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# Infective endocarditis: still a challenge for paediatricians and cardiologists – a case report

## Dziecko z infekcyjnym zapaleniem wsierdzia wciąż wyzwaniem dla pediatry i kardiologa – opis przypadku

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Abstract

Infective endocarditis is an inflammatory disease affecting the innermost layer of the heart wall, which continues to pose a significant medical challenge for physicians. Due to the dynamic development of cardiology and cardiac surgery, the growing number of surgical procedures performed, and increasing bacterial resistance to antibiotics, there is an increase in the incidence of this disease. This article describes the difficulties encountered in diagnosing infective endocarditis in a boy with trisomy 21 and a history of surgical correction of a congenital heart defect. Correct diagnosis and treatment based on the European Society of Cardiology guidelines helped to prevent serious complications for the boy.

**Keywords:** sepsis, infective endocarditis, *Streptococcus pyogenes*, trisomy 21

Streszczenie

Infekcyjne zapalenie wsierdzia to choroba o podłożu zapalnym, obejmująca najbardziej wewnętrzną warstwę ściany serca, wciąż stanowiąca poważne wyzwanie medyczne dla lekarzy. W związku z prężnym rozwojem kardiologii i kardiochirurgii, liczbą wykonywanych zabiegów chirurgicznych oraz rosnącą opornością bakterii na antybiotyki aktualnie obserwowany jest wzrost zachorowań na to schorzenie. Poniższy artykuł opisuje trudności diagnostyczne w rozpoznaniu infekcyjnego zapalenia wsierdzia u chłopca obciążonego trisomią 21. chromosomu oraz wrodzoną wadą serca operowaną w przeszłości. Ustalenie właściwego rozpoznania oraz włączenie leczenia w oparciu o wytyczne Europejskiego Towarzystwa Kardiologicznego umożliwiło uniknięcie u chłopca ciężkich powikłań związanych z chorobą.

**Słowa kluczowe:** posocznica, infekcyjne zapalenie wsierdzia, *Streptococcus pyogenes*, trisomia 21. chromosomu

## INTRODUCTION

Infective endocarditis (IE) is an ongoing infection of the valves, atria, ventricles, endothelium of blood vessels, or foreign bodies within the heart. In the paediatric population, the incidence of IE is 0.43–0.69 cases per 100,000 children per year[1].

Currently, the incidence of IE in the paediatric population is increasing, primarily due to the development of cardiology and cardiac surgery, and the resulting rise in the number of invasive diagnostic tests and heart defect repair procedures[2].

The most common pathogens causing IE in children are Gram-positive bacteria: *Streptococcus viridians* and *Streptococcus aureus*, less frequently coagulase-negative *Staphylococcus aureus*, *Streptococcus pneumoniae*, *Enterococcus* sp. and Gram-negative bacteria constituting the physiological flora of the oral cavity, including *Haemophilus*, *Aggregatibacter actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella corrodens*, *Kingella kingae* (HACEK), as well as fungi from the *Aspergillus* and *Candida* groups[3,4].

The most important risk factors for IE in children include congenital heart defects, particularly cyanotic and left-sided lesions. It is estimated that patients with heart disease have a 15–140 times higher risk of developing IE compared to the general population[5,6].

Immune deficiencies, central venous catheters, cancer, hospitalisation in medical or long-term care facilities, extensive wounds, and skin infections also predispose to IE[1,2]. Furthermore, oral procedures, especially those requiring manipulation in the gingiva or periapical area of the teeth, carry a high risk of bacteraemia[2].

Diagnosing IE in children is challenging due to its non-specific clinical manifestations. Patients may present with symptoms such as fever, weakness, joint pain, weight loss, and cardiovascular symptoms, which often mimic other, more common infectious diseases[1]. The lack of characteristic signs can lead to diagnostic delays, which can increase the risk of complications. Therefore, paediatricians should remain clinically vigilant and consider IE in children with persistent fever, especially when standard treatment fails to improve the patient's condition.

This publication presents the case of a 6-year-old boy with infective endocarditis and comorbidities.

## CLINICAL CASE DESCRIPTION

A 6-year-old boy with trisomy 21 and a history of repair of a second-degree atrial septal defect (ASD II) and a ventricular septal defect (VSD) at 9 months of age was admitted to the paediatric ward with a 9-day history of fever accompanied by skin lesions, cough, runny nose, and lower limb pain. Before admission, the boy had received symptomatic treatment in an outpatient clinic.

On admission, the boy's general condition was moderate to severe. Physical examination revealed dysmorphic features characteristic of trisomy 21, numerous petechial haemorrhages on the trunk and extremities, extensive dental caries, a bright red throat, and swollen palatine tonsils without exudate. On auscultation, isolated rales were heard at the bases of both lungs. Furthermore, slight swelling and tenderness of the left ankle joint were noted. Vital signs on admission included body temperature 38.4°C, saturation 98%, heart rate 124/min, blood pressure 102/65 mm Hg. Owing to the epidemiological situation, an antigen test for influenza viruses, COVID-19, and respiratory syncytial virus was performed, yielding a positive result for influenza A.

Tests performed after admission revealed slight leucocytosis with a predominance of granulocytes in the blood count (white blood cells, WBC: 12.14 × $10^9$/L; neutrophils, NEU: 11.58 × $10^3$/μL), thrombocytopaenia (94 × $10^9$/L), elevated inflammatory markers (C-reactive protein, CRP: 9.7 mg/dL, procalcitonin, PCT: 2.46 ng/mL), hyponatraemia (133 mmol/L), hypokalaemia (3.0 mmol/L), increased levels of N-terminal prohormone of brain natriuretic peptide (1,158 pg/mL) and D-dimer (5.23 μg/mL), and a slight

| Parameter | Value on admission | Value on the 28th day of hospitalisation | Laboratory standard |
|---|---|---|---|
| WBC [×$10^9$/L] | 12.14 | 6.12 | 5.0–15.0 |
| NEU [×$10^3$/μL] | 11.58 | 2.00 | 1.5–8.5 |
| PLT [×$10^9$/L] | 94 | 338 | 219–497 |
| CRP [mg/dL] | 9.7 | 0.4 | <0.8 |
| PCT [ng/mL] | 2.46 | 0.1 | ≤0.046 |
| Sodium [mmol/L] | 133 | 140 | 134–143 |
| Potassium [mmol/L] | 3.0 | 4.5 | 3.3–4.6 |
| Glucose [mg/dL] | 189 | | <99.0 |
| NT-proBNP [pg/mL] | 1158.0 | | 5–391 |
| D-dimers [μg/mL] | 5.23 | | 0.00–0.50 |
| ASO [IU/mL] | 316 | | <100 |

**ASO** – anti-streptolysin O; **CRP** – C-reactive protein; **NEU** – neutrophils; **NT-proBNP** – N-terminal prohormone of brain natriuretic peptide; **PCT** – procalcitonin; **PLT** – platelet count; **WBC** – white blood cells.

*Tab. 1. Results of the patient's blood tests during hospitalisation*



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*Fig. 1. Patient's chest X-ray – AP view. Inflammatory densities seen bilaterally in the hila. Original material*

rise in anti-streptolysin O titre (316 IU/mL) (Tab. 1). Renal function markers remained within the normal range. Urinalysis and culture did not reveal any abnormalities. Electrocardiography showed intraventricular conduction disturbances in leads V1–V4. Chest X-ray revealed inflammatory densities in the hilar regions of both lungs (Fig. 1). Based on the correlation of clinical findings and additional tests, the initial diagnosis was influenza A complicated by pneumonia. After obtaining blood cultures, treatment was initiated with intravenous amoxicillin-clavulanic acid at a dose of 90 mg/kg/day and oral oseltamivir at 45 mg twice daily. Intravenous fluid therapy and pain management were also administered.

During hospitalisation, a skin lesion with erysipelas morphology was observed on the medial side of the left ankle joint. The diagnosis was confirmed by a consulting dermatologist (Fig. 2).

A soft tissue ultrasound performed due to progressive swelling of the left lower limb revealed an inflammatory reaction in the muscles with effusion in the tendon sheaths. Treatment was then supplemented with intravenous metronidazole at a dose of 7.5 mg/kg three times a day and subcutaneous enoxaparin (Clexane) at 10 mg daily (Tab. 2).

Despite the therapeutic approach, no improvement was observed. On the third day of hospitalisation, blood culture results revealed *Streptococcus pyogenes*, which was sensitive to the ongoing treatment. Given the patient's cardiac history and the aetiology of the infection, an echocardiogram was performed, showing vegetations on the aortic valve with thickening and mild regurgitation. Such lesions had not been observed in the boy's previous echocardiographic studies (Figs. 3 and 4).

The test results were discussed at a cardiology consultation at a reference centre, where the diagnosis of infective endocarditis was made according to the 2023 diagnostic criteria of the European Society of Cardiology (ESC). In view of the lack of clinical improvement, treatment modification (addition of an aminoglycoside) was recommended, together with repeat blood cultures and a follow-up echocardiogram after two weeks.

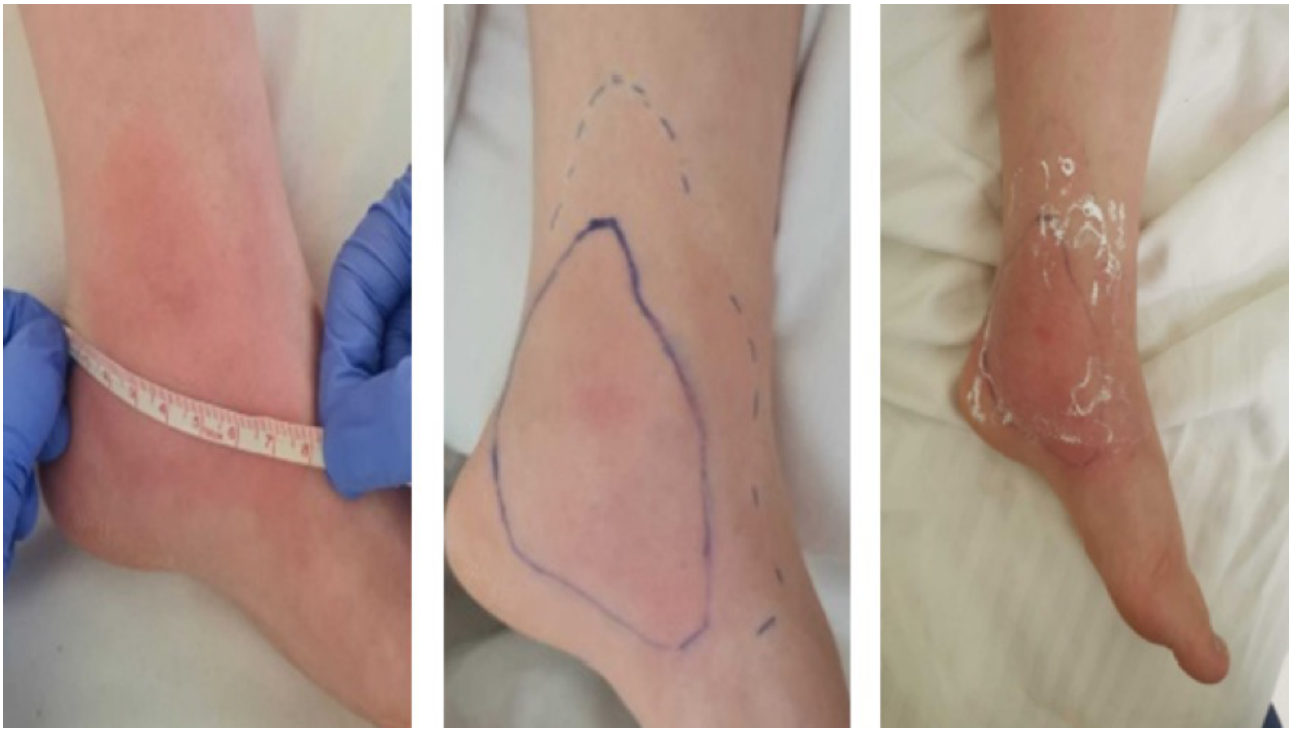

*Fig. 2. Erysipelas-like skin lesions over several days. Original material*



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| Treatment used | Treatment time [days] |
|---|---|
| Amoxicillin-clavulanic acid 2 g, once daily | 28 |
| Amikacin 7.5 mg/kg of body weight i.v., twice daily | 6 |
| Metronidazole 7.5 mg/kg of body weight i.v., three times daily | 9 |
| Oseltamivir 45 mg p.o., twice daily | 5 |
| Fluconazole 6 mg/kg of body weight i.v., once daily | 7 |
| Clexane 10 mg s.c., once daily | 26 |

*Tab. 2. Treatment administered to the patient*

During the subsequent days of hospitalisation, the boy's general condition improved and symptoms gradually subsided. Laboratory monitoring showed normalisation of abnormalities, including reductions in leucocytosis and granulocyte counts (WBC: 4.24 × $10^9$/L, NEU: 1.26 × $10^3$/μL, PLT: 416 × $10^9$/L), a decrease in inflammatory markers (CRP: 0.4 mg/dL, PCT: 0.1 ng/mL), correction of electrolyte imbalances (Na: 140 mmol/L, K: 4.5 mmol/L), and a negative follow-up blood culture (Tab. 2). Echocardiography performed after two weeks of treatment revealed no evidence of progression.

After completing the 28th day of hospitalisation, the child was discharged from the hospital at the parents' request. Treatment with amoxicillin-clavulanic acid was recommended for a total of five months, along with probiotics and antifungal medications. This long-term treatment was recommended after a second dermatological consultation due to erysipelas of the lower limb. Urgent dental treatment and continued cardiology care were also recommended. Furthermore, the parents were instructed to contact a physician if fever recurred, with emphasis on the patient's medical history.

## DISCUSSION

Infective endocarditis in children remains a significant diagnostic and therapeutic challenge, primarily due to its nonspecific clinical presentation. Symptoms often mimic other infectious diseases, which may delay diagnosis. In particular, persistent fever in patients receiving antibiotic therapy, especially in the presence of risk factors for infective endocarditis, should prompt further diagnostic evaluation (Tab. 3)[7].

The epidemiology of infective endocarditis in the paediatric population has changed over the past few decades. Historically, approximately 30–50% of paediatric IE cases were associated with rheumatic heart disease[7]. Currently, congenital heart disease is the main risk factor, accounting for approximately 75% of paediatric IE cases. Patients who have undergone cardiac surgery are also particularly exposed to developing the disease[1,2].

In light of the discussed case, it should be emphasised that children with trisomy 21 are also at increased risk of infective endocarditis, mainly due to the high prevalence of congenital heart defects in this population[8]. Furthermore, immune system abnormalities in the form of T and B lymphopaenia are observed in this group of patients[9]. Unfortunately, numerous studies have shown that parental awareness of infective endocarditis and its prevention in children with heart defects is often insufficient[10,11].

Oral health disorders, especially dental caries and periodontal disease, are also recognised as significant risk factors for IE[12]. Studies in the Polish population of children

*Fig. 3. Thickening of the aortic valve leaflets with bacterial vegetation on one leaflet. Original material*

*Fig. 4. Severe aortic valve regurgitation with lack of coaptation of the aortic valve leaflets. Original material*



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| **Cardiac risk factors of IE** |
|---|
| Congenital heart disease |
| Valvular heart disease |
| Artificial heart valve |
| Central venous or arterial catheter |
| Implantable cardiac electrotherapy device |
| Prior history of infective endocarditis |
| **Non-cardiac risk factors of IE** |
| Immunosuppression |
| Ventral venous catheter |
| Self-administered injection |
| Recent dental or surgical procedures |
| Recent hospitalisation |
| Haemodialysis therapy |

*Tab. 3. Risk factors for IE based on the 2023 ESC guidelines*

aged 3–7 years have shown that dental caries affects 65.6% of them[13]. This indicates the need for parental education regarding children's oral care.

In the paediatric population, the dominant pathogen of endocarditis remains *Staphylococcus aureus*, and in children with heart disease, *Streptococcus viridans*[1]. Furthermore, *Enterococcus faecalis* is currently considered a typical bacterium causing endocarditis, regardless of the source of infection[2].

In 2023, the ESC issued its latest guidelines for the management of endocarditis. Diagnosis of IE requires meeting specific criteria, including blood culture results and cardiac imaging, the presence of IE risk factors, fever, vascular signs suggestive of embolism, and immunologic symptoms[2]. Laboratory tests typically reveal elevated inflammatory markers, although their diagnostic value is limited due to low specificity[2].

Pharmacological treatment of infective endocarditis in children requires an individualised approach, taking into account the aetiology of the infection, comorbidities, the presence of implanted foreign materials, and the severity of the clinical course. Delays in diagnosis and treatment can lead to serious consequences, such as heart failure, abscess formation, systemic embolism, or sepsis[2].

According to the 2023 ESC guidelines, standard treatment for IE includes long-term antibiotic therapy adjusted to blood culture results, treatment of complications, and post-hospital care[1,2].

Empirical antibiotic therapy should be initiated immediately after obtaining blood cultures and then adjusted based on microbiological test results. For infections caused by *Staphylococcus aureus*, including methicillin-resistant strains (MRSA) vancomycin or daptomycin is recommended in combination with gentamicin and/or rifampicin for prosthesis-related infections. Streptococcal infections, especially those caused by *Streptococcus viridans* and *bovis* group streptococci, require penicillin G or ceftriaxone-based therapy, often in combination with gentamicin during the initial phase of treatment. For enterococcal infections, combination therapy with ampicillin and gentamicin is recommended, while for highly resistant strains, the addition of linezolid or daptomycin should be considered. Fungal infections, which are rare but particularly challenging, require the use of echinocandins (e.g. caspofungin) or amphotericin B, and in some cases, surgical removal of the affected structures. In some cases, cardiac surgery may be necessary[2].

The prognosis for children with infective endocarditis depends on the timing of diagnosis, treatment efficacy, and the presence of complications[2]. Early implementation of antibiotic treatment and timely surgical interventions increase the chances of survival and improved heart function. In paediatrics, the most common complication of infective endocarditis is septic embolism, which affects up to 40% of children. The mortality rate in the paediatric population ranges from 5 to 10%[6]. In the described case, the parents, despite being informed about possible complications, decided to discharge their child from the hospital at their own request before completing the treatment process. This highlights the need for intensified educational efforts aimed at caregivers of children at risk of infective endocarditis.

Following hospital treatment, regular follow-up visits are recommended, including echocardiography, usually after 1, 3, 6, and 12 months, as well as regular medical check-ups[1].

## SUMMARY

Infective endocarditis remains a rare condition, but due to advances in medicine and the increasing number of patients with heart disease, the number of patients diagnosed with IE is steadily increasing. Optimal diagnostic management requires an integrated approach encompassing clinical assessment, laboratory tests, and comprehensive imaging. Early diagnosis of the disease and its complications is crucial for effective treatment and improved prognosis in children with suspected infective endocarditis. Efforts should be made to increase awareness among both parents and physicians regarding risk factors contributing to the development of IE and potential preventive measures.

**Conflict of interest**

*The authors do not report any financial or personal connections with other persons or organisations which might negatively affect the content of this publication and/or claim authorship rights to this publication.*

**Author contribution**

*Original concept of study: KMP, MŚ. Collection, recording and/or compilation of data: KMP. Analysis and interpretation of data: KMP, LZ. Writing of manuscript: KMP, AB, MŚ. Critical review of manuscript: AB, MS. Final approval of manuscript: BK.*

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