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Infective Endocarditis

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Abstract:
Is a serious infection of the valves and endocardium caused mainly by bacteria and is characterized by the formation of infected friable vegetations.(1) Dental procedures and even non-surgical dental procedures, can often cause bacteremia of oral commensals. Bacteremia itself rarely affect healthy people but they can result in mortal infective endocarditis in those who have a predisposed risk for this disease.(2)

Introduction:
There are two types of infective endocarditis (IE), 1. acute: which is a sever, destructive of previously normal heart valve , with high virulent organism, such as Staphylococcus aureus. 2.Subacute: organisms of low virulence can cause infection in previously abnormal heart (deformed valves) such as : streptococcus viridian. Symptoms of (IE) may include: fever, malaise, loss of weight, anaemia, splinter haemorrhages, petechiae, cardiac murmur, haematuriaand splenomegaly.(1).
So there is many organisms that cause infective endocarditis (IE), and usually there are classified according to the type. These organisms are: Streptococcus viridians, which is found in the mouth contracted from dental procedure. And affect the valves that had previously damaged (50-60% of cases). staphylococcus aureus, which is a highly virulent bacteria that is found in the skin and usually contracted from intravenous drug, and attack either healthy or deformed valves (10% to 20% of cases). Streptococcus faecalis 13%, S.epidermidisa , this bacteria likes the prosthetic valves (usually it's nosocomial infection), and culture negative (gram negative bacteria) HACEK 10%.(2)
There are predisposing factors that increases the risk of (IE), Almost any type of heart lesion is susceptible for this infection, such as congenital cardiac defect, rheumatic heart disease, prosthetic cardiac valves, history of endocarditis, hypertrophic cardiomyopathy, aortic valve disease(bicuspidvalve), and antibiotics will be needed(3).

Discussion:
Dental therapy and other invasive procedures have been linked to seeding of heart valves and the development of IE. Healthy patient are less than 5% acquire IE as result of dental treatment.(3) Infective endocarditis normally occurs in patients with some pathological condition of the endocardium, although those with apparently normal heart valves may rarely be affected. Infective endocarditis is the end-result of the sequential interaction of 1. A breach of the endocardium, or an abnormality of the endocardial surface per se, is the first event which makes the valvular surface finally succumb to infection. Such a breach may occur because of the acute inflammatory
valvulitis of rheumatic fever , infection; or, in congenital heart diseases such as aortic valve disease and ventricular septal defect, when alterations of the blood flow patterns hemodynamic turbulence) may result in the deposition of fibrin and platelets at foci where high-velocity jets of blood hit the valvular surface. 2. The microscopic platelet aggregates which form on the breached endocardium detach and embolize harmlessly or stabilize and consolidate through fibrin deposition, forming a sterile thrombus. The latter is a potential trap for circulating microbes. Such sterile thrombus formation is called non-bacterial thrombotic endocarditis. Platelets also have the potential to adhere to other 'foreign' surfaces such as prosthetic valves. 3. The next critical event occurs when organisms circulating in the blood (e.g. after a tooth extraction or scaling) attach to or become trapped in the thrombotic endocardium or the prosthetic device. The resultant platelet-fibrin-bacterial mass, now called the bacterial vegetation, constitutes

the primary pathology of infective endocarditis 4. Once the organisms are attached to the lesion they multiply and colonize this niche in an exuberant manner. As a result, further aggregation of platelets and fibrin deposition ensues, protecting the organisms from the body defences. The organisms now reside in a sanctuary inaccessible to phagocytes by virtue of the fibrin-platelet barrier. Further, the bacteria may be sheltered from antibiotics and host antibodies as the vegetation is essentially avascular in nature. As a result it is necessary to use an intensive course of prolonged, high-dose antibiotic therapy to eradicate such an infective focus. 5. Even if endocarditis is successfully treated, the healed valve is permanently scarred and thickened and such residual abnormalities make the patient highly vulnerable to episodes of reinfection (2). Endocarditis Prophylaxis: 1. oral hygiene instruction. 2.Dietary advice and regular dental examinations. 3. Antiseptic prophylaxis; the main source of microorganisms in significant dental bacteremia is supragingival and subgingival plaque, to reduce this risk of infection 1. Irrigating the gingival crevice area with antiseptics (e.g. chlorhexidine gluconate gel 1% or mouthwash 0.2%, used 5 min before the procedure) (4). Antibiotic prophylaxis :1 amoxicillin 3gm orally 1h before operation (high risk patient that will go through local anesthesia) 2. oral clindamycin 600m should be given to patient allergic to penicillin or who have taken penicillin more than once in the previous month (other alternatives: 500 mg azithromycin or clarithromycin) 3. IV vancomycin and erythromycin (oral) (5).

Conclusion:

Dental procedure can be one of the causes of development of infective endocarditis, especially whom with previously diseased valve, patients with high risk will need proper antibiotic administration.

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