SEPSIS CAUSED BY STREPTOCOCCUS BETA HEMOLITIC GROUP A, IN A PATIENT WITH ERYSIPELAS CRURIS CASE REPORT

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PËRMBLEDHJE

Sepsisi percaktohet si nje sindrome e pergjigjes inflamatore sistemike, i shoqeruar me shenjat e nje infeksioni te vertetuar me pranine e mikroorganizmave patogene ne inde ose ne lengje, qe zakonisht jane stemike ,ai paraqet nje spekter te ndryshimeve klinike, shkaktuar nga pergjigja imunitariae nje infeksioni ose traume karakterizuar nga inflamacioni sistemik dhe disfinksioni multiorganik, qe shpesh eshte shkak i vdekjeve te shume pacienteve. Ne gjithe boten sepsisi eshte konsideruar si shkaku kryesor i semundshmerise dhe mortalitetit, ne menyre te vecante ne paciente me gjendje kritike per jeten, te moshuarit,dhe imunodeficientet. Letaliteti nga sepsisi ne bote varion nga 28%- 50 %.. Nder faktoret e riskut vecojme: infeksionet hospitaliere, interventet operatore, ne menyre te vecante ato kirurgjikale *Pis ose jo sterile*,kateterizimet e ndryshm, urinare, intravaskular, dhe procedurat kirurgjikale, semundjet kronike, renia e imunitetit, obeziteti, diabeti etj.

Fjalet kyc : Sepsis ,inflamacion,streptokok

SUMMARY

Sepsis presents a wide clinical presentation caused by an immune presentation of an infection or a trauma, characterized by systemic inflammation and coagulation alteration. It represents a spectrum of clinical changes caused by the immune response of an infection or trauma characterized by systematic inflamation and multiorganic failure wich is often the cause of the death of many patients. Sepsis is considered in the many parts of the world as the main cause of morbidity and mortality in intensive care centres, especially in patients with critical condition for life, elderly and immunodeficiency^{4,5}. The mortality in this group is high, approaching 50%–70% in patients with hypotension and organ failure²¹⁻²² Risk factors for group β - streptococcal sepsis in adults include diabetes mellitus, nosocomial acquisition, malignancy, surgical procedures, especially those called "dirty or not sterile", hepatic failure, chronic disease, immunodeficiency, obesity, ect.

Key words: sepsis, inflammations, streptococcus

INTRODUCTION

Sepsis is the hosts' reaction to infection and is characterized by a systemic inflammatory response. Because of difficulties in defining sepsis, the SIRS was introduced trying to summarize the inflammatory response in a limited set of elementary characteristics (fever or hypothermia, leucocytosis or leucopenia,

tachycardia, hyperventilation. The majority of adult infections apparently occurred as a result of nosocomial acquisition and was associated with a high mortality rate of 38%. Infections developing after surgical procedures involving nonsterile tissue, such as colonic, vaginal, biliary or respiratory mucosa, may be caused by a combination of aerobic and anaerobic bacteria.

These infections can rapidly progress and involve deeper structures than just the skin, such as fascia, fat, or muscle3. This spectrum ranges from a systemic inflammatory response, which often is the cause of death. It is a potentially dangerous condition threatening life that is found in association with an infection known or suspected. In definicion ,sepsis is defined as infection plus systemic manifestations of infection . Severe sepsis is defined as sepsis plus sepsis-induced organ dysfunction or tissue hypoperfusion.

According to the American College of Chest Physicians and the Society of Critical Care Medicine, there are different levels of sepsis:

- Systemic inflammatory response syndrome (SIRS) is the presence of two or more of the following abnormal body temperature, heart rate, respiratory rate or blood gas, and white blood cell count.
- Sepsis is defined as SIRS in response to an infectious process. It however can be triggered by many things other than sepsis.
- Severe sepsis is defined as organ dysfunction due to an infection while septic shock is severe sepsis plus persistently low blood pressure following the administration of intravenous fluids

To have sepsis should have at least 2 signs:

-Temperature: >38°C or <36 °C

-Heart rate: >90/min

-Respiratory rate: >20/min or PaCO2<32 mmHg -WBC: <4x109/L (<4000/mm³), >12x109/L (>12,000/mm³), or 10% bands

Sepsis causes millions of deaths globally each year. In the United States sepsis affects approximately 3 in 1000 people a year. It is the second-leading cause of death in non-coronary intensive care unit (ICU) patients, and the tenthmost-common cause of death overall according to data from the (the first being heart disease). Sepsis is common and serious in the elderly, the immunocompromised, and the critically ill. It occurs in 1–2% of all hospitalizations and accounts for as much as 25% of ICU bed utilization. Approximately 20–35% of people with

severe sepsis and 30–70% of people with septic shock die. The average cost for treating Sepsis is over 22.800 euro .

Risk factors for group β - streptococcal sepsis in adults include:

- 🛮 diabetes mellitus
- nosocomial infections
- malignancy
- ② surgical procedures, especially those called "dirty or not sterile"
- hepatic failure
- chronic disease
- !immunodeficiency
- ② obesity

Severe sepsis, acute organ dysfunction secondary to infection. is major healthcare problems, affecting millions of individuals around the world each year, killing one in four (and often more), and increasing in incidence. The most frequent causal agents are Staphylococcus aureus, E.coli, Streptococcus β-hemoliticus gr.A, Candida species, Clostridium difficile, or Enterococcus faecium. In addition to S. aureus, the Grampositive bacteria Streptococcus pyogenes is a major cause of complicated skin and skin structure infections. Reliably distinguishing between infections caused by these two agents is difficult because of overlaps in clinical presentation. Foci of infection include an intraabdominal abscess or gastrointestinal perforation. cholangitis or pyelonephritis, intestinal ischemia or necrotizing soft tissue infection, and other deep space infection such as an empyema or septic arthritis.

Complications With early diagnosis and proper treatment, the prognosis is excellent. Rarely, however, the infection may extend to deeper levels of the skin and soft tissues3. Sepsisi can give complications in various organs, as the heart, lung reins as renal insuffciency, cardiac, KID, wich causes a severe hemorhagic diathesis.

Causes of death: The decline in cardiac function, respiratory insufficiency, KID hemorhagic

diathesis and renal insufficiency, shock and multiple organ failure.

Prognosis: For adult the rate of morbidity and mortalitety is depended from the number of damaged organs, most of whom will have multiple organ failure. Patients with low risk of death, most of whom will have single organ dysfunction. Objective: To highlight streptococcal infections as a cause of sepsis as well as infections in appearance not so problematic as erysipelas, but that might lead to septic conditions life threatening, its prevention, diagnosis and effective treatment

CASE REPORT

This case report describes our experience with a patient, male, 70years old. On admission, he was in a poor general state, an axillary temperature of 40ºC, TA 70/40 mg Hg, tachycardia (90beats/min), tachypnea (51breats/min), abdominal distention, signs of nerve dysfunction, anuri. Throughout the anterior fascia of the right leg, hard edema with a violet skin color and blistered lesions containing liquid with a fetid odor and bloody regions with decreased peripheral perfusion were observed. The clinical course was marked by a rapid deterioration of the lesion's appearance and extension.

Anamnesis: He has about 5-6 days with temperature, fever, swelling and redess of the highlighted part crurale dexter. He was treated with Ampicillin 3grams/per day for a month but his condition is not improved. He comes in critical conditions in hospital. Patients who present to the hospital with severe infection or whose infection is progressing despite empirical antibiotic therapy should be treated more aggressively, and the treatment strategy should be based upon results of appropriate Gram stain, culture, and drug susceptibility analysis2. Penicillin, given either parenterally or orally depending on clinical severity, is the treatment of choice for erysipelas3. In a randomized, prospective multicenter trial16, the efficacy of roxithromycin, a macrolide antimicrobial, was equivalent to that for penicillin. We started initial

empirical anti-infective therapy include (Piperacillini+Bacitracini 4.5x4 fl i.v, Amikacini500 x 2i.v. ??)

Adequate fluid resuscitation was done before vasopressors and inotropes were used. He had hypotension with anuri (and with blader catheter) so we started administration of a dopamine infusion (up to a maximum of 20 µg.kg.min),and after the bed vazal was full, began large dose diuretic (lasix 20 amp i.v) .After 3-4 hours TA rose in numbers 110/60 mm Hg,but diureza was decided only after 8 hours,while in examinations was observed:

Results of the laboratory examinations were: RBC= 3 700 000/mm3, Leukocytes = 13 000/mm3,

uremia 18mg%, creatinine 4.3mg%, glicemia 87mg%. The material of wound was analyzed and it resulted to be Streptococcus beta hemolytic group A. Because of their very low yield, blood cultures are not fruitful for the typical case of erysipelas or cellulitis, unless it is particularly severe20. In the second day patient began to have signs petekie and ekimoza in the right leg and abdominal region. In the rightleg he had injuries as form of cellulite and the akrocianoze of toes, both legs and in the part of the erytherma had blisters seropurulent juice and areas of necrosis, in the form of nekrotizant erysipelas. On the third day patient had no temperature, Ta 120/70, normal urination but signs of akrocianozes in the toes of the feet, cellulits and ekimoza as a result of hypotension and KID were present. Was also observed in necrosis area crurale partsin, for wich we did surgical exploration or debridement of necrotic parts. As during the examinations it was noted that renal and inflammatory alterations were continuing, it was decided to change the medicaments and to use Teikoplanine, Metronidazol and Ciprofloxacine.



Figure1 appears erysipelas in 2-3days, the necrosis



Figure 2, after surgical exploration or debridement of necrotic parts (after 5 days).

TREATMENT

Surgical intervention is the major therapeutic modality in cases of necrotizing fasciitis. The decision to undertake aggressive surgery should be based on several considerations. First, no response to antibiotics after a reasonable trial is the most common index. A response to antibiotics should be judged by reduction in fever and toxicity and lack of advancement. Second, profound toxicity, fever, hypotension, or advancement of the skin and soft-tissue infection during antibiotic therapy is an indication for surgical intervention. Third, when the local wound shows

any skin necrosis with easy dissection along the fascia by a blunt instrument, more complete

incision and drainage are required. Fourth, any soft-tissue infection accompanied by gas in the affected tissue suggests necrotic tissue and requires operative drainage and/or debridement.

The patient with erysipelas returned to the operating room 24–36 h after the first debridement and daily thereafter until the surgical team finds no further need for debridement.

DISCUSSION

If the patient has septicemia streptococci it must exist a septic hearth, which should be the source of microbes which come in blood circulation. Even in our case, the patients had erysipelas which caused the septic situation. Some of the factors which cause Sepsis were present as: temperate 400 C, tachycardia 90', leucocytosis polipnea anuria and neural excitation. Also some coexisting factors as diabetes mellitus and obesity, the situation can be grave and complicated caused by Streptococcusa If there is a coexisting factor as diabetes mellitus, the situation can be grave and complicated in a serration center The right medicament according to antibiorgame made the patient wake up and treate their self

CONCLUSIONS

To develop streptococcal septicemia is necessary to exist a septic focus in the organism, from which microbes occasionally jump circulation.Streptococcal infections may assume the character of septicemia in the presence of favorable conditions which decrease the resistance of the organism from malnutrition, great fatigue, severe diseases convalescence. Streptococci Infections can be converted into septicemia under conditions as reduction of organism resistance caused from malnutrition, exhaustion, diabetes, heart diseases, obesity, etc. The grave bacteriology infections should be followed rigorously because if they are not detected and treated on time, that can be fatal. The septic situation remains an infective monster

even in the third millennium in different countries. It is very important to be diagnosed on time, adequate and effectively treated in order to prevent systematic complications that can be caused.

REFERENCES

- 1. Wong CH, Khin LW, Heng KS, Tan KC, Low CO. The LRINEC (Laboratory risk indicator for necrotizing fasciitis) score: a tool for distinguishing necrotizing fasciitis from other soft tissue infections. Crit Care Med 2004;32:1535–41.
- 2. Thorell E, Jackson MA, Bratcher D, Swanson DS, Selvaragan R. Antimicrobial resistance of Staphylococcus aureus from Kansas City children: what is the appropriate current therapy for pediatric staphylococcal infections [abstract 252]? In: Proceedings and abstracts of the 42nd Annual Meeting of the Infectious Diseases Society of America Guidelines for Skin and Soft-Tissue Infections • CID 2005:41 (15 November) • 1401 (Boston). Alexandria, VA: Infectious Diseases Society of America. 2004:81. 3. Guidelines for Skin and Soft-Tissue Infections • 2005:41 (15 November) 4. Adams BB. Dermatologic disorders of the athlete. Sports Med 2002;32: 309-21. 5. Fehrs LJ, Flanagan K, Kline S, Facklam RR, Quackenbush K, Foster LR. Group A betahemolytic streptococcal skin infections in a US meat-packing plant. JAMA 1987; 258:3131-4. 6.Barton LL, Friedman AD. Impetigo: reassessment of etiology and therapy. Pediatr Dermatol 1987; 4:185-8.
- 7. Barton LL, Friedman AD, Sharkey AM, Schneller DJ, Swierkosz EM. Impetigo contagiosa III: comparative efficacy of oral erythromycin and topical mupirocin. Pediatr Dermatol1989;6:134–8.
- 8. Britton JW, Fajardo JE, Krafte-Jacobs B. Comparison of mupirocin and erythromycin in the treatment of impetigo. J Pediatr 1990; 117: 827–9
- 9. Weinstein L, Le Frock J. Does antimicrobial

- therapy of streptococcal pharyngitis or pyoderma alter the risk of glomerulonephritis? J Infect Dis 1971;124:229–31.
- 10. Meislin HW, Lerner SA, Graves MH, et al. Cutaneous abscesses: anaerobic and aerobic bacteriology and outpatient management. Ann Intern Med 1977: 87:145-9. 11. Ghoneim AT, McGoldrick J, Blick PW, Flowers MW. Marsden AK. Wilson DH. Aerobic and anaerobic bacteriology subcutaneous of abscesses. Br J Surg 1981; 68:498-500 12. Brook I, Frazier EH. Aerobic and anaerobic bacteriology of wounds and cutaneous abscesses. Arch 1990: 125:1445-51. Surg 13. Bisno AL, Stevens DL. Streptococcal infections in skin and soft tissues. N Engl J Med 1996; 334:240-5. 14. Chartier C, Grosshans E. Erysipelas: an update. Int Dermatol 1996; 35:779-81. 15. Bernard P, Plantin P, Roger H, et al. Roxithromycin versus penicillin in the treatment of erysipelas in adults: a comparative study. Br J Dermatol 1992: 127:155-9. 16. Dupuy A, Benchikhi H, Roujeau JC, et al. Risk factors for erysipelas of the leg (cellulitis): casecontrol study. BMJ 1999; 318:1591-4. 17. Bernard P, Toty L, Mounier M, Denis F, Bonnetblanc JM. Early detection of streptococcal group antigens in skin samples by latex particle agglutination. Arch Dermatol 1987; 123:468-70. 18.. Bernard P, Bedane C, Mounier M, Denis F, Catanzano G, Bonnetblanc JM. Streptococcal cause of erysipelas and cellulitis in adults: a microbiologic study using direct immunofluorescence technique. Arch Dermatol 1989: 125:779-82. 19. Perl B, Gottehrer NP, Raveh D, Schlesinger Y, Rudensky B, Yinnon AM. Cost-effectiveness of blood cultures for adult patients with cellulitis. 29:1483-8. Clin Infect Dis 1999; 20. Stevens DL, Tanner MH, Winship J, et al. Reappearance of scarlet fever toxin A among streptococci in the Rocky MountainWest: severe group A streptococcal infections associated with a toxic shock-like syndrome. N Engl J Med 1989; 321:1-7.