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Infective Endocarditis Caused by Haemophilus Influenzae Type 1 in a Healthy, Immunocompetent Patient

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Case Report

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Abstract

Haemophilus parainfluenzae is a common HACEK (Haemophilus species, Aggregatibacter species, Cardiobacterium hominis, Eikenella corrodens, Kingella species) associated with infective endocarditis, however, it is exceedingly rare to find Haemophilus influenzae as the causative organism. We present a case of infective endocarditis caused by Haemophilus influenzae and discuss the common presenting symptoms, appropriate diagnostic evaluation, and recommended treatments for such cases.

Keywords: Infectious Disease; Infective Endocarditis; HACEK; Bacteremia

Abbreviations

HACEK: Haemophilus Species, Aggregatibacter Species, Cardiobacterium Hominis, Eikenella Corrodens, Kingella Species; IE: Infective Endocarditis; TTE: Transthoracic Echocardiography; IDSA: Infectious Disease Society of America; AHA: American Heart Association; MRSA: Methicillin-resistant Staphylococcus Aureus and CAP: Community-acquired Pneumonia.

Introduction

Infective endocarditis (IE) carries significant risk for morbidity and mortality, with high rates of in-hospital mortality (20%), 1-year mortality (40%), peripheral embolization (23%), stroke (17%), and need for cardiac surgery (48%) [1,2]. Gram-positive bacteria account for approximately 80% of the cases of native valve IE, with staphylococcus and streptococci species accounting for the majority of these cases [3]. HACEK species, fungi, polymicrobial infection, and aerobic gram-negative bacilli are isolated in 5-10% of IE cases [4].

Haemophilus influenzae is a facultatively anaerobic, nonmotile, pleomorphic, and capnophilic gram-negative coccobacillus [5,6]. Encapsulated H. influenzae species are classified into serotypes a-f. Serotype b (Hib) has historically been considered the most invasive and infectious serotype but is now well controlled with routine childhood Hib vaccinations. An epidemiological study in Europe from 2007 to 2014 confirmed the efficacy of the vaccine with decreasing incidence of the Hib serotype, but emerging cases of infections with non-typeable H. influenzae, marking the non-typeable bacteria as the leading cause of invasive H. influenzae disease in European countries [7]. Similar findings are reported on Active Bacterial Core surveillance in the USA as part of the Emerging Infections Program from the Centers for Disease Control [8].

We report a case involving a 71-year-old woman with Haemophilus influenzae bacteremia and infective endocarditis. The case highlights the clinical features,

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diagnostic elements, and treatment plan associated with H. influenzae endocarditis, and emphasizes the importance of considering cardiac involvement with high-grade H. influenzae bacteremia.

Case Report

A 71-year-old female with a past medical history including hypertension, hyperlipidemia, cerebrovascular accident, transient ischemic attack, breast cancer status post bilateral mastectomy (approximately 30 years ago) followed by a recurrence and resection of lymph nodes in 2021, and left total knee arthroplasty was admitted to the hospital for evaluation of a 1-month history of generalized weakness and progressively worsening pain to her low back and bilateral lower extremities. She had received an epidural corticosteroid injection 2 weeks prior to onset of her presenting symptoms. Review of symptoms was remarkable for fever, fatigue, and sharp pain radiating from her low back to her bilateral posterior thighs.

On presentation, her temperature was 39°C/ 102.2°F, heart rate was 121 beats per minute, blood pressure was 148/71 mm Hg, and respiratory rate was 19 breaths per minute, with SpO2 of 93% on room air. Physical exam was remarkable for cachexia, generalized weakness, and decreased range of motion in her bilateral hips. No joint swelling, erythema, or tenderness was appreciated. Lung auscultation was remarkable for diminished bi-basilar breath sounds; no discernable cardiac murmurs could be auscultated.

Laboratory tests were notable for hemoglobin 11.3 g/dl, white blood cell count 10.1k/dl, serum glucose 235 mg/dl, ESR 46 mm/hr, and CRP 15.9 mg/dl. Chest X-ray on admission was largely unremarkable. The patient's history of malignancy and back pain initially raised clinical suspicion of metastatic disease to the spine. A CT abdomen/pelvis and MRI lumbar spine were obtained, which revealed bibasilar subsegmental lung atelectasis, multilevel degenerative spine changes at L4-S1 with mild disc bulging and bilateral foraminal stenosis; there was no evidence of metastatic disease or infectious process involving the spine.

Given her fever and elevated acute phase reactants, empiric antibiotic therapy was initiated with Ceftriaxone at a dose of 1 g IV daily. Two blood cultures were drawn, and after gram-negative rod bacteremia was discovered on both blood cultures, the Ceftriaxone dose was increased to 2 g IV daily. Her gram-negative rod bacteremia initially raised concern for a gastrointestinal infectious source, such as cholecystitis or ascending cholangitis. However, CT imaging of the abdomen and ultrasound imaging of the gallbladder did not show findings consistent with biliary tree infection (i.e. gallbladder wall thickening, pericholecystic edema, common bile duct dilation, abscess).

Because the source of infection remained elusive, transthoracic echocardiography (TTE) was ordered to evaluate for endocarditis, which revealed possible echogenic density in the posterior aortic valve leaflets measuring 0.7 x 0.9 cm. This prompted further evaluation with transesophageal echocardiography (TEE). TEE confirmed the diagnosis of vegetative infective endocarditis with echogenic density measuring 1.02 x 0.8 cm in the noncoronary cusp of the aortic valve leaflet (Figures 1-3). The gram-negative blood cultures subsequently resulted in H. influenzae, type I.

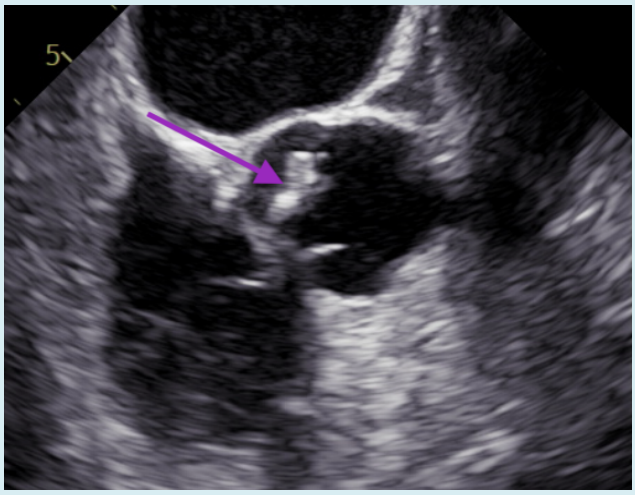


Figure 1: (left). Transesophageal echocardiogram images, short axis plane, showing open aortic valve vegetation on non-coronary cusp (purple arrows).

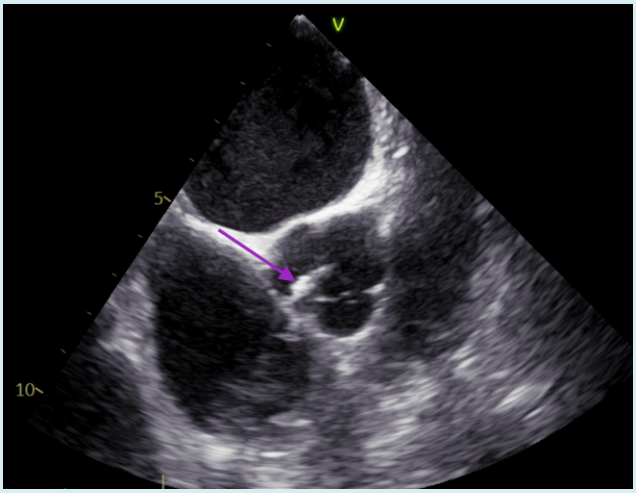


Figure 2: (right). Transesophageal Echocardiogram Images, Short Axis Plane, Showing Nearly Closed Aortic Valve Vegetation on Non-Coronary Cusp (Purple Arrows).

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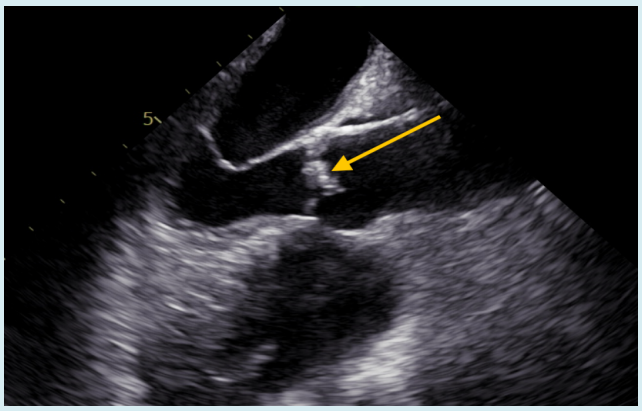


Figure 3: Transesophageal echocardiogram image, long axis view, showing aortic valve vegetation on non-coronary cusp (gold arrow).

Two days into her hospital course, her family mentioned that the patient had been experiencing recurrent sinus infections over a several-month time period prior to admission. CT sinus imaging was subsequently obtained, demonstrating moderate pansinusitis with mucosal thickening of the right maxillary sinus, ethmoid air cells, and the sphenoid sinuses (Figures 4-6). Our source of infection was ultimately determined to be H. influenzae bacterial sinusitis.

Treatment with Ceftriaxone 2 g IV daily was continued, with plans for a 4-week course of antibiotic therapy, per Infectious Disease Society of America (IDSA) and American Heart Association (AHA) guidelines for the treatment of H. influenzae infective endocarditis [2].

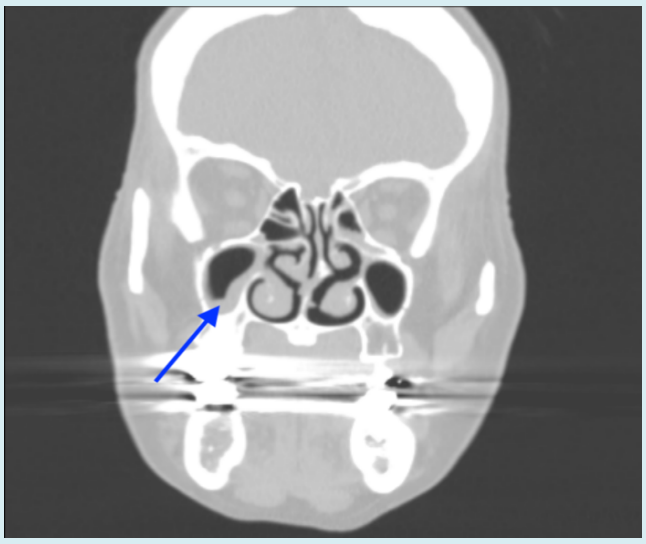


Figure 4: CT Sinus Image, Coronal Plane, Showing Right Maxillary Sinus Mucosal Thickening (Blue Arrow).

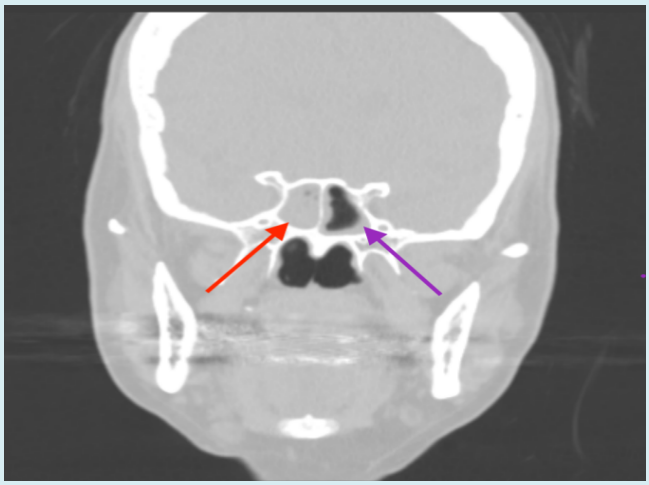


Figure 5: CT Sinus Image, Coronal Plane, Showing Right Sphenoid Sinus Mucosal Thickening/Occlusion (Red Arrow) and Left Sphenoid Sinus Mucosal Thickening (Purple Arrow).

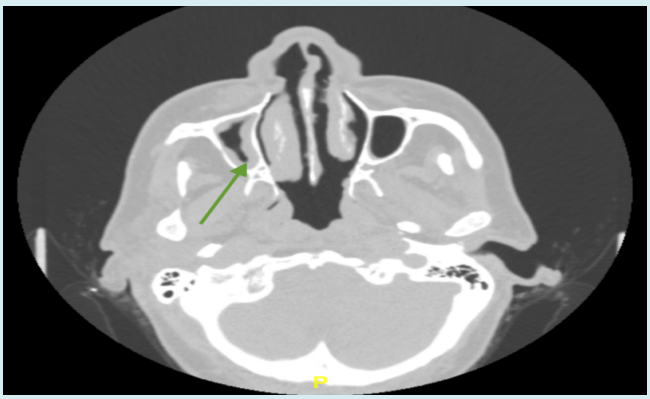


Figure 6: CT Sinus Image, Axial Plane, Showing Right Maxillary Sinus Mucosal Thickening (Green Arrow).

Follow-up and Outcome

The patient was discharged to a skilled nursing facility with a midline in place for administration of Ceftriaxone 2 g IV daily for a total treatment duration of 4 weeks. Repeat blood cultures after 3 days of antibiotic treatment were negative for bacterial growth, and the patient reported feeling improved. Repeat TTE was planned for 8 weeks after the last dose of ceftriaxone.

Discussion

Infective endocarditis (IE) is an infection of the endocardium involving native heart valves or implanted cardiac devices, including prosthetic valves, pacemakers, or defibrillators. The incidence of IE has been steadily

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rising, likely due to an aging population, and increased use of intravenous drug use. The United States has the highest incidence of IE in developed countries, with a rate of 15 per 100,000 people [9]. The mortality rate associated with IE remains high: in-hospital mortality ranges from 15-20%, while one-year mortality is nearly 40%, despite improvement in prompt prophylactic management and quicker diagnosis with improved ability to identify and characterize pathogenic organisms. However, mortality rates are higher among patients with IE caused by S. aureus compared to other pathogens (43.9% one-year mortality with S. aureus vs. 32.5% with others) [10]. The most common causative agents of IE are Staphylococci and Streptococci species [10,11]. Cardiac risk factors for IE include structural heart disease and valvular disease, and the presence of a cardiac implantable electronic device. In developing countries, rheumatic heart disease is also a common risk factor [11]. The most frequent non-cardiac risk factors include hemodialysis, IV drug use, cancer, immunosuppression, and diabetes mellitus [12].

Endocarditis associated with gram-negative bacteria is traditionally uncommon. Notably, HACEK species, which are part of normal flora strictly in the human oropharynx and gastrointestinal tract, only account for 1-3% of IE cases [6,13]. These pathogens often cause periodontitis, lower respiratory tract infections, meningitis, epiglottitis, cellulitis, pleuritis, septic arthritis, empyema, and bacteremia. Haemophilus parainfluenzae is the HACEK organism most commonly associated with infective endocarditis, while Haemophilus influenzae involvement is rare, accounting for 0.8 to 1.3% of all reported endocarditis [14].

Fever is the most common finding in endocarditis, followed by a new onset cardiac murmur [15]. Utilization of Duke's criteria is the gold standard in clinical diagnosis of IE (Tables 1 & 2). Duke's criteria require blood cultures, echocardiographic imaging, and other extracardiac manifestations, such as embolic and immunological findings. Cases in which transthoracic echocardiogram is insufficient, transesophageal imaging is required to evaluate the condition and function of the cardiac valves. Also of note, new technology such as Matrix-Assisted Laser Desorption/ Ionization Time of Flight Mass Spectrometry [16] has improved our ability to rapidly and accurately identify causative agents, including HACEK species. These species can be challenging to culture in a timely manner due to their traditionally slow growth.

Our patient's clinical presentation satisfied 2 major and 1 minor Duke criteria for infective endocarditis of a native valve: 2 positive blood cultures of H. influenzae (major criteria), positive echocardiogram findings (major criteria), and fever >38°C (minor criteria). Because she met 2 major criteria, we were able to confidently diagnose her with "definite IE."

Definite IE	Possible IE	Rejected IE
Pathological Criteria • Microorganisms demonstrated by culture or histological examination of a vegetation, a vegetation that has embolized, or an intracardiac abscess specimen OR • Pathological lesions: Vegetation or intracardiac abscess confirmed by histological examination showing active endocarditis	• 1 major criterion and 1 minor criterion OR • 3 minor criteria	• Confirmed alternative diagnosis explaining evidence of IE OR • Resolution of IE syndrome with antibiotic therapy for <4 d OR • No pathological evidence of IE at surgery or autopsy, with antibiotic therapy for <4 d OR • Does not meet criteria for possible IE as listed in this table
Clinical Criteria 2 major criteria OR • 1 major criterion + 3 minor criteria OR • 5 minor criteria		

Table 1: Diagnosis of Infective Endocarditis Using Modified Duke Criteria.

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Major criteria	Minor criteria
A. • Two separate positive blood cultures with typical microorganisms for IE (Typical microorganisms consistent with IE: Viridans streptococci, Streptococcus bovis, HACEK group, S. aureus; or community-acquired enterococci, in the absence of a primary focus) OR • Single positive blood culture result for Coxiella burnetii or antiphase I IgG antibody titer >1:800	• Predisposing heart condition or injection drug use • Fever (>38°C / 100.4°F) • Vascular phenomena: major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhages, Janeway lesions • Immunological phenomena: glomerulonephritis, Osler's nodes, Roth's spots, positive rheumatoid factor • Microbiological evidence: positive blood culture that does not meet a major criterion, or serological evidence of active infection with organism consistent with IE
B. Evidence of endocardial involvement • Echocardiogram positive for oscillating intracardiac mass on valve or supporting structures, in the path of regurgitant jets, or on implanted material in the absence of an alternative anatomic explanation OR • Abscess OR • New partial prosthetic valve dehiscence OR • New valvular regurgitation	

Table 2: Major and Minor Criteria for Modified Duke Criteria.

According to the Infectious Disease Society of America and the American Heart Association, the current guidelines for the treatment of H. influenzae endocarditis include intravenous Ceftriaxone, 2 grams daily for at least 4 weeks for native valve involvement, and 6 weeks for prosthetic valves. While patients with mild symptoms can wait for their blood culture results, acutely ill patients require empiric therapy with Vancomycin to cover methicillin-resistant staphylococcus aureus (MRSA). During treatment, it is reasonable to obtain repeat sets of blood cultures from different sites every 24-48 hours until the bloodstream infection is cleared. Additionally, HACEK organisms are considered ampicillin-resistant unless indicated otherwise on microbiology sensitivity testing [15]. In cases of cephalosporin allergy/intolerance, fluoroquinolones (ciprofloxacin, levofloxacin, or moxifloxacin) may be considered, with recommendations from infectious disease consultants. Surgical intervention (i.e. valve repair or replacement) is considered for patients with new-onset heart failure, annular or aortic abscess, recurrent emboli while on appropriate therapy, fungal endocarditis, or persistent infection (more than 5-7 days) [2,3]. We have also collected and reviewed nine case reports describing cases of H. influenzae infective endocarditis (Table 3).

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Author	Year	Age	Sex	Race	Valve	Presenting symptoms	Bacteremia	Complications	Antibiotic used	PMH
Harama, et al. [16]	2020	5	M	N/A	Mitral	fever (38.5 °C), right ear pain, abdominal pain	(+)	None specified	Ceftriaxone (100 mg/kg/day) for 6 weeks	Congenital hydrocephalus
Doyle, et al. [17]	2021	55	M	N/A	Tricuspid	fever, dyspnea, night sweats, productive cough	(+)	Tricuspid regurgitation	Ceftriaxone 2 g IV daily for 4 weeks	None
Kwok, et al. [18]	2022	69	M	N/A	Aortic	fever and chills	(+)	Aortic valve replacement and 2-vessel CABG	Ceftriaxone for 6 weeks	CAD s/p stent placement, Nicotine dependence, HTN, HLD, emphysema
Bunde, et al. [19]	2022	81	F	N/A	Aortic	fever, myalgias, chills	(+)	Ruptured popliteal aneurysm– Mycotic aneurysm	Ceftriaxone IV daily for 4 weeks	Rheumatoid arthritis and DVT
Dylewski, et al. [20]	2020	65	F	N/A	Mitral	fever, chills, right ankle, and foot pain	(+)	Septic emboli and destruction of mitral valve	Ceftriaxone 2g IV q12 hours for 1 week, then daily for 4 weeks	None specified
Kunce, et al. [21]	2022	35	M	N/A	Aortic and Mitral	fever, weight loss, fatigue, night sweats	(+)	AV and MV replacement due to perforation of native valves, septic emboli–bilateral cerebral and cerebellar microhemorrhage	None specified	None specified
Patel, et al. [22]	2023	49	M	N/A	Mitral	hemoptysis, nausea, vomiting, diarrhea	(+)	Septic shock, MV replacement	Ceftriaxone IV 2 g daily for 6 weeks	IVDU, nicotine dependence
Sasi prapha, et al. [23]	2018	34	M	N/A	Mitral	fever and myalgias	(+)	Left atrial myxoma infection	Ceftriaxone IV 2 g daily for 4 weeks	None
Headrick, et al. [24]	2018	4	M	AA	Mitral	Altered mental status, abdominal pain, and vomiting	(+)	disseminated H. influenzae type A: HF, HAP, septic vs reactive arthritis	Ampicillin and Ceftazidime	Undiagnosed asplenic heterotaxy

Table 3: Summary of Nine Clinical Reports of H. Influenzae Infective Endocarditis.

In seven of the nine cases, fever was a presenting symptom, as in our case. Our case involved endocarditis of the aortic valve in isolation, a finding in only two of the nine other cases we found. Common differential diagnoses for IE include acute coronary syndrome, acute heart failure, pulmonary embolism, pneumonia, underlying malignancy with marantic endocarditis, and Libman Sacks endocarditis. Doyle D, et. al. [17] reported a case in which a patient was initially misdiagnosed with community-acquired pneumonia (CAP) instead of right-sided infective endocarditis due to symptoms of fever, shortness of breath, and coughing [17]. It is not hard to imagine how this could happen, as there can be an overlap between symptoms associated with CAP and IE. Delay in the correct diagnosis and prompt

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treatment may complicate the hospital course with multi-organ involvement, such as valvular insufficiency (the most common cause of death), embolic stroke, renal infarction, vertebral osteomyelitis, or musculoskeletal abscess [18-24]. In the cases we reviewed, valvular damage was one of the most frequent complications, especially involving the left-sided valves [3,25].

Conclusion

This case report highlights the importance of considering IE in patients with H. influenzae bacteremia, despite having no underlying cardiac history or history of intravenous drug use. Symptoms vary by disease extent and severity [24,26]. Previously published case reports discuss instances of IE with symptoms ranging from mild fever and fatigue to aortic insufficiency and cardiogenic shock [18,21]. Given our diverse patient population, increasing life expectancy, and high rates of risk factors associated with IE in the United States, it is important to be familiar with the common presenting symptoms, appropriate diagnostic tools, and guideline-directed treatments involved with IE, including common microbial pathogens, such as staphylococcus and streptococcus, and less common ones, such as Haemophilus influenzae. Declarations: This research was not supported by any organization. The views expressed in this publication represent those of the author(s) and do not necessarily represent the official views of Texas tech University health Sciences Center.

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