SEVERE SEPIS AND INCIPIENT RENAL FAILURE IN A TYPE 1 DIABETES CHILD

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Abstract

The authors present the case of a 15-year-old girl with a brutal onset of juvenile diabetes in the context of multiple organ dysfunction syndrome (MODS). The symptoms appeared suddenly: vomiting, diarrhea, hyperthermia, dehydration symptoms. Two days after admission, were noted diabetic coma symptoms (dry skin, fruity breath odor, confusion) associated with Bullous Pemphigoid lesions on the surface of left hemibody, right basal pneumonia with both acute respiratory and kidney failure. After three weeks of intensive treatment, the evolution was slowly favourable: renal function recovering, blood sugar level stabilization and epithelialization of skin lesions.

Usually, infections may precipitate type 1 diabetes onset in children and teens. Severe infections and sepsis could be associated with high sugar levels that are stabilized as soon as infection is solved. The particularity of this case consists of permanent type 1 diabetes, although the sepsis has been solved.

Keywords: diabetes mellitus, sepsis, MODS.

Introduction

Sepsis is the systemic response to infection and is defined as the presence of SIRS (systemic inflammatory response syndrome) in addition to a documented or presumed infection.

MODS (multiple organ dysfunction syndrome) means at least two organ failure and it requires the presence of the following criteria (in the context of infection) or the patient must meet one or more of the following conditions in the last 24 hours (1,2,3):

- Cardiac: heart rate (HR) <54/min, mean arterial blood pressure <49 mmHg, serum pH <7.24 or Pa O2 <49 mmHg, or ventricular tachycardia to fibrillation;
- Haematology: WBC <1000/mm3, platelets <20000/mm^3, Ht <20%, Hb <7.5 g/dl;
- Renal: urine output <479 ml/24h or <159 ml/8 h, blood urea >100 mg/dl, serum creatinine >3.5 mg/dl;
- Respiratory: respiratory rate <5/min, PaCO2 >50 mmHg, or ventilator dependency > three days;
- CNS: Glasgow Coma Scale <5 (no sedation). Neurological dysfunction - agitation, disorientation, altered state of consciousness to schoolchildren.

At these conditions could be added at least one of the following (2): gastrointestinal signs: loss of appetite, vomiting, diarrhea, to acute gastrointestinal bleeding due to ulcer (caused by infectious stress), or dynamic ileus, intense meteorism. Clinical consequences of acute liver failure may include: jaundice, hepatosplenomegaly, total bilirubin level >4 mg%, ALT >2 x normal.

Insulin-dependent juvenile diabetes, can develop anytime, but generally occurs in children or young adults. The onset of the disease often follows a viral infection. As for the clinical case described below, it appeared in the context of sepsis.

Case report

We present the case of a young girl, 15 years of age (fig.1), who was admitted to the Pediatric Clinic Emergency Hospital Craiova on March 2010 (no. 11680).

The family does not report chronic diseases. She is the second child of young parents (35 and 39 years old); she was born at term, with birth weight 2600g. She was breastfed for the first six months and then nourished diversified, being weaned at three years only. After three years presented numerous (treated at home) respiratory tract infections; in school period was uncommon. The onset of acute illness was suddenly, with repeated vomiting, watery diarrhea, frequent abdominal cramps, asthenia, hypodinamism, and high fever (39-40°C), these symptoms appearing after eating a piece of chocolate. She was admitted to the Infectious Diseases Hospital where she has been treated with antibiotics and intravenous fluids for rebalancing. After two days she went into medium level coma, blood sugar level was 501mg/dl, urea 206 mg%, creatinine 4.3 mg%, ALT = 97 U/l and she was transferred to the Intensive Care Unit, where she kept undergoing insulin treatment and intravenous fluid rebalancing.

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Clinical characteristics were described as follows: height 156 cm, weight 49 kg, unconscious, suffering, ringed face, dry lips, oral-labial herpes, with necrotic lesion on her left flag, giant bullous lesion on her left calf (fig. 1), confluent petechial elements, disseminated both on the surface of the trunk and limbs, HR 99 b/min, BP 116/84 mm Hg, distended abdomen, moderate meteorism, loss of appetite, oliguria (<800ml/24 hours), she had no stool and no signs of meningeal irritation. Evolution in this patient presented eyelid swelling and legs' edema.

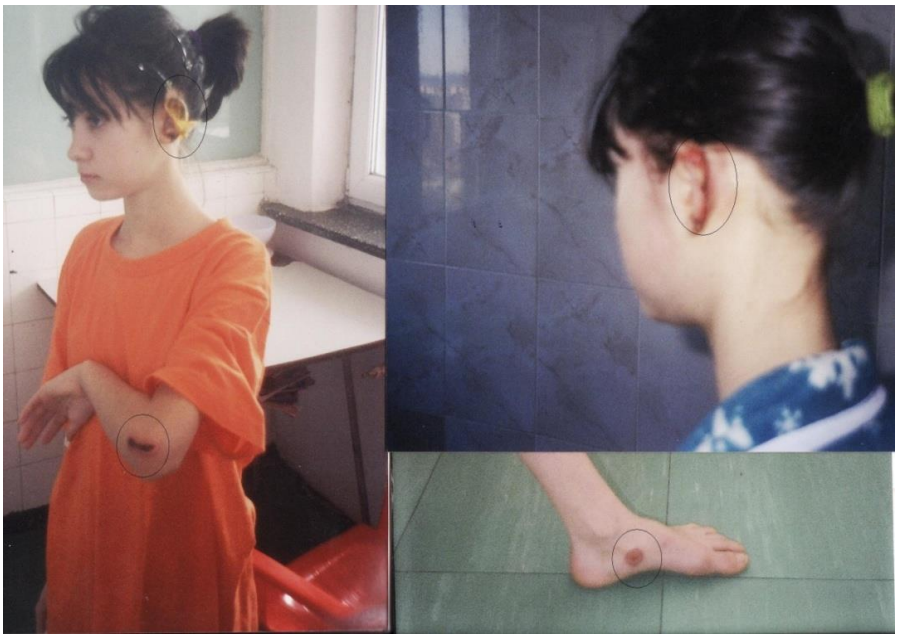

Fig.1: The teenager-patient suffering of diabetes and sepsis: skin necrotic lesions.

Laboratory investigations:

- Hemoleucogram was modified as follows:

Date	Hb(g%)	Ht (%)	Plt (mm^3)	WBC (mm^3)	Gs (%)	Lf (%)	Nn (%)
03 Mar 2010	11.6	32	191.000	14.300	15	15	5
04 Mar 2010	10.5	27	274.000	20.000	78	13	4
05 Mar 2010	9.2	27	299.000	15.800	82	10	4
08 Mar 2010	9.3	27	534.000	12.900	66	16	12
11 Mar 2010	8.8	27	413.000	11.600	65	18	9
16 Mar 2010	8.8	27	260.000	6.600	45	42	8

- ESR: 10 Mar 2010: 75/100 mm; 11 Mar 2010: 65/94 mm; 16 IMar2010: 40/60 mm.

- Urea and creatinine ranged as follows: urea rose steadily from 182 to 229 mg/dl and has been normalised (38mg/dl) after two weeks of treatment. Creatinine increased from 4.10 mg/dl at 6.53 mg/dl, then has reached the normal value of 0.81 mg/dl at discharged. Creatinine clearance: 44.12 ml/min.

- Exam Urinalysis: albumin fine trails, rare flat epithelium, leukocyte common, rare red blood cells. Urinary density 1005.

- Profile glucose: ranged between values 75-501mg/dl;

- Cholesterol 392mg%, triglycerides 265mg%, lipids 1078mg%;

- Astrup: Ph 7.31, pCO_2 29.3 mmHg, pO_2 25.9 mmHg, Bicarbonate 14.7; BE -10.2; Na 138 mEq/l, K 3,4 mEq/l, Cl 98 mEq/l.

- T Quick 40%, T Howell 140";

- Ex. pleural fluid cytology: relatively common mesothelial cells isolated and grouped, a few with hypertrophied nucleus, relatively rare, bare nuclei, relatively rare granulocytes leukocytes, red blood cells frequently.

- Chest X-ray: Consistent opacity with moderate, vague outline in the upper segment above basal right lower lobe, pulmonary type is opaque. At lung's base: opacities diffuse band with medium intensity.

- An abdominal ultrasound scan was performed: *no* intra-*abdominal pathology* was encountered.

- The patient's *hydroelectrolytic and acido-basic equilibrium* was restored, a dietetic therapy for the

management was provided along with a hyposodium and hypoproteic diete. Her medical treatment consisted of insulin therapy (1UI/kd/day), antibiotics, diuretics and Heparin. The dressings' lesions in her left calf have been daily changed.

After three weeks of intensive treatment, the evolution was slowly favourable: renal function recovering, blood sugar level stabilization and *epithelialization of skin lesions.*

At discharge, the patient has been recommended chronic treatment with insulin (4 doses), along with calculated carbohydrate diet and protein restriction on 35g/day for 1 year. A regular monitoring of renal function has also been recommended.

Discussions

Pathogenesis of type 1 diabetes is caused by autoimmune destruction of insulin-producing beta cells of the pancreas, usually after a viral infection. In this case, we have no data that would reveal a history-specific symptoms: polyuria, polydipsia, polyphagia with weight loss in the last month before admission. High sugar level was detected, two days after the abrupt onset of digestive disease. Serious gastrointestinal infection is likely to have precipitated the onset of diabetes or whether it is possible to produce it, due to severe sepsis. Severe sepsis is often associated with transient hyperglycemia (4), for many patients.

Diagnosis of sepsis was based on the presence of digestive infection as well as lung and skin one which pleads for SIRS criteria: onset of fever, leukocytosis over 12000 with a maximum of 20000 /mm^3 and tachypnea.

We suppose that the infection has been caused by gastrointestinal gram-negative bacteria whose endotoxins were spread to the bloodstream. These triggers stimulated inflammatory cytokines, interleukin such as: IL1, IL6, IL8, TNF, initiating the cascade of disseminated intravascular coagulation, both in macro and microcirculation. There has been also a direct toxic effect on the lung and kidney (5,6,7). Multiple organ dysfunction occured, as presented, at least for 24 hours: pneumonia, kidney failure, metabolic acidosis, vigil coma. Since the infection has been cured and endotoxins have been removed from bloodstream, renal function was recovered entirely.

She has also been discharged with insulin-dependent diabetes. After 2 years from MODS, girl teenager is equilibred. The single hospitalisation during this period was for a diabetic coma, one year ago.

Severe sepsis is quite frequent in intensive care units, being associated with a high rate both of morbidity and mortality. Usually, patients suffering from chronic diseases, such as: chronic hepatitis, HIV infection, cancer, are predisposed to sepsis and severe sepsis. Also, diabetic patients have high prevalence for infections as well as for sepsis (8,9).

Various authors have studied the pre-existing impact upon organ failures in sepsis and reached the conclusion that the diabetic patients develop kidney failure more frequently than the non-diabetic persons (10,11). However, there are still uncertain the causes that could influence these connections. It is very important though, to identify high-risk groups for acute organ failure that the mechanism involved might be understood and the treatment for these patients might be improved.

References

1. Angelescu N. Tratat de patologie chirurgicală, vol. 2, pag. 2120-2122.
2. Grigorescu-Sido P. – Tratat elementar de pediatrie – Editura Casa Cartii de Stiinta Cluj, 2000, vol. I pg. 91-92, vol. 4 pg. 441-450
3. Purcaru F, Georgescu I, Ciurea P, Cupşa A. – "Sepsis. Şoc septic. Disfuncţii multiorganice" (MODS) – Editura Medicală Universitară, Craiova, 2000, pg. 12-15; 11-13;231-274
4. Abraham E, Matthay MA, Dinarello CA. Consensus conference definitions for sepsis, septic shock, acute lung injury, and acute respiratory distress syndrome: time for a reevaluation. Crit Care Med. Jan 2000; 28(1): 232-5.
5. Burdette S.D, Parillo M.A. Systemic Inflammatory Response Syndrome, 2009, E Medicine.medscape. com/article168943.
6. Cupşa A – Boli infecţioase transmisibile. Ed. Medicală Universitară, 2007, pag:3.1-3.26;14.1-14.21.
7. Dellinger RP, Carlet JM, Masur H, et al. Surviving Sepsis Campaign guidelines for management of severe sepsis and septic shock. Crit Care Med. 2004;32(3):858-73.
8. Goldstein B, Giroir B, Randolph A. International pediatric sepsis consensus conference: definitions for sepsis and organ dysfunction in pediatrics. Pediatr Crit Care Med. 2005 (1):2-8.
9. Harrison, Munford R.S. Principii de medicină internă, 2005, pg 852-857.
10. Esper MA, Moss M, Martin SG. The effect of diabetes mellitus on organ dysfunction with sepsis: an epidemiological study. Critical Care 2009, 13:R18 (doi:10.1186/cc7717).
11. Kaplan SL. Bacteremia and septic shock. In: Textbook of Pediatric Infectious Diseases.2004:810-25.

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