

SEPTIC SHOCK IN A SCHOLAR MALE PATIENT

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Abstract

Septic shock can be defined as a severe sepsis with low blood pressure, prolonged over an hour, which does not respond to intravenous administration of fluids and requiring vasoactive substances management. The authors report the case of a scholar Ș.M. male patient, aged 10 years, who was admitted to the Intensive Care Department of Pediatric Clinic I Craiova presenting with coma, severe hypotension, occurred due to fever, vomiting and diarrhea emission. Laboratory investigations revealed: infectious anemia, leukopenia (2900/mm³), followed by leukocytosis (> 13000/mm³, six days), thrombocytopenia (<85000/mm³) which lasted for 7 days and altered coagulation, elevated serum ALT, AST (100-500u / l), hypoglycemia, metabolic acidosis and transient hyponatremia, hypokalemia. Being comatose, with O₂ saturation (determined by pulse oximeter) of 76%, it has been required assisted ventilation for 5 days. Severe hypotension (40/30 mm Hg) in the second day of admission imposed both an adequate fluid rebalancing and proper administration of Dopamine 5μg/kg/min. These are antibiotics that were sequentially used: Sulcef, Meropenem, and Ceftriaxone associated with Metronidazole and Ciprofloxacinum. Clinical evolution was favorable, fever reducing gradually in 4 days. Five days later, he regained consciousness without disabling motor deficits. Due to mechanical ventilation, he presented subcutaneous emphysema as a transient incident. Because of the lesions diagnosed "dry gangrene" to toes, he has been transferred on the 14th day to Pediatric Surgery Department, where he received antibiotics, local treatment, being discharged on the 18th day from admission. In conclusion, our patient was admitted in the Emergency Room for septic shock, a redoubtable complication of sepsis, due to a gastroenteritis. His evolution was a favorable one, unto healing, but with disabling ischemic lesions in the legs caused by microcirculation disturbances in the context of a septic shock low blood pressure. The particularity of this case was a "restitutio ad integrum" healing of the organ damage and his complete recovery after septic shock with life-threatening hypotension.

Key words: septic shock, hypotension.

Introduction

Sepsis is defined as a systemic inflammatory response syndrome (SIRS) induced by infection. Severe sepsis can be diagnosed when an organic injury occurs, as a sign of hypo-

perfusion and / or hypotension. Septic shock is defined by the maintenance of hypotension (SBP <85mmHg) over an hour, according to the correct fluid and electrolyte rebalancing, requiring vasoactive drug administration(1). If it does not respond to any injection of vasopressors, it is a refractory shock. Septic shock is a medical condition as a result of severe infection and sepsis, though the microbe may be systemic or localized to a particular site. Its most common victims are children. It can progress to death within the first 24-48 hours, due to multiple organ failure (MOF)(1), or to reversible multiple organ dysfunction syndrome (MODS) and healing, often with sequelae.

Case report

We present the case of a male scholar, named Ș.M., aged 10 years, from rural environment, who has been hospitalized in Intensive Care Department of First Pediatrics Clinic of Craiova, then moved to the ward. Afterwards, he has been transferred to the Department of Pediatric Surgery. The month of admission was november 2011.

Reasons for admission to Intensive Care Department were: the abolition of consciousness, with cold and cyanotic hands and feet, hypotension (SBP decreased from 90 to 40 mm Hg) occurred due to fever, vomiting and diarrhea emission.

Family history reveals that he is the 6th child of a poor family, whose mother undergoes therapy for pulmonary tuberculosis. Past medical history includes a hospitalization for gastroenteritis, a year ago and a trauma of skull 6 days ago.

On admission, there were described as it follows: a 10-years-old patient, delay of statur-ponderal growth, abolished consciousness, unresponsive to verbal or painful stimuli, fever 39 °C, cyanosis of the face and extremities, pale skin, dehydration, dry mucous membranes, hypotonia of ocular globes, respiratory acidosis, at chest auscultation: rough lung-breathing, HR= 130 b / min, TCR> 3 sec., aloin tongue, adhering to spatula, flushing pharynx, excavated stomach, emesis, watery and fetid diarrhea, at the palpation of the scalp, in frontal area, is decelated a sutured wound, neck stiffness.

Initially, his BP was 90/57 mm.Hg. On the second day of hospitalization, in despite of hydration and fluid rebalancing, he presented extreme hypotension: 40/ 30 mmHg, with HR = 142-170 b / min.

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Blood pressure measured one hour later after administration of Dopamine, Norepinephrine was 77/39 mmHg. He presented fever (39 to 40.5°C) for another three days, but 2nd level coma lasted for 5 days (requiring intubation with mechanical ventilation). On the sixth day he regained consciousness and breathed spontaneously.

Laboratory findings:

First and second days of hospitalization (22-23-11-2011):

There were diagnosed anemia, leukopenia, thrombocytopenia.

HL + FL: E = 3900000/mm³, Hb = 10.7 g / dl, Ht = 33%, HEM = 28pg/cell, MCHC = 36%, MCV = 76fl, Platelets = 53000/mm³, White blood cells = 2900-3800/mm³, FL: PMNs = 81%, basophils = 17%, Mo = 2%. The smear: anisocytosis, hypochromic red blood cells. INR = 3.38. Quick Time = 20%, APTT = 41%. pH = 7.17; pO₂ = 157.3 mmHg, pCO₂ = 10.8 mmHg. HCO₃act = 7.6 mmol / l; HCO₃std = 13.6 mmol / l. BE = - 13.8 mmol / l, serum urea = 53mg/dl, serum creatinine = 1.31 mg / dl, Na = 126-132 mEq / l, K = 3.2 - 4.9 mEq / l, Cl=114mEq / l. Blood glucose = 42-89 mg / dl; PVC = 5cmH₂O; AST = 61u / l, ALT = 47u / l

ECG revealed tachycardia, HR = 142b/min, and shortening PR interval to 92 ms.

Blood tests run on the third day of hospitalization revealed: anemia, thrombocytopenia and leukopenia replaced by leukocytosis. E = 3230000/mm³, Hb = 8.8 g / dl, Ht = 25% ; HEM, HEM = 27pg, MCHC = 36%, MCV = 76. Platelets = 68000-84000/mmc, White blood cells = 13700-19400/mmc: in 24-11, Fl: PMNs = 76%, LF = 7%, Mo = 4%, basophils = 13%, then 25-11, FL: PMNs = 11% PMNs = 80%, LF = 6%, Mo = 3%

Glucose = 89mg/dl, serum urea = 35.8-50mg / dl, serum creatinine = 1.05 mg / dl. PVC-13cmH₂O = 5-10. pH = 7.44. Alkaline reserve = 19. Na = 126mEq / l. INR = 2.23, TQ = 33%, TH = 42 sec. AST = 524-675u / l, ALT = 128-346 U / l. AST, ALT returned to normal in 12/01/2011.

Urinalysis test: detected = albumin; Ubg = normal, bile pigments = absent.

Rotavirus antigen in stool = negative. Adenovirus Ag = negative.

Chest X-ray: " Normal heart. Lungs unchanged. "

CT skull: "Diffuse cerebral edema".

His neurologic examination in 23/11/2011 revealed: " Serious general condition. GCS: coma 6 / 2. Miotic pupils with

Photomotor Reflex present, poorly reactive, fever with stiff neck, OTI, mechanically ventilated. "

Pediatric Surgery examination, "Phenomena and blistering skin necrosis in both legs. Necrotic skin lesions in the fingers. The recommended treatment: Clorhexin to be applied on local area".

Treatment

Having a severe dehydration, the patient needed a proper fluid rebalancing. Being comatose, with O₂ saturation (determined by pulse-oximeter) of 76%, it has been required mechanical ventilation, for 5 days. Severe hypotension (40/30 mm Hg.), in the second day of admission, imposed both an adequate fluid rebalancing, supine and proper administration of Dopamine and Norepinephrine(1), 5µg/kg/min. These are the antibiotics that were sequentially used: Sulcef, Meropenem, and Ceftriaxone combined with Metronidazole and Ciprofloxacinum.

Clinical evolution was favorable, fever reducing gradually in 4 days. After the first day of hospitalization, he has not presented vomiting anymore, but had diarrheic stools still three days. Five days later, he regained consciousness without disabling motor deficits. Due to mechanical ventilation, he presented subcutaneous emphysema as a transient incident. In evolution, he presented hepatomegaly for 7 days and ischemic skin lesions, consecutive septic shock status.

Because of the lesions diagnosed „dry gangrene" to toes, he has been transferred on the 14th day to Pediatric Surgery Department, where he received antibiotics, local treatment, being discharged on the 18th day from admission.

Photos: lesions after septic shock of legs and toes of male patient.





Discussion

Shock means acute circulatory deficiency, which leads to a poor tissue perfusion, inadequate for cellular needs.

Septic shock is a pathogenic form having in substrate intricate mechanisms (hypovolemic, vasogenic).

Sepsis - concept that refers to those situations in which a clinically proven or suspected infection localized or disseminated, is accompanied by a systemic inflammatory response in the body (SIRS) (2). SIRS includes at least two of following: fever (or hypothermia), leukocytosis (or leukopenia), tachypnea, tachycardia (14).

In this case has been a SIRS (presence of fever, leukopenia/leukocytosis, tachycardia) induced by an infection-gastroenteritis.

As a particularity of this case we can mention a large percentage of basophils. It may appear in various inflammatory diseases as: inflammatory bowel diseases (in this patient was acute gastroenteritis), upper airway disease, chronic dermatitis, in viral infections, infectious endocarditis.

Sepsis with negative cultures means SIRS and empiric antibiotic treatment for clinically suspected infection, but that all cultures are negative(11). In this case, infection was clinically suspected, not proven by laboratory investigations. Leukocytosis with increased of PMNs, is usually induced by a microbial infection. We suspected a gram negative infection.

Due to coma and hypotension, even a severe one, 40/30mmHg (second day of admission), in despite of proper fluid rebalancing and due to its persistence more than an hour of SBP<80mmHg, we can diagnose septic shock (SS).

SS is a serious clinical -biological syndrome triggered by different pathogens and / or their products (especially endotoxins). Free LPS attaches to a circulating LPS-binding protein, and the complex then binds to a specific receptor (CD14) on monocytes, macrophages, and neutrophils. Engagement of CD14 (even at doses as minute as 10 pg/mL) results in intracellular signaling via an associated "Toll-like receptor" protein 4 (TLR-4), resulting in profound activation of mononuclear cells and production of potent effector

cytokines such as IL-1 and TNF- α . These cytokines act on endothelial cells and have a variety of effects including reduced synthesis of anticoagulation factors such as tissue factor pathway inhibitor and thrombomodulin. Endotoxins play leading role as trigger in sepsis, septic shock and MODS, adhering to platelets by PAF, by vasoactive Kinines, inducing DIC- with thrombocytopenia, consumption of coagulation factors \rightarrow a marked alteration of organs infusion \rightarrow small thromboses \rightarrow installation of septic shock \rightarrow MODS (multiple organ dysfunction syndrome) and eventually death (9). In clinical analysed case, MODS was developed and proved by hepatic cytolysis syndrome, by need for prolonged mechanical ventilation (more than three days) and by second level coma that lasted for five days (5;7).

Endotoxins lead to activation of proinflammatory cytokines (especially IL-1, IL-6, TNF α), which, released in bloodstream, produce vasodilators and damaging to the capillary endothelial cells. Vasodilators lead to hypotension and hypoperfusion of skin and abdominal viscera. The capillaries can be obstructed by proliferation of WBC that have invaded area to attack bacteria and will cause further cell damages, creating area of tissue ischemia, with insufficient infusion of tissue, that characterizes septic shock.(3) This is the explanation of „dry gangrene” at patient's feet. It is, also, released nitric oxide (NO) by damaged vascular endothelial cells. NO was shown to reach high levels in septic shock, producing vasodilation and vasoplegia(4). NO and vasoplegia may be the explanation for life-threatening low blood pressure (40/30 mmHg) measured in our analysed patient, who was enough hydrated in the second day of admission. It was not a hypovolemic shock, but a certain septic shock.

Altered microcirculation is central pathophysiological factor of shock's progression to MODS (11). Low flow syndrome induces ischemic lesions. Extensive lesions of the soft parts may be a trigger that activate humoral systems. They precede the activation of cellular systems, as in sepsis. Thus, disturbances of peripheral irrigation are amplified,

showing a genuine "vicious circle" infection - ischemic lesions – infection (12).

Syndrome "low flow" produces an increased pulmonary release of mediators that contribute to severe endothelial cell damage, especially in the lungs (6). This might explain low oxygen saturation (76%) in a patient without respiratory infection.

DIC was proven by clinical and laboratory findings (INR = 2.23, TQ = 33%, TH = 42 sec, thrombocytopenia). DIC's activation by endotoxins and other humoral factors will produce small thromboses that aggravate hypoxia, contributes to the morphological substrate of organ failure(4).

There was a transient and minor injury of kidneys (a high serum urea and creatinine level). The damage of liver

(which became enlarged for 7 days) was more important, proven by high levels of ALT, AST, for a week.

Conclusion

In conclusion, our patient was admitted in the Intensive Care Unit with sepsis due to a gastroenteritis, followed by septic shock (a redoubtable complication) and reversible MODS. However, his evolution was a favorable one, unto healing, but with disabling ischemic lesions in the legs caused by microcirculation disturbances in the context of a septic shock low blood pressure. One particularity of the case was: the presence of a large percentage of basophils.

„Happy-end” particularity of this case was a "restitutio ad integrum" healing of the organ damage and his complete recovery after septic shock with severe hypotension.

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