 2009. Table 1 below summarizes all cases of intracardiac *S. lugdunensis* infection including ours.

### Table 1: Summary of all cases of *Staphylococcus lugdunensis* intracardiac lead endocarditis; *International Journal of Infectious Disease* (2010) 4

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Age</th>
<th>Gender</th>
<th>Source of infection</th>
<th>Comorbidities</th>
<th>Initial management and outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anguera 1998</td>
<td>60/F</td>
<td>Pacemaker pocket infection</td>
<td>Pacemaker</td>
<td>Medical, culprit at 6 weeks, alive</td>
<td></td>
</tr>
<tr>
<td>Sohail 2008</td>
<td>65/M</td>
<td>Pacemaker pocket infection</td>
<td>OOC, Pacemaker</td>
<td>Surgical, alive</td>
<td></td>
</tr>
<tr>
<td>Sohail 2008</td>
<td>65/M</td>
<td>Pacemaker pocket infection</td>
<td>Pacemaker</td>
<td>Surgical, alive</td>
<td></td>
</tr>
<tr>
<td>Anguera 2005</td>
<td>60/F</td>
<td>Not reported</td>
<td>Pacemaker</td>
<td>Medical, culprit at 1 year</td>
<td></td>
</tr>
<tr>
<td>Renzulli 2005</td>
<td>65/F</td>
<td>Not reported</td>
<td>Pacemaker</td>
<td>Surgical, death</td>
<td></td>
</tr>
<tr>
<td>Oltmanns 2005</td>
<td>75/M</td>
<td>Chronic pacemaker pocket infection</td>
<td>Pacemaker</td>
<td>Surgical, alive</td>
<td></td>
</tr>
<tr>
<td>Seifert 2009</td>
<td>61/M</td>
<td>Multiple abscesses at the pacemaker site</td>
<td>Left nephrectomy (renal), Pacemaker</td>
<td>Medical, culprit at 2 and 4 months</td>
<td></td>
</tr>
<tr>
<td>Chopra 2009</td>
<td>41/M</td>
<td>Diabetic nephropathy, Non-technical multivisceral, Pacemaker (2002), 2MO, Stage V Heart Disease, Nephrology</td>
<td>Surgical, alive</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sohail 2017</td>
<td>65/M</td>
<td>AICD, myocardial valve</td>
<td>OOC, Non-technical multivisceral</td>
<td>Surgical, death</td>
<td></td>
</tr>
</tbody>
</table>

Risk factors for endocarditis from this organism seem to be prosthetic valves especially the aortic valve and intracardiac hardware. Pacemaker lead endocarditis from *S. lugdunensis* has a mortality rate of 14%. Mortality rate is high and antimicrobial therapy alone is inadequate to eradicate the infection. Removal of the device in conjunction with antimicrobial therapy is the best treatment.2

### References:


### Acknowledgements:

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