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Prikaz slučaja – Case Report
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NECROTIZING PNEUMONIA CAUSED BY STREPTOCOCCUS PNEUMONIAE IN A FIVE-YEAR-OLD BOY – A CASE REPORT

NEKROTIČNA PNEUMONIJA PROUZROKOVANA STREPTOKOKUSOM PNEUMONIJE KOD PETOGODIŠNJEG DEČAKA – PRIKAZ SLUČAJA

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Summary

Introduction: Necrotizing pneumonia is one of the most serious complications of community-acquired pneumonia, caused by infection with particularly virulent bacteria. *Streptococcus pneumoniae* is the most common agent. It typically occurs in previously healthy children and leads to a prolonged clinical course despite appropriate antibiotic therapy.
Case outline: A five-year-old boy, who was regularly vaccinated and had no chronic illnesses, presented with a 14-day history of fever and cough, followed by difficulty breathing and right-sided abdominal pain. Oral antibiotic therapy (azithromycin) was started just before admission. As the symptoms persisted, radiological diagnostics were performed, and the diagnosis of pleuropneumonia was made, leading to hospitalization. Upon admission to the hospital an ultrasound and chest computed tomography were performed, confirming bilateral necrotizing pneumonia with massive right-sided pleural effusion, as well as thrombosis at the junction of the left jugular and subclavian vein. Treatment included parenteral antibiotic therapy, thoracic drainage with the administration of alteplase, and anticoagulant therapy. The pleural effusion, based on its biochemical characteristics, corresponded to empyema, and the culture was positive for *Streptococcus pneumoniae*. The disease required a prolonged hospitalization of 24 days with a positive clinical outcome.
Conclusion: The diagnosis of necrotizing pneumonia should be considered in any child with pneumonia that does not improve 72 hours after appropriate antibiotic therapy. Early diagnosis and timely treatment are crucial for a positive outcome, while regular vaccination remains the primary preventive measure.

Keywords: child, necrosis, pneumonia, complication, *Streptococcus pneumoniae*, prevention

Sažetak

Uvod: Nekrotična pneumonija je jedna od ozbiljnih komplikacija vanbolnički stečene pneumonije, koja je uzrokovana infekcijom posebno virulentnim bakterijama, od kojih je najčešća *Streptococcus pneumoniae*. Obično se javlja kod prethodno zdrave dece i dovodi do prolongiranog kliničkog toka i pored primene odgovarajuće antibiotske terapije.
Prikaz slučaja: Petogodišnji dečak, koji je redovno vakcinisan i bez hroničnih bolesti, razboleo se 14 dana pre prijema u bolnicu pojavom febrilnosti i kašlja, a potom i otežanog disanja i bola sa desne strane trbuha. Neposredno pred prijem započeta je peroralna terapija azitromicinom. Kako su se tegobe održavale, ambulantno je načinjena radiološka dijagnostika i postavljena dijagnoza pleuropneumonije, te je dete upućeno na hospitalno lečenje. Tokom hospitalizacije nakon načinjenog ultrazvuka i kompjuterizovane tomografije toraksa ustanovljena je obostrana nekrotična pneumonija uz masivni desnostrani pleuralni izliv, kao i tromboza na spoju leve jugularne i potključne vene. Sprovedeno je lečenje primenom parenteralne antibiotske terapije, torakalne drenaže uz primenu alteplaze, kao i antikoagulantne terapije. Pleuralni izliv je po svojim biohemijskim karakteristikama odgovarao empijemu, dok je kultura bila pozitivna na Streptococcus pneumoniae. Bolest je zahtevala prolongiranu hospitalizaciju u trajanju od 24 dana uz povoljan klinički tok.
Zaključak: Razvoj nekrotične pneumonije treba razmotriti kod svakog deteta sa pneumonijom koja ne pokazuje poboljšanje nakon 72 sata od primenjene adekvatne antibiotske terapije. Rana dijagnoza i primena pravovremene terapije su ključne za povoljan ishod lečenja, dok je redovna vakcinacija osnovna mera prevencije.

Ključne reči: dete, nekroza, pneumonija, komplikacije, *Streptococcus pneumoniae*, prevencija

INTRODUCTION

Necrotizing pneumonia (NP) is one of the most serious complications of community-acquired pneumonia (CAP). NP usually occurs in previously healthy children and leads to a prolonged clinical course despite the administration of appropriate antibiotic therapy (1).

The incidence of NP in children, although low, is increasing, representing 5% to 10% of cases of CAP, and it is a disease with a high mortality rate (2,3).

NP occurs when a pulmonary infection triggers inflammation that leads to damage to the lung parenchyma, necrosis, and liquefaction of lung tissue. This inflammation often results in damage to the pulmonary circulation and the formation of thrombi, reducing blood flow to the area affected by inflammation, which leads to uncontrolled bacterial replication and reduced antibiotic penetration (3).

NP are caused by particularly virulent bacteria, most commonly *Streptococcus pneumoniae* (especially serotype 3 and serogroup 19). Other, frequent causative agents, include *Staphylococcus aureus*, *Haemophilus influenzae*, and *Group*

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A Streptococcus. In sporadic cases, the causative agents are *Mycoplasma pneumoniae*, *Legionella*, *Aspergillus*, *Escherichia coli*, *Acinetobacter*, and anaerobes (3,4).

In addition to the specific pathogen, other clinical factors, such as co-infection with the influenza virus, can also influence the development of lung parenchyma necrosis. It is believed that this virus negatively affects macrophage activity, thereby reducing the immune response to bacterial infection. Patients with neutropenia are also at risk for a reduced phagocytic response, which predisposes them to the development of NP (3).

NP is usually presented with a severe clinical picture and sepsis. Treatment consists of parenteral antibiotic therapy, initially empirical, followed by targeted therapy against the specific pathogen if it is isolated. Prolonged antibiotic therapy for 2-4 weeks is recommended (3).

CASE OUTLINE

A five-year-old boy, who was regularly vaccinated (including the pneumococcal vaccine PCV 10) and had no chronic illnesses, presented with a 14-day history of fever and cough, followed by difficulty breathing and pain on the right side of the abdomen. Just before admission, oral antibiotic therapy (azithromycin) was introduced. As the symptoms persisted, a chest X-ray was performed in the outpatient setting, which showed an extensive bilateral pneumonia and pleural effusion at the right side (Figure 1). The boy was then referred to the tertiary care children's hospital. Upon admission, the child was tachypnoeic with reduced oxygen saturation (SpO2 88% on room air). Bilateral late inspiratory crackles were heard upon auscultation, as well as diminished breath sounds over the right hemithorax. Laboratory test revealed elevated parameters of the acute phase of inflammation (procalcitonin 2.86 mg/ml, CRP 176 mg/L, WBC 22 G/L, with a predominance of segmented neutrophils). The values of other blood components were normal. Albumin levels were low (24.39 g/L), D-dimer was elevated (>2500 ng/ml), serum immunoglobulin levels were normal, as well as other biochemical findings. The PCR from the nasopharyngeal swab for Influenza was positive. Blood culture was negative. Lung ultrasound was performed, revealing pleural effusion in the right costophrenic angle, measuring 18mm. The effusion extended along the lateral wall of the right hemithorax and posteriorly, with thickness (AP diameter) up to 34mm. It was very dense, but without detectable septa. A transhepatic approach to the right lung base revealed a large area of lung parenchymal consolidation with the presence of air bronchogram. In the left costophrenic sinus, a clear pleural effusion was detected, measuring 8 mm, along with a small area of lung parenchymal consolidation. In addition to ultrasound, CT scan was performed, which confirmed a massive pleural effusion on the right side, with bilateral signs of necrotizing pneumonia, as well as partial thrombosis at the junction of the left jugular and subclavian veins (Figure 2 and 3). The thrombosis was confirmed by ultrasound, while the echocardiography was normal. On the first day of hospitalization, a right-sided thoracostomy was performed, purulent fluid was obtained with cytological and biochemical characteristics of empyema (Figure 4). *Streptococcus pneumoniae* was isolated from the pleural fluid culture. Empirical therapy with ceftriaxone and clindamycin was started, and due to the confirmed influenza virus infection, oseltamivir was introduced. Due to the described jugular vein thrombosis, low-molecular-weight heparin therapy

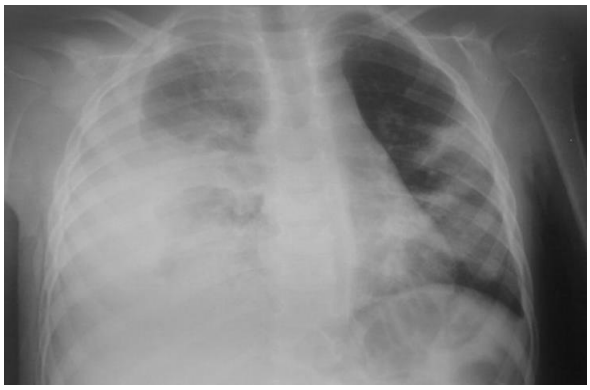

Figure 1. Chest X-ray on the day of admission - bilateral lung parenchymal consolidation, more extensive on the right, with complete opacification of the right costophrenic sinus and pleural effusion extending to the lung apex. There is a mediastinal shift towards the left hemithorax.
Slika 1. RTG pluća na dan prijema u bolnicu na kome se vide obostrana konsolidacija plućnog parenhima, izraženije desno, sa prisutnim potpunim zasenčenjem desnog FC sinusa uz liniju izliva koje se penje do plućnog apeksa.

was initiated. As the fever persisted with elevated acute-phase reactants, the antibiotic therapy was changed, with introduction of vancomycin on the 4th and meropenem on the 6th day of hospitalization. Despite the therapy, the fever persisted along with the persistence of the radiological findings and minimal drainage through the thoracic drain. In addition to the previous therapy, on the 9th day, intrathoracic alteplase was administered for 3 days. There was a good clinical response to the applied therapy, with significant evacuation of detritus and fibrin deposits, and improvement of radiological findings. The patient became afebrile from the 11th day of hospitalization, with a gradual normalization of the acute phase reactants, and from the 12th day no oxygen therapy was needed. The thoracic drain was removed on the 15th

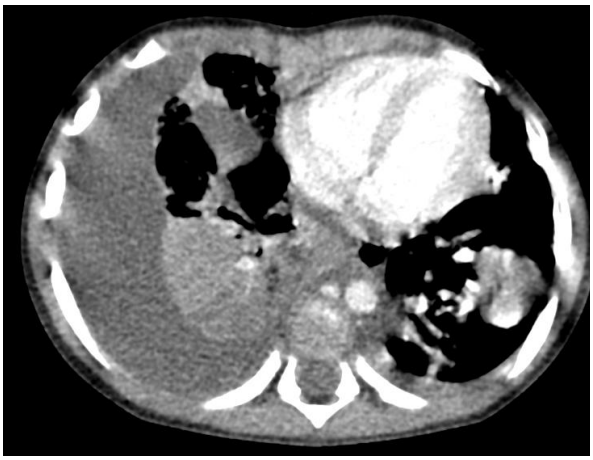

Figure 2. Contrast enhanced chest computed tomography (axial section) - massive pleural effusion at the right side with compressive atelectasis of the underlying parenchyma and mild shift of the mediastinum to the left, bilateral signs of necrotizing pneumonia without clear distinction of air-filled cavity / abscess.
Slika 2. Kompjuterizovana tomografija grudnog koša – masivni pleuralni izliv sa desne strane sa kompresivnom atelektazom podležućeg parenhima i blagim pomeranjem medijastinuma put levo. Obostrano su bili prisutnu znaci nekrotične pneumonije bez jasno formiranih apscesnih kolekcija.

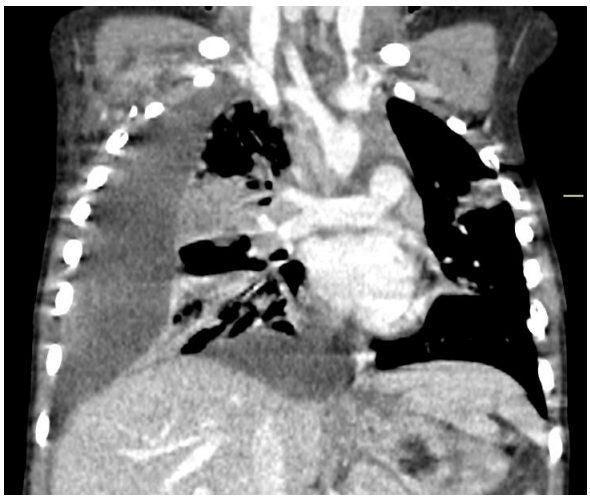

Figure 3. Contrast enhanced chest computed tomography (coronal section) - partial thrombosis at the junction of the left jugular and subclavian vein. An extensive right-sided pleural effusion with collapsed right lung and necrosis of the lung parenchyma.
Slika 3. Kompjuterizovana tomografija grudnog koša – parcijalna tromboza na spoju leve jugularne i potključne vene

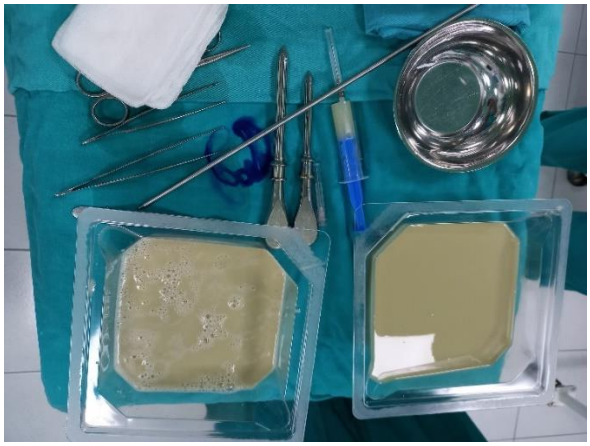

Figure 4. Intraoperative finding after thoracic drainage- drained purulent pleural content.
Slika 4. Intraoperativni nalaz nakon torakalne drenaže – dreniran purulentan pleuralni sadržaj.

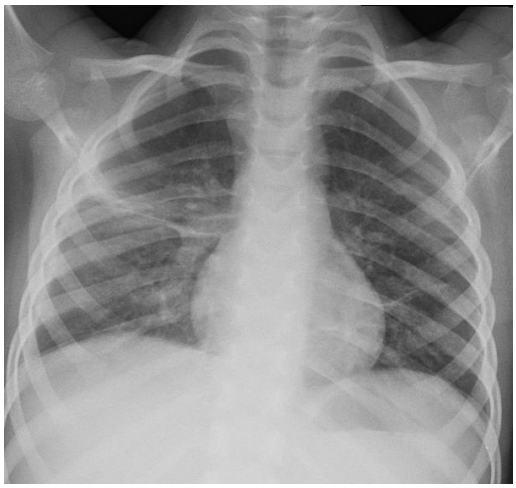

Figure 5. Chest X-ray on the day of discharge from the hospital - nearly complete resolution of bilateral complicated pleuropneumonia.
Slika 5. RTG pluća na dan otpusta iz bolnice koji pokazuje gotovo potpuno regresiju prethodno opisivanih konsolidacija.

hospital day. During hospitalization, the lung findings were monitored by lung ultrasound, showing a gradual regression of the consolidations. The boy was discharged after 24 days of hospitalization with oral antibiotic therapy (amoxicillin, clavulanic acid) and oral anticoagulant therapy. On the day of discharge, a chest X-ray was performed, which showed satisfactory resolution of complicated bilateral pleuropneumonia (Figure 5).

DISCUSSION

Complicated pneumonia in the paediatric population have been on the rise over the past twenty years (5). NP most commonly occurs in children under the age of 5, who were previously healthy. Nearly half of children in this age group with community-acquired pneumonia are hospitalized (6,7). This is also confirmed by our case. Despite the outpatient antibiotic therapy, his clinical condition required hospitalization. NP are caused by particularly virulent bacteria, with *Streptococcus pneumoniae* being the most common agent. However, studies show that microbiological analysis results are positive in only 8-55% of cases (1). In our patient, *Streptococcus pneumoniae* was isolated from the pleural fluid culture, while the blood culture was negative. Additionally, literature suggests that influenza virus infection is associated with increased nasopharyngeal colonization by *Streptococcus pneumoniae* and *Staphylococcus aureus*, leading to a higher risk of secondary bacterial infections. A study conducted by Ramoglu et al, during the H1N1 pandemic in 2009, reported an unusually high number of NP cases in children (8). In case of our patient, influenza virus infection was confirmed by PCR, which likely represented a predisposing factor for the development of complicated bacterial pneumonia.

In the majority of cases, NP is associated with an increase in acute-phase inflammatory markers (C-reactive protein and procalcitonin), with maintaining of elevated levels throughout the course of the disease. Additionally, laboratory findings often reveal the presence of mild to moderate anemia, electrolyte imbalance, and hypoalbuminemia (9). This was also the case in our patient, who presented with elevated CRP levels and procalcitonin within the range indicative of systemic bacterial infection. These values remained elevated during the clinical course, necessitating adjustments in antibiotic therapy. Furthermore, hypoalbuminemia was noted upon admission, requiring supplementation with human albumin.

The diagnosis of necrotizing pneumonia should be considered in any child with pneumonia that does not improve 72 hours after appropriate antibiotic therapy. As necrotizing pneumonia causes liquefaction of lung tissue and can lead to pulmonary cavities, differential diagnosis should exclude the presence of tuberculosis, secondarily infected congenital pulmonary cysts, as well as traumatic pseudocysts (6). The diagnosis of NP is established based on radiological imaging. The gold standard for diagnosing NP is chest CT. Lung ultrasound is the primary radiological method for evaluating the pleural space, but it is also important for assessing lung parenchyma necrosis (10,11,12).

In a study conducted by Lai et al a good correlation between lung ultrasound and chest CT was demonstrated in the diagnosis of community-acquired necrotizing pneumonia in children (10). Recently, MRI is used for diagnosis and follow-up of complicated pneumonias, but it is still not widely applicable (12). In our case, initial ultrasound examination was performed, followed by chest CT (before performing thoracic drain-

age), where the presence of necrotizing pneumonia with massive pleural effusion was confirmed. As a disease complication, jugular vein thrombosis was also radiologically verified. The patient was followed-up via ultrasound until discharge, without additional, unnecessary exposure to ionizing radiation.

Prolonged intravenous antibiotic therapy represents the cornerstone of NP treatment. Empirical antibiotic therapy, in previously healthy children, should target gram-positive microorganisms, particularly *Streptococcus pneumoniae*, followed by *Staphylococcus aureus* and *Streptococcus pyogenes*, with consideration of local epidemiological and microbiological data (5). The optimal duration of antibiotic therapy for NP remains a subject of debate in the scientific literature. The average duration of antibiotic treatment in the literature ranges from 13 to 42 days (5, 6). In our patient, empirical dual parenteral antibiotic therapy (clindamycin, ceftriaxone) was initiated, which was subsequently adjusted based on the clinical presentation, laboratory, and radiological findings, with the introduction of vancomycin and meropenem. The pleural effusion culture, which was positive for *Streptococcus pneumoniae*, showed sensitivity to all the aforementioned antibiotics. Parenteral antibiotic therapy was administered for 24 days. Transition to oral antibiotic therapy was initiated when the child had been afebrile for at least 24 hours, without signs of respiratory distress, tolerated enteral intake, and with a decrease in the acute-phase inflammatory parameters. It was recommended that oral antibiotic therapy continues for at least 10-14 more days (9). In accordance with these recommendations, our patient was discharged with oral antibiotics.

Unlike pneumonias that are not associated with necrosis, NP often leads to complications such as parapneumonic effusions and the development of empyema (3,13), as was the case with our patient. In such cases, NP requires pleural drainage (with or without fibrinolytic therapy) and/or surgical procedures, primarily video-assisted thoracoscopic surgery (VATS) (14). However, pleural drainage lasting more than 7 days, as well as the use of fibrinolytics in necrotizing pneumonia, carries the risk of developing a bronchopulmonary fistula. Therefore, it is necessary to carefully assess the therapeutic benefit and, in consultation with a paediatric surgeon, make an appropriate decision regarding treatment (5). In our patient, initial pleural drainage was performed, but as the clinical response was unsatisfactory, treatment was supplemented with the use of fibrinolytics (alteplase), which was carried out without complications and resulted in significant clinical improvement.

Children with necrotizing pneumonia often have a prolonged and extended clinical course, leading to a lengthy hospital stay, typically ranging from 12 to 30 days. Hospitalization is further prolonged if patients require surgical treatment (9). This trend was also observed in our patient, who was discharged after 24 days of hospital treatment.

The primary preventive measure for NP is vaccination against *Streptococcus pneumoniae*. In the United States of America (USA), as well as in other countries, following the initial introduction of the 7-valent pneumococcal conjugate vaccine (PCV), there was a significant reduction in the incidence of CAP by more than 40%. Vaccination also led to a decline in the incidence of infections caused by invasive serotypes covered by the vaccines (15,16). Since 2018, the 10-valent and 13-valent PCV vaccines have been part of the mandatory vaccination program for children over 2 months old in Serbia. The 10-valent PCV contains serotypes 1, 4, 5, 6B, 7F, 9V, 14, 18C, 19F, and 23F, while the 13-valent PCV includes three additional serotypes: 3, 6A, and 19A (16). Cathalau and colleagues, in their study covering children with necrotizing pneumonia who were hospitalized at a tertiary healthcare center from 2008 to 2018, observed an unchanged incidence of NP during this period, along with a shift in bacterial pathogens following the introduction of the PCV13 vaccine (with fewer isolated cases of *S. pneumoniae* and an increase in *S. aureus and S. pyogenes*) (17). Our patient received the PCV 10 vaccine. The most common serotypes leading to necrotizing pneumonia are serotype 3 and serogroup 19A, which are not covered by PCV 10, which may be one of the potential reasons for the development of this complication in our patient.

Despite the severe clinical presentation, the final outcome of necrotizing pneumonia (NP) is positive in the majority of patients, with complete recovery. Studies have shown that the mortality rate for patients with NP ranges from 0% to 7.5% (5,7). Our patient also had a positive outcome, being discharged without symptoms and with a satisfactory radiological finding.

CONCLUSION

The diagnosis of necrotizing pneumonia should be considered in any child with pneumonia that does not improve 72 hours after appropriate antibiotic therapy. Early diagnosis and timely treatment are crucial for a positive outcome. Vaccination against pneumococcus is the primary preventive measure, especially if the vaccine covers the serotypes that lead to necrotizing pneumonia. Further research, with significantly larger patient cohorts and prospective studies, is needed to better understand the epidemiological and clinical aspects of this disease and to align recommendations for the optimal duration of antibiotic therapy.

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