Endokarditis Infektif pada Laki-laki Berusia 60 Tahun di RSUP Dr. Kariadi Semarang

Infective Endocarditis in 60 Years Old Man at Dr. Kariadi Hospital

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Abstrak


Kata kunci : endokarditis infektif, Streptococcus β haemolyticus, kultur darah, pemakaian antibiotik yang tepat

ABSTRACT

Infective endocarditis is an infection of the lining of the heart, particularly the cusps of valves characterized by fever and heart murmur, with or without malaise and fatigue. Clinical features can be divided into early manifestations of infection, embolic events, and late complications of sepsis and inflammation. It was reported that 60 years old man was brought to Kariadi hospital with prolong febris as his chief complaint. Patient has history of high grade fever and accompanied by weight loss, 3 kilograms for about 2 months. From physical examination result, in internal ward, we found he looked moderate ill. Heart systolic bruit at triscupidal valve and lungs were within normal limit. The result of blood culture from 3 different sites and throat swab culture were positive, lead to the performance Streptococcus β haemolyticus's colonies. From the result of the ASTO, Echocardiography and physical examination show infective endocarditis's sign. The therapy was Ceftriaxone 2 gram daily. Having obtained the results of negative cultures, and show an improvement of symptoms, finally the patient was getting improvement clinically and then treat as an outpatient. Patient was programmed to get Ceftriaxone 2 gram daily for 4 weeks. The decision of treatment not only from clinical manifestation but also from microbiology examination on the first admission in emergency unit. Blood culture with 3 bottles, interval 30 minutes is important for infective endocarditis. Furthermore, swab of throat area were taken to confirm for endocarditis cultures too. Finally there is no growth in culture until eighth day hospitalizasition. Spesific method on blood culture sampling is important to confirm the diagnostic of infective endocarditis and very useful for the clinicians to do the right clinical management for it. Subsequently, the role of clinical microbiologist in this case not only to support the clinical diagnostic but also the treatment in term of selecting of right antibiotic.

Keyword: infective endocarditis, Streptococcus β haemolyticus, blood culture, right antibiotic

CASE REPORT

The case reported 60-years old man hospitalized since July 3th 2012 until July 11th 2012. His main complaint was prolong febris. Since 2 months before hospitalization he was observed fever in general practitioner’s clinic and was treated for about 2 weeks in the local hospital hospital, 2 months, the man suffered from fever, high-grade fever, no seizure, no shivering, cough (-), dysphagia (-), no discharge from ears, no nose bleeding, no gums bleeding but there was a dental carries at superior left pre molar, no ptechie, no nausea, no vomite, no gastrointestinal and urinary tract complaints. He was got to physician and given some medicine. Two weeks in hospitalization, the man was still fever, given treatment but no improving. After that he refer to Kariadi’s hospital. Accompanied by weight loss, 3 kilograms in 2 months. These were loss of appetite, without nausea, and no vomiting. Body image looked underweight. No convulsion and no complaint both in urination and defecation. From
physical examination in emergency room and internal ward, found he looked moderate ill. Heart systolic bruit at tricuspidal valve and lungs were within normal limit. The result of throat swab culture was positive with Streptococcus β haemolyticus. So do blood agar cultures from 3 different sites lead to the performance Streptococcus β haemolyticus’s colonies. He was hospitalized in internal ward in C3L2.

He lived in urban area in Semarang. There were no data about the same infections in his family and community. He is a manual labour with average income is ± Rp 1.000.000,-/month. Health cost was covered by Jamkesmas. Social economic impression is poor.

From physical examination, a 60-year old man, GCS 15: E4M6V5. The blood pressure was 130/80 mmHg; respiration rate 20 times per minute; pulse rate 100 times per minute, regular and with sufficient pressure; body temperature was 40°C (rectal). Body image looked underweight. Eye with conjunctiva palpebrae not pale; sclera was not icteric. Pupils were equal (Ø 2mm) and reactive to light stimulation. Mouth, there was no stomatitis. No lymph node enlargement at neck regio, normal JVP, no tracheal deviation, no stiff neck. Ear: discharge (-), nose: bleeding (-). Chest: no spider nevi, no muscles atrophy, no gynecomastia, no axillary lymph node enlargement, and no intercostals retraction. Heart systolic bruit at tricuspidal valve and lungs were within normal limit. Liver and spleen were unpalpable.

Laboratory examinations during hospitalization were Hb11,2 gr/%, Ht 33,4%, leucocyte 11.600/ mmk, activated lymphocyte, erythrocyte 4,06jt/ mmk, thrombocyte 11 ��.000/ mmk. Ureum 33 mg/dl, Creatinine 0,94 mg/dl, albumin 2,�� mg/dl, ASTO positive, CRP 3,21 mg/dl.

Echocardiography found vegetation in tricuspidal valve, it suggest an infection track was occured there. In ward patient received nasal oxygen 2 l/minute, RL infusion 30 drops per minute, Ceftriaxone injection 2 grams i.v two times daily, paracetamol 500 mg if necessary, and vitamin B complex three times daily.

There was no change at physical examination, but on July 10th 2012 temperature 37,3°C. Patient was finally went back home on July 11th 2012. He was programmed to get Ceftriaxone 2 grams daily for 4 weeks at outpatient clinic.

**DISCUSSION**

**Etiology**

Many bacteria can be etiology of infective endocarditis. *Streptococcus viridians, Enterococcus faecalis, Enterococcus faecium, Enterococcus durans, Staphylococcus aureus, Staphylococcus lugdalensis, Haemophilus parainfluenza, Haemophilus ophrophilus, Haemophilus paraphrophilus, Haemophilus influenza, Actinobacillus actinomyctecomitans, Cardiobacterium hominis, Eikenella corrodens, Kingella kingae, Kingella denitrificans* (HACEK group) (Baddour et al., 2007). In this case, swab staining and blood culture were positive for Streptococcus β haemolyticus and the patient was diagnosed with infective endocarditis.

**Pathology of Streptococcus β haemolyticus**

*Streptococcus β haemolyticus* is a member of group A streptococci. Typically appear in purulent lesions or broth cultures as spherical or ovoid cells in chains of short to medium length (4 to 10 cells). On blood agar plates, colonies are usually compact, small, and surrounded by a 2 to 3 mm zone of hemolysis that is easily seen and sharply demarcated hemolysis is caused by either of two hemolysins, streptolysin S and the oxygen labile streptolysin O, both of which are produced by most group A strains. Strains that lack streptolysin S are hemolytic only under anaerobic conditions, because the remaining streptolysin O is not active in the presence of oxygen. This feature is of practical importance, because such strains would be missed if cultures were incubated only aerobically (Ryan, 2004; Forbes et al., 2007; Brooks et al., 2006).

**M Protein**

The M protein itself is a fibrillar coiled-coil molecule with structural homology to myosin. Its carboxy terminus is rooted in the peptidoglycan of the cell wall, and the amino-terminal regions extend out from the surface. The specificity of the more than 80 serotypes of M protein is determined by variations in the amino sequence of the aminoterminal portion of the molecule. Because of its location, this part of the M protein is also the most available to immune surveillance. The middle part of the molecule is less variable, and some carboxy terminal regions are conserved across...
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many M types. There is increasing evidence that some of the many known biologic functions of M protein can be assigned to specific domains of the molecule. This includes both antigenicity and the capacity to bind other molecules such as fibrinogen, serum factor H, and immunoglobulins (Fischetti and Ryan, 2009).

Other Surface Molecules

A number of surface proteins have been described on the basis of their similarity with M protein or some unique binding capacity. Of these, a fibronectin binding protein F and LTA are both exposed on the streptococcal surface and may have a role in pathogenesis.

An IgG binding protein has the capacity to bind the Fc portion of antibodies in much the same way as staphylococcal protein A. In principle, this could interfere with opsonization by creating a covering of antibody molecules on the streptococcal surface that are facing the wrong way. Group A streptococci may have a hyaluronic acid capsule, which is a polymer containing repeating units of glucuronic acid and N-acetylglucosamine (Forbes et al., 2006; Fischetti and Ryan, 2009).

Biologically active extracellular products

Streptolysin O

Streptolysin O is a general cytotoxin, lysing leukocytes, tissue cells, and platelets. The toxin inserts directly into the cell membrane of host cells, forming transmembrane pores in a manner similar to complement and staphylococcal toxin. Streptolysin O is antigenic and the quantitation of antibodies against it is the basis of a standard serologic test called antistreptolysin O (ASTO) (Forbes et al., 2006; Fischetti and Ryan, 2009).

In this case, laboratory result ASTO as the sign for streptolysin O which positive for Streptococcus β haemolyticus.

Pyrogenic Exotoxins

The manifestations of classical scarlet fever have long been associated with the action of an erythrogenic toxin. This toxin is now included in a family of nine proteins called streptococcal pyrogenic exotoxins (SPEs), one of which is produced by approximately 10% of group A streptococci. The SPEs are identified by letters (e.g., A, B, C) and are similar in structure and biological activity to the pyrogenic exotoxins produced by Staphylococcus aureus. They have multiple effects including fever, rash (scarlet fever), T-cell proliferation, B-lymphocyte suppression, and heightened sensitivity to endotoxin. At least some of these actions are due to cytokine release through the superantigen mechanism. SPE-B also has enzymatic activity cleaving elements of the extracellular matrix, including fibronectin and vitronectin (Forbes et al., 2007).

Other Extracellular Products

Most strains of group A streptococci produce a number of other extracellular products including streptokinase, hyaluronidase, nucleases, and a C5a peptidase. The C5a peptidase is an enzyme that degrades complement component C5a, the main factor that attracts phagocytes to sites of complement deposition. The enzymatic actions of the others likely play some role in tissue injury or spread, but no specific roles have been defined. Some are antigenic and have been the basis of serologic tests. Streptokinase causes lysis of fibrin clots through conversion of plasminogen in normal plasma to the protease plasmin (Brooks et al., 2006).

Pathology of infective endocarditis

The term endocarditis or Infective endocarditis (IE) describes infection of the lining of the heart, particularly the cusps of valves (Karchmer, 2006). Infective endocarditis (IE) is caused by microorganisms adhering to and multiplying on the innermost aspect of the chamber of the heart and its valves (the endocardium). Infections often involve an abnormal heart valve such as one previously damaged by rheumatic fever, atherosclerotic plaques, or a prosthetic (artificial) valve. Less frequently, infection occurs at the site of congenital cardiac abnormalities such as a septal defect or an arteriovenous shunt. It is very important to remember that infection can occur on a normal heart valve (Table 1) (Gould et al., 2012).

<table>
<thead>
<tr>
<th>Table 1. Important predisposing factors for infective endocarditis</th>
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<tr>
<td><strong>Congenital heart disease</strong></td>
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<tr>
<td>• Previous rheumatic heart disease</td>
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<tr>
<td>• Atherosclerotic aortic valve disease</td>
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<tr>
<td>• Prosthetic valve heart surgery</td>
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<tr>
<td>• Severe mitral valve prolapse</td>
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<td>• Intravenous drug use</td>
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In this case, it’s not clear about predisposing factor, but could be from dental carries. Although many species of bacteria and fungi cause sporadic episodes of endocarditis, only a few bacterial species cause the majority of cases (Table 2) (Limited, 2008).

Clinical Presentation

Clinical features can be divided into early manifestations of infection; embolic events; and late complications of sepsis and inflammation. The earliest features are usually fever and heart murmur, with or
without malaise and fatigue. Features of embolization appear after days or weeks. Early emboli are seen in more aggressive endocarditis. The long-term effects of endocarditis, now rarely seen, are immunologically mediated. Immunological effects include splenomegaly, nephritis, vasculitic rashes or lesions of the eyes and skin (Table 3) (Karchmer, 2010).

In this case, clinically this patient showed symptoms of endocarditis infectious. ASTO, culture, echocardiography and physical examination show it. Based on a careful history taking, it can be concluded that prolonge febris was caused by endocarditis.

Treatment

Therapy suggestion from clinical microbiology is Ampicillin sulbactam but social indication not permitted for this drug to give because Ampicillin sulbactam must be given 3 grams four times daily. Ceftriaxone become an alternative because it’s dosage can be given 2 grams once daily.
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Subsequently, the role of clinical microbiologist in this case not only to support the clinical diagnostic but also the treatment in term of selecting of right antibiotic based on clinical presentation and laboratory result.

DAFTAR PUSTAKA


