The Journal of Emergency Medicine, Vol. 57, No. 3, pp. e91–e93, 2019

0736-4679/$ - see front matter

https://doi.org/10.1016/j.jemermed.2019.06.020

Visual Diagnosis in Emergency Medicine

CLINICAL REPORT OF AN UNUSUAL LIFE-THREATENING COMPLICATION OF GINGIVOSTOMATITIS

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CASE REPORT

A previously healthy boy aged 15 months presented to the emergency department (ED) with a 3-day history of fever, ulcerative and vesicular perioral lesions, and refusal to drink and eat, without improvement despite topical therapies. The patient had come to the ED 2 days earlier with good health and normal vital signs, so supportive care and recommendations were indicated, including maintenance of hydration and topical treatment.

He was born at term, his medical history was not remarkable, and he was appropriately immunized for his age.

On physical examination he was febrile (38.2°C), tachycardic (170 beats/min), and tachypneic (38 beats/min). Blood pressure was 86/51 mmHg, and rapid blood glucose was 45 mg/dL. His weight was 9.200 kg. He presented multiple oral ulcers and erosions involving buccal mucosa, tongue, posterior pharynx, and gingival and palatal mucosae (Figure 1). Other physical examination was normal.

As signs of clinical sepsis were present, a peripheral venous access was obtained and a rapid fluid bolus of 20 mL/kg of normal saline solution was administered, followed by empirical antibiotic therapy. Hypoglycemia was corrected by rapid i.v. infusion of dextrose. Complete blood cell count revealed leukopenia (white blood cell count of 2900 cells/μL, with 5.9% neutrophils, 66.6% lymphocytes, and 26.1% monocytes), hemoglobin concentration of 10.4 g/dL, and platelet count of 199,000 platelets/μL. Serum biochemistry showed C-reactive protein level of 362 mg/L (normal, < 3 mg/L), procalcitonin level of 35 ng/mL (normal, < 0.5 ng/mL). Hemostasis parameters were also altered (plasma prothrombin activity 46%, partial thromboplastin time 58 s) and venous blood gas showed acidosis (pH 7.32, CO_2 31 mm Hg, bicarbonate 16 mmol/L, base excess 6 mmol/L, and lactate 2 mmol/L). In spite of rapid fluid resuscitation (a total of 40 mL/kg) and administration of antimicrobial therapy (cefepime, clindamycin, and acyclovir), the patient continued to be tachycardic, with decreasing blood pressure levels (70/30 mm Hg), so he was transferred to the pediatric intensive care unit.

On arrival to the pediatric intensive care unit, blood pressure support was transitioned to epinephrine and norepinephrine continuous infusions and hydrocortisone was added. A red blood cell transfusion and fresh frozen plasma were administered for the management of coagulopathy. The antimicrobial therapy was empirically replaced by meropenem, vancomycin, amikacin, micafungin, and acyclovir.

Twenty-four hours after admission to the pediatric intensive care unit, *Streptococcus oralis* was isolated

RECEIVED: 25 February 2019; FINAL SUBMISSION RECEIVED: 8 June 2019;
ACCEPTED: 15 June 2019

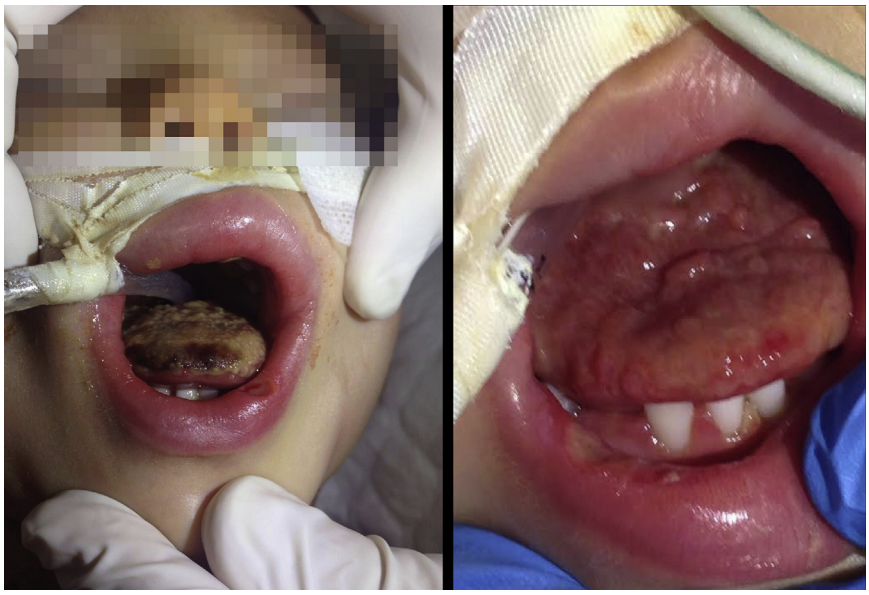

Figure 1. Extensive ulcerative and vesicular lesions of the gingiva and mucous membranes of the mouth.

from the blood culture collected in the ED. The bacterial culture from the ulcers also grew *Streptococcus oralis.* In addition, enterovirus was detected in the ulcerative lesions. Subsequently, he made a progressive full recovery, with complete resolution of the anomalies in the blood tests. He was transferred to an ordinary ward on hospital day 7 and was discharged home on hospital day 17. An exhaustive immunological study was performed that showed no abnormalities.

DISCUSSION

The main clinical manifestations of gingivostomatitis are ulcerative and vesicular perioral lesions, fever, and refusal to drink or eat (1,2). Sometimes, complications such as dehydration (most frequently), laryngitis, esophagitis, pneumonitis, or bacterial infection, may appear (3).

The oral mucosa lesions might serve as a port of entry for opportunistic pathogens, which are usually part of the normal human oral microbiota (4,5). These bacteria can benefit from the immunologic situation of the patient and lead to bacterial complications and even cases of septic shock (6).

Bacteremias caused by group A *Streptococcus, Staphylococcus aureus*, and *Kingella kingae* have been reported as a complication of herpetic gingivostomatitis (4,5,7). These micro-organisms are considered normal components of the oral flora in young children. They presumably invade the bloodstream when the anatomic integrity of the mucosal surface is damaged by the viral infection and they are capable of pathogenicity, so pediatricians should be aware of bacterial complications in children with stomatitis.

Streptococcus oralis (the agent isolated in our patient both in the blood culture and in the culture from the ulcers) is a human oral colonizer and an opportunistic pathogen that belongs to the viridans group streptococci, so in cases of altered integrity of the oral mucosae, it can lead to bacteremia (5,8).

Our case illustrates a rare but potentially serious complication of a common pediatric infection. It emphasizes the fact that gingivostomatitis is usually a benign and self-limited process in healthy children, but occasionally, lesions of the oral mucosa might serve as a port of entry of opportunistic pathogens leading to bacterial life-threatening complications. Although this bacterial complication usually affects immunosuppressed patients, it can occur in previously healthy children. Early recognition and prompt administration of i.v. therapy are essential.

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