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A CASE OF INFECTIVE ENDOCARDITIS AND PULMONARY SEPTIC EMBOLISM IN A 10-YEAR-OLD BOY

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Abstract:

Infective endocarditis (IE) is an uncommon infection of the cardiac endothelium. IE includes both acute bacterial endocarditis and sub-acute bacterial endocarditis (SBE). IE is an old problem in a new cover. Infective endocarditis (IE) in children can result in significant morbidity and mortality. A severe complication of infective endocarditis (IE) is organ embolization such as septic pulmonary embolism (SPE). The aim of this paper is to report an EI case in a 10-years boy with fatal outcome due to septic pulmonary embolism.

Keywords: Infective endocarditis, Septic pulmonary embolism, Pediatric

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Case Report:

A 10-year-old previously healthy boy referred to the hospital with a three-week history of fever, headache, left flank pain and chills. The patient had been admitted to the hospital for 24 days and was thought to be related to a viral illness. The patient had no chronic diseases. He was born full-term with no complications and his growth and development were age-appropriate. On admission he had a temperature of 38.5°C, a pulse of 128 beats/minute, a blood pressure of 100/65 mmHg, a respiratory rate of 30 breaths/ minute, and an oxygen saturation 98% in room air. His physical examination was significant for an alert and oriented child who was mildly dehydrated. He had a regular heart rate and rhyt hm, and third heart sounds with 3/6 systolic murmurs.

His lungs were clear to auscultation bilaterally, and his abdomen was soft, non-distended, and non-tender without hepatosplenomegaly. He had a normal dentition with tooth decay. He had no skin rash or petechiae. The rest of his examination findings were unremarkable. A complete blood count revealed a normal white blood cell count of $10.1x10^3$ /mm^3, anemia with a hemoglobin and hematocrit of 8.5 g/dl and 26% respectively, and platelets $360x10^3$/mm. Electrolytes showed a sodium of 139 mmol/L, a potassium of 4.4 mmol/L, a blood urea nitrogen of 35 mg/dl, a creatinine of 0.31 mg/dl. His C-reactive protein (CRP) was positive and ASTO 400 IU/ml. His urinalysis showed no hematuria. Three blood cultures were again negative (blood cultures at our institute are routinely cultured for 7 days). The blood culture remained negative for bacterial growth after he was started azytromycine from previous hospital. Abdominal-ultrasound shown bilateral nephritis.

Transthoracic echocardiography (TTE) was obtained due to concern of infective endocarditis (IE). This showed a-vegetation on the aorta valve, which confirmed a 3x3 mm vegetation below right coronary cusp of aorta valve and resultant mild to moderate aorta valve regurgitation without other valvular abnormalities. No congenital defect or patent foramen ovale and patent ductus arteriosus were demonstrated. We thought

that transthoracal echocardiography (TTE) was enough diagnostic and clear so transesophageal echocardiography (TEE) was not performed.

Since we had only a positive CRP and elevation of ASTO, but no positive culture, there is no way to perform an antibiogram. Initial empirical therapy Intravenous ampicillin-sulbactam 300 mg/kg/d divided every 6 hours was given. During his entire hospital stay, he still had daily fevers (38.3-39.1 C). It was determined that his daily fevers were due to the large size of the vegetation and difficulty to eradicate the organism, not due to treatment failure or complications.

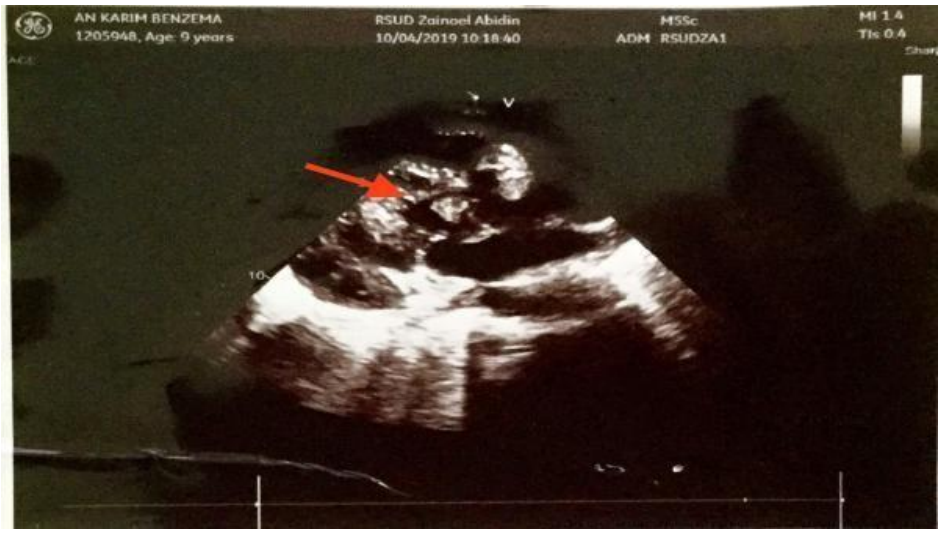

Figure 1. Vegetation below right coronary cusp of aorta valve

On the fourth day of hospitalization, the patient presented with sudden shortness of breath, intense abdominal and chest pain. He had a temperature of 36.7°C, a pulse of 130 beats/minute, a blood pressure of 80/50 mmHg, a respiratory rate of 42 breaths/ minute, and an oxygen saturation 82% in room air. On physical examination he was unwell-looking. His lungs were clear to auscultation bilaterally. Three hours later, patient went into respiratory failure and cardiac arrest and failed resuscitated. From the last presentation and physical examination, we assume the causal of his death was due to pulmonary septic embolism. Unluckily, we missed to perform simple test such as electrocardiogram to confirm the pulmonary embolism due to the rapid and sudden onset.

Discussion:

Infective endocarditis (IE) is an uncommon infection of the cardiac endothelium. Infective endocarditis (IE) in children can result in significant morbidity and mortality.[1-8] The epidemiology of IE in children has changed in recent years as congenital heart disease (CHD) turn into the essential predisposing factor from the developed world and rheumatic heart disease becomes much less frequent.[2,8] There is increased prevalence rate of IE in children with no history of heart disease likely due to elevated use of central venous catheters (CVC) especially in premature children with chronic illness. However, in up to 10% of cases, IE is seen in children with no known structural heart disease or other risk factors similar to this case.[1-3]

Viridans streptococci and staphylococcus aureus remain the most common pathogens responsible for pediatric IE with or without CHD.[1-8] On the other hand, a small percent (5%) is due to a group of fastidious gram-negative organisms known as HACEK (*Haemophilus species, Aggregatibacter species, Cardiobacterium hominis, Eikenella corrodens,* and *Kingella* species). Culture-negative IE, which is estimated to be in 5% of cases as well, has been characterized in patients with clinical and echocardiographic evidence of IE with blood culture yields no organisms. Bacteremia is believed to be the essential two factors in the pathogenesis of IE.[2] Damaged endothelium, occurs usually from turbulent blood flow in CHD, creates a sterile platelets-fibrin thrombus (nonbacterial thrombotic endocarditis). It is then the transient bacteremia (from dental procedure or daily activities like toothbrushing) that colonizes this thrombus and replicate to built the infected vegetation.[2,4,5,8]

Table 1. Principal Pathogenic Bacterial Agents[2]

	Series			
Organism	Johnson et al[8] (n=149)	Martin et al[11] (n=76)	Stockheim et al[13] (n=111)	Day et al[24] (n=632)
Years reviewed	1933–1972	1958–1992	1978–1996	2000–2003
Viridans group streptococci	43	38	32	20
Staphylococcus aureus	33	32	27	57
Coagulase-negative staphylococci	2	4	12	14
Streptococcus pneumoniae	3	4	7	1
HACEK	N/A	5	4	N/A
Enterococcus species	N/A	7	4	N/A
Culture negative	6	7	5	N/A

Values indicate percentage of patients in the series.

HACEK indicates *Haemophilus* species, *Aggregatibacter* species, *Cardiobacterium hominis*, *Eikenella corrodens*, and *Kingella* species; and N/A, not applicable.

The clinical presentation of IE has been well established as sub-acute and acute presentation. Sub-acute IE presents as prolonged low-grade fever for weeks or even months with other symptoms like fatigue, chills, myalgia, and weight loss. While Acute IE on the other hand presents with high fever and rapid deterioration if not recognized in a timely manner. Patient might have mixed features similar to this case as patient presented acutely, but was clinically stable overall and did not become worse.[2,4]

The modifications of the Duke criteria for diagnosis of IE have been validated to be helpful in diagnosing IE in children.[1-8] The patient was diagnosed with infective endocarditis based on modified Duke Criteria. The presented boy had one major criterion, which was evidence of endocarditis on echocardiography (right coronary cusp of aorta valve and resultant mild to moderate aorta valve regurgitation) along with three minor criteria: (1) fever, and (2) bilateral nephritis (3) pulmonary emboli.

Table 2. Modifications of the Duke criteria[1-8]

DEFINITE INFECTIVE ENDOCARDITIS A. Pathological criteria 1. Microorganisms demonstrated by culture or histologic examination of a vegetation, a vegetation that has embolized, or an intracardiac abscess specimen *or* 2. Pathological lesions; vegetation or intracardiac abscess confirmed by histologic examination showing active endocarditis B. Clinical criteria 1. Two major criteria *or* 2. One major criterion and three minor criteria *or* 3. Five minor criteria
POSSIBLE INFECTIVE ENDOCARDITIS 1. One major criterion and one minor criterion *or* 2. Three minor criteria
REJECTED 1. Firm alternative diagnosis explaining evidence of IE *or* 2. Resolution of IE syndrome with antibiotic therapy for <4 days *or* 3. No pathological evidence of IE at surgery or autopsy with antibiotic therapy for <4 days *or* 4. Does not meet criteria for possible IE as above

Three blood cultures taken at his presentation and during the course of hospitalization did not grow any pathogen. According to literature, the rate of culture-negative endocarditis varies with different studies, ranging from 2.5% to 31%. In patients with acute IE who are severely ill and unstable, 3 separate vena-punctures for blood cultures should be conducted over a short period such as 1 to 2 hours and empirical antibiotic therapy initiated as soon as possible. That was the reason why we could not delay the empirical antibiotic therapy initiated for the patient. Laboratory abnormalities that can be seen in IE are elevated acute phase reactants (erythrocyte sedimentation rate and C-reactive protein), anemia, thrombocytopenia, hematuria, and positive rheumatoid factor.[2]

Cardiac echocardiography is fundamental for the diagnosis and monitoring vegetation size and cardiac function.[2-5] That is important to concern that lack of vegetation on echocardiography does not preclude IE.[2] Echocardiography is primary for serial studies in a patient with known or suspected IE. Not only is objective documentation of progressive changes in cardiac function, but also identification of other important complications of IE by echocardiography can have immediate reevaluation and decision making with regard to early surgical intervention, which can be crucial for better outcome.[2-5] In contrast to the condition in adults, transthoracic echocardiography (TTE) is generally adequate for children (particularly if they weigh <60 kg) to completely comprehend cardiovascular findings in definite or presumptive IE. However, transesophageal echocardiography (TEE) is superior to TTE. Despite the fact that TTE has been shown an adequate notice endocarditis in young children (up to 97% sensitivity), for those >10 years of age and weighing >60 kg, the TEE, as in adults, has been shown to be a more sensitive tool and it can be helpful to perform TEE in patients who are at high risk for this complication.[2]

A severe complication of infective endocarditis (IE) is organ embolization such as septic pulmonary embolism (SPE).[2] The characteristics of SPE at presentation are nonspecific and usually go unrecognized by clinicians. The underlying clinical highlights change from a low-grade fever and respiratory symptoms, including cough, hemoptysis, chest pain, abdominal pain, purulent sputum, and dyspnea. Most SPE patients have similar presentations with pneumonia. An SPE diagnosis is therefore, frequently delayed, which consequently influences the outcome.[2,8] Most cardiac SPE was caused by right-sided IE, but several cases also exhibited left-sided IE. Patients with infective endocarditis in a bicuspid aortic valve had a higher incidence of periannular complications. Periannular abscess may cause right atrial vegetation, which may be the cause of cardiac SPE in cases with aortic valve vegetations.[2,9,10] The anticoagulation therapy is important for treating non-infective pulmonary embolism, yet it is not typically used in cases of septic embolization due to the increased risk of bleeding in the area of the infected embolus. The successful treatment of IE and cardiac SPE depends on the eradication of microbes by antimicrobial drugs. American Heart Association published the recommendation of antibiotic treatments for pediatric infective endocarditis in 2015 (table 3 and table 4).[2]

Conclusion:

Infective endocarditis (IE) in children can result in significant morbidity and mortality. A severe complication of infective endocarditis (IE) is organ embolization such as septic pulmonary embolism (SPE). The successful treatment of IE and cardiac SPE relies upon the destruction of microorganisms by appropriate antimicrobial medications.

Table 3. Recommended Antibiotic Treatments for Pediatric Infective Endocarditis [2]

Organism/Condition	Recommended Antibiotic Drug/Daily Antibiotic Dose†	Alternative Antibiotic Drug Choice
Unknown agent (initial empirical therapy or culture-negative endocarditis, generally after at least 48 h of attempting to culture the causative organism except in severely ill children)		
Native valve (community acquired) or "late" prosthetic valve (>1 y after surgery) infection	Ampicillin/sulbactam plus gentamicin	Vancomycin (plus gentamicin)
	With or without vancomycin	
	For prosthetic valve endocarditis, add rifampin	
	Ampicillin-sulbactam 200–300 mg·kg^{-1}·d^{-1} IV divided every 4–6 h up to 12 g daily	Vancomycin 60 mg·kg^{-1}·d^{-1} IV divided every 6 h up to 2 g
	Gentamicin 3–6 mg·kg^{-1}·d^{-1} IV divided every 8 h. Adults: 3–5 mg·kg^{-1}·d^{-1}	Gentamicin 3–6 mg·kg^{-1}·d^{-1} IV divided every 8 h. Adults: 3–5 mg·kg^{-1}·d^{-1}
	Rifampin 15–20 mg·kg^{-1}·d^{-1} divided every 12 h up to 600 mg	
	Vancomycin 60 mg·kg^{-1}·d^{-1} IV every 6 h up to 2 g	
Nosocomial endocarditis associated with vascular cannulae or "early" prosthetic valve endocarditis (≤1 y after surgery)	Vancomycin plus gentamicin (± rifampin if prosthetic material present)	?
	Plus cefepime or ceftazidime†	
	Vancomycin 60 mg·kg^{-1}·d^{-1} IV divided every 6 h up to 2 g	
	Gentamicin 3–6 mg·kg^{-1}·d^{-1} IV divided every 8 h Adults: 3–5 mg·kg^{-1}·d^{-1}	
	Rifampin 20 mg·kg^{-1}·d^{-1} divided every 8 h up to 900 mg/d	
	Cefepime 100-150 mg·kg^{-1}·d^{-1} divided every 8–12 h up to 6 g/d	
	Ceftazidime 100–150 mg·kg^{-1}·d^{-1} IV divided every 8 h up to 2–4 g daily‡	
Streptococci		
Highly susceptible to penicillin G (MBC ≤0.1 μg/mL); includes most viridans streptococci, groups A, B, C, G nonenterococcal, group D streptococci (*S bovis*, *S equinus*)	Penicillin G or ceftriaxone	Vancomycin or first-generation cephalosporin or ceftriaxone
	Penicillin G 200 000–300 000 U·kg^{-1}·d^{-1} IV divided every 4 h up to 12–24 million U daily	Vancomycin 40 mg·kg^{-1}·d^{-1} IV divided every 8–12 h up to 2 g daily
	Ceftriaxone 100 mg·kg^{-1}·d^{-1} IV divided every 12 h or 80 mg·kg^{-1}·d^{-1} IV every 24 h up to 4 g daily (if over 2 g, divide BID)	Cefazolin 100 mg·kg^{-1}·d^{-1} IV divided every 8 h up to 12 g daily
		Ceftriaxone 100 mg·kg^{-1}·d^{-1} IV divided every 12 h or 80 mg·kg^{-1}·d^{-1} IV every 24 h up to 4 g daily
Relatively resistant to penicillin (MBC ≥0.2 μg/mL; includes enterococci and less-susceptible viridans streptococci)	Penicillin G (or ampicillin) plus gentamicin (for first 2 wk, or whole course for enterococci)	Vancomycin plus gentamicin for enterococci
	Penicillin G 200 000–300 000 U·kg^{-1}·d^{-1} IV divided every 4 h up to 12–24 million U daily	Ampicillin plus ceftriaxone (for aminoglycoside-resistant enterococci or aminoglycoside-intolerant patient)
	Ampicillin 200–300 mg·kg^{-1}·d^{-1} IV divided every 4–6 h up to 12 g daily	Ceftriaxone plus gentamicin (not for enterococcal endocarditis)
	Gentamicin 3–6 mg·kg^{-1}·d^{-1} IV divided every 8 h. Adults: 3–5 mg·kg^{-1}·d^{-1}	Vancomycin 40 mg·kg^{-1}·d^{-1} IV divided every 8–12 h up to 2 g daily
		Gentamicin 3–6 mg·kg^{-1}·d^{-1} IV divided every 8 h. Adults: 3–5 mg·kg^{-1}·d^{-1}
		Ceftriaxone 100 mg·kg^{-1}·d^{-1} IV divided every 12 h or 80 mg·kg^{-1}·d^{-1} IV every 24 h up to 4 g daily
		Ampicillin 200–300 mg·kg^{-1}·d^{-1} IV divided every 4–6 h up to 12 g daily
Staphylococci (*S aureus* or coagulase-negative staphylococci)		
Susceptible to ≤1 μg/mL penicillin G (rare)	Penicillin G	Oxacillin or nafcillin or first-generation cephalosporin or vancomycin
	Penicillin G 200 000–300 000 U·kg^{-1}·d^{-1} IV divided every 4 h up to 12–24 million U daily	Oxacillin or nafcillin 200 mg·kg^{-1}·d^{-1} IV divided every 4–6 h up to 12 g/d
		Cefazolin 100 mg·kg^{-1}·d^{-1} IV divided every 8 h up to 12 g daily
		Vancomycin 40 mg·kg^{-1}·d^{-1} IV divided every 8–12 h up to 2 g daily

(*Continued*)

Table 3. Continue[2]

Organism/Condition	Recommended Antibiotic Drug/Daily Antibiotic Dose†	Alternative Antibiotic Drug Choice
Resistant to 0.1 μg/mL penicillin G	Penicillinase-resistant penicillin (oxacillin or nafcillin) ± gentamicin × 3–5 d	Vancomycin or a first-generation cephalosporin
	Oxacillin or nafcillin 200 mg·kg^{-1}·d^{-1} IV divided every 4–6 h up to 12 g/d	Cefazolin 100 mg·kg^{-1}·d^{-1} IV divided every 8 h up to 12 g daily
	Gentamicin 3–6 mg·kg^{-1}·d^{-1} IV divided every 8 h. Adults: 3–5 mg/kg	Vancomycin 40 mg·kg^{-1}·d^{-1} IV divided every 8–12 h up to 2 g daily for those highly allergic to β-lactam antibiotic drugs
Resistant to 4 μg/mL oxacillin (MRSA)	Vancomycin	Daptomycin for right-sided endocarditis, maybe for left-sided
	Vancomycin 40 mg·kg^{-1}·d^{-1} IV divided every 8–12 h up to 2 g daily	Daptomycin 6 mg/kg IV every 24 h; <6 y: 10 mg/kg
Vancomycin resistant or intolerant	Daptomycin	?
	Daptomycin 6 mg/kg IV every 24 h; <6 y: 10 mg/kg	
• For all staphylococci plus rifampin, plus gentamicin (for first 2 wk) if prosthetic material present		
Gram-negative enteric bacilli	Ceftazidime, cefepime, cefotaxime, or ceftriaxone plus gentamicin (or tobramycin or amikacin, depending on susceptibility)	A broad-spectrum penicillin plus gentamicin (or tobramycin or amikacin)
	Ceftazidime 100–150 mg·kg^{-1}·d^{-1} IV divided every 8 h up to 2–4 g daily‡	Piperacillin/tazobactam 240 mg·kg^{-1}·d^{-1} divided every 8 h up to 18 g daily
	Cefotaxime 200 mg·kg^{-1}·d^{-1} IV divided every 6 h up to 12 g daily	Gentamicin or tobramycin 3–6 mg·kg^{-1}·d^{-1} IV divided every 8 h. Adults: 3–5 mg·kg^{-1}·d^{-1}
	Ceftriaxone 100 mg·kg^{-1}·d^{-1} IV divided every 12 h or 80 mg·kg^{-1}·d^{-1} IV every 24 h up to 4 g daily	Amikacin 15 mg·kg^{-1}·d^{-1} IV divided every 8–12 h up to 15 mg·kg^{-1}·d^{-1}
	Gentamicin or tobramycin 3–6 mg·kg^{-1}·d^{-1} IV divided every 8 h. Adults: 3–5 mg·kg^{-1}·d^{-1}	
	Amikacin 15 mg·kg^{-1}·d^{-1} IV divided every 8–12 h up to 15 mg·kg^{-1}·d^{-1}	
HACEK group	Ceftriaxone or cefotaxime	Ampicillin (for susceptible organisms) plus aminoglycoside
	or	
	Ampicillin-sulbactam	Ampicillin 200–300 mg·kg^{-1}·d^{-1} IV divided every 4–6 h up to 12 g daily
	Cefotaxime 200 mg·kg^{-1}·d^{-1} IV divided every 6 h up to 12 g daily	Gentamicin or tobramycin 3–6 mg·kg^{-1}·d^{-1} IV divided every 8 h. Adults: 3–5 mg·kg^{-1}·d^{-1}
	Ceftriaxone 100 mg·kg^{-1}·d^{-1} IV divided every 12 h or 80 mg·kg^{-1}·d^{-1} IV every 24 h up to 4 g daily	
	Ampicillin-sulbactam 200–300 mg·kg^{-1}·d^{-1} IV divided every 4–6 h up to 12 g daily	Amikacin 15 mg·kg^{-1}·d^{-1} IV divided every 8–12 h up to 15 mg·kg^{-1}·d^{-1}
Fungi *Candida* spp, *Aspergillus* spp	Surgical resection plus amphotericin B with or without flucytosine	Amphotericin B followed by imidazole (eg, fluconazole, itraconazole, voriconazole) suppression if surgery cannot be performed§
	Amphotericin B 1 mg·kg^{-1}·d^{-1} IV administered over 3–4 h	
	Flucytosine 150 mg·kg^{-1}·d^{-1} orally divided every 6 h	Amphotericin B 1 mg·kg^{-1}·d^{-1} IV administered over 3–4 h
	Amphotericin liposomal/lipid-associated (3 formulations) 3–5 mg·kg^{-1}·d^{-1} in a single dose daily	Amphotericin liposomal/lipid-associated 3–5 mg·kg^{-1}·d^{-1} in a single dose daily

BID indicates twice per day; HACEK, *Haemophilus* species, *Aggregatibacter* species, *Cardiobacterium hominis*, *Eikenella corrodens*, and *Kingella* species; IV, intravenously; MBC, minimum bactericidal concentration; and MRSA, methicillin-resistant *Staphylococcus aureus* (includes resistance to oxacillin, nafcillin, and cephalosporins).

Treatment is generally for 4 to 6 weeks. See Table 8. Longer therapy may be required for recurrent endocarditis, prosthetic valve endocarditis, endocarditis attributable to uncommon species.

*As discussed in the text, these recommendations are based on consensus of experts and not experimental comparative studies (*Class IIa; Level of Evidence C*).

†Doses for neonates and infants not included. For cases of infective endocarditis in infants, consult infectious diseases and pediatric clinical pharmacists with special expertise in neonatal and infant clinical pharmacology.

‡Maximum daily dose or adult dose should not be exceeded on a per kilogram basis when treating children.

§Possibly lifelong suppression if no surgery or relapse after surgery.

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