Brief report

Septic shock caused by group G \( \beta \)-haemolytic streptococci as presenting symptom of acute myeloid leukaemia

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Abstract

A patient with rapidly fatal septic shock caused by group G \( \beta \)-haemolytic streptococci as presenting symptom of acute myeloid leukaemia is presented. Although the association of septic shock due to Group G \( \beta \)-haemolytic streptococci and different kinds of malignancy is known, presentation of acute myeloid leukaemia in this form is rare.

Keywords: Group G \( \beta \)-haemolytic streptococci; Septic shock; Acute myeloid leukaemia

1. Introduction

Haemolytic streptococci represent a minority of micro-organisms that can cause septic shock. Although mortality due to infections by haemolytic streptococci is dependent on the Lancefield group, underlying disease is of great importance considering the outcome of the disease state [1]. We present a patient in whom a rapidly fatal septic shock caused by Group G \( \beta \)-haemolytic streptococci (HSGG) was the presenting symptom of acute myeloid leukaemia.

2. Case history

A 41-year-old male patient with no relevant clinical history complained a week before admission of pain in his right shoulder and both calves. A trauma a few days before was thought to be responsible and diclofenac was prescribed. Four days after the beginning of his complaints he became feverish with chills, but no temperature was taken and no medical aid was sought. Two days after the chills had occurred he started vomiting, became progressively restless and dull, and was sent to the emergency department.

On admission we saw a restless man with whom no conversation was possible. His limbs were cyanotic, and haematomas on the right shoulder,
upper arm and elbow and left thigh were seen. There were no petechiae and no signs of skin abrasions, erysipelas or cellulitis could not be found and the skin between the toes was intact. Temperature was 37.2°C, blood pressure 100/60 mmHg, pulse frequency 120 beats per minute. Pathological lymph nodes were not palpable and there was no sign of meningism. Investigation of heart and lungs was normal, bowel sounds were normal and no signs of peritonitis could be found.

Laboratory values were as follows: Hb 5.6 mmol/l, Ht 0.27 l/l, WBC $25.7 \times 10^9$/l (myeloblasts 2%, promyelocytes 12%, myelocytes 5%, metamyelocytes 20%, band forms 15%, segment forms 7%, lymphocytes 22%, monocytes 17%), no Auer rods. Urea 19.0 mmol/l, creatinine 174 \mu$mol$/l, LDH 1360 U/l, CPK 5226 U/l, platelets $< 10 \times 10^9$/l, fibrinogen 405 mg/l, FDP 95 mg/l, APTT > 272 s. Blood gas analysis: pH 7.31, pO$_2$ 15.5 kPa, pCO$_2$ 1.6 kPa, base excess $-16.6$ mmol/l, bicarbonate 6.0 mmol/l.

Urinalysis by means of dipstick was positive for bacteria. Chest X-ray on admission was normal, X-ray of the right shoulder showed swollen soft tissue but no fractures. Ultrasonography of the abdomen revealed no abnormalities in the pancreatic region or kidneys.

Septic shock with diffuse intravascular coagulation was diagnosed. After blood cultures were taken, intravenous antimicrobial treatment with piperacillin (4 g 3 times daily) and gentamicin (120 mg twice daily) was initiated. Almost immediately after admission the patient became hypotensive, for which he was subsequently treated with plasma expanders, packed red blood cells, fresh frozen plasma, thrombocytes and vasopressors. Mechanical ventilation was necessary. A chest X-ray taken 12 h after admission showed diffuse pulmonary oedema. On the morning after admission blood cultures grew \beta-\haemolytic streptococci of Lancefield Group G. Susceptibility testing by micro-broth dilution showed the organism to be susceptible to piperacillin (MIC $< 0.5 \mu$g/ml). Urine culture remained negative. Twenty hours after admission the patient died due to multiple organ failure. The post-mortem investigation revealed petechiae, ecchymoses and haematomas in the skin, conjunctivae, pleurae, lungs, heart, thyroid gland, kidneys, bowels and testes. There were signs of cardiomyopathy. All organ cultures remained negative. Analysis of bone marrow showed massive infiltration of leukaemic cells, compatible with acute myeloid leukaemia. There was little leukaemic infiltration of liver and spleen.

3. Discussion

HSGG are normal inhabitants of the upper airways, gastrointestinal tract, female genital tract and skin, although a restriction is made by some authors to areas of moist skin such as toe webs and perineum [2].

Infections caused by HSGG usually present as pharyngitis or superficial skin lesions, but more serious infections such as endocarditis, meningitis, arthritis, osteomyelitis, sacro-iliitis, pyomyositis and endophthalmitis have been described [3–10]. Clinically, these often severe infections resemble those caused by Group A and B streptococci.

HSGG are a rare cause of septic shock. Percentages less than 2 have been reported [1,11,12]. In our hospital, during the period 1988–1992 only 15 (0.2%) out of 6982 positive blood cultures grew HSGG.

It has been reported that septic shock due to \haemolytic streptococci can occur without any visible skin lesion. Several case studies have yielded conflicting results concerning the presence of skin lesions in patients with septic shock due to HSGG [12–16]. Predisposing factors such as alcoholism, diabetes, high age and neutropenia may be responsible for a facilitated entrance of bacteria. Our patient had a history of a recent trauma that might have caused skin lesions as possible portal of entry, but on physical examination no such lesions could be found.

Whereas infections caused by Group A streptococci can occur in otherwise healthy persons, infections or septic shock caused by HSGG have been reported to occur in the presence of underlying disease states such as malignancies [17]. Septic shock as presenting symptom of a previously undiagnosed malignancy, however, is rare.
To the best of our knowledge there has only been one other report describing a patient with septic shock due to HSGG as presenting symptom of acute myeloid leukaemia [13]. This patient survived his septic shock and was subsequently treated for leukaemia. Our patient was a young and previously healthy man who was admitted in an almost moribund condition. While examining a peripheral blood slide shortly after admission, the diagnosis of acute leukaemia was considered, but no bone marrow study was performed because this would not have had therapeutic consequences at that time. Histological confirmation of our suspicion of acute myeloid leukaemia was attained by post-mortem investigation.

Treating infections caused by HSGG is not different from treating infections caused by haemolytic streptococci of other Lancefield groups, although an exception has to be made for Group D streptococci or enterococci which are not susceptible to penicillin. Antibiotics as penicillin G, vancomycin, first-generation cephalosporins, erythromycin and piperacillin are active against HSGG.

In our hospital any patient suspected of having septic shock is treated with a combination of a broad-spectrum β-lactam antibiotic and an aminoglycoside. This combination gives rapid adequate blood concentrations and a synergistic bactericidal effect has been observed in vitro [18]. We conclude that septic shock caused by HSGG as presenting symptom of acute myeloid leukaemia is a rare entity that adds to the previously described association of septic shock caused by these bacteria and different kinds of malignancies. Any patient diagnosed of having septic shock caused by HSGG requires a full investigation to search for underlying malignancy.

References