

Nose Necrosis in Female Shocked Patient: Conservative Treatment with Heparin

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Introduction

Acute skin failure is an event in which skin and underlying tissue die due to hypoperfusion concurrent with a critical illness [1]. Usually skin hypoperfusion is a late symptom in a shock setting and it is present in patients with an advanced multiorgan failure (MOF) dysfunction. Acute skin damage is mainly present in feet and hands as consequence of distal hypoperfusion due to hypotension or microthrombotic events [2]. Face is less commonly affected. In some cases of purpura fulminans there is anyway an important face skin involvement. Purpura fulminans is usually associated with meningococcal sepsis, varicella, and pneumococcal infections and/or coagulation disorders [3-4]. We present the clinical case of a young woman occurring at our hospital with nose skin necrosis as first sign of shock setting.

Case History

A 38 years old woman presented at the Emergency Department in sleepy state, generalized malaise and fever unresponsive to paracetamol. The patient had marbling on the extremities and on the face. The anamnestic investigation did not reveal anything of clinical significance. After 24 hours, the clinical picture turned into septic shock with intravascular disseminated coagulation. Marbling of the extremities persisted and on the nose it progressed in partial necrosis. Clinical blood and cerebrospinal fluid samples tests were negative; anyway empiric therapy with vancomycin and meropenem was started. Renal and hepatic function resulted compromised. Blood support circle (noradrenaline and dopamine therapy) was administered for the first 3 days. Breathing support was necessary for 2 days, and then only occasionally oxygen support was requested. Subcutaneous therapy with enoxaparine was administered (4000 UI per day) until discharge. Single dose of Antithrombin III was given on third day. After 72 hours the patient quickly recovered. The clinical picture and the multiorgan failure state progressively improved and patient was transferred from intensive care to infective department. The specialist haematological consultation and all tests carried didn't revealed pathologies of haematological branch.

Three days after recovery the plastic surgeon was called for the evaluation of facial injuries. The patients showed a full thickness necrosis of the skin of the tip of nose and faded marbling of the rest of the nose and cheeks (Figure 1). Conservative treatment was chosen and started with daily topic applications of "Epsodilave 250 UI / 5ml" (sodic heparin) and greasy gauze dressing. Two days after, the necrosis showed an improvement and it was limited to the more distal areas of the nose (Figure 2). The topical treatment continued for 18 days until the patient was discharged. No surgical or chemical debridement was carried out. Extremities marbling had a spontaneous regression. One month later, the patient came to control showing a complete remission and resolution without significant scarring (Figure 3).

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Citation: Marco Stabile, Luca Rosato, Valeria Navach. Nose Necrosis in Female Shocked Patient: Conservative Treatment with Heparin. Archives of Clinical and Medical Case Reports. 7 (2023): 335-337.

Received: January 06, 2023

Accepted: February 07, 2023

Published: August 03, 2023



Figure 1: Full thickness necrosis of the skin of the tip of nose and faded marbling of the rest of the nose and cheeks.



Figure 2: The necrosis showed an improvement and limited to the more distal areas of the nose.



Figure 3: Complete remission and resolution without significant scarring.

Discussion

Acute skin necrosis due to severe and prolonged hypotension can be present in advanced shock settings. Usually extremities are affected, because of their distinctive vascular pattern. These signs become evident later respect to other shock typical symptoms [5]. In our case, skin failure was an early expression of critical illness status. Later the patient got worse in a MOF status, with compromised renal and hepatic function. The unique localization of the nose skin necrosis addressed clinicians towards a purpura diagnosis. All examination (blood coagulation pattern, absence of meningococcal infection in blood and cerebrospinal fluid) contradicted this hypothesis. The hematologist also excluded any disease of his clinical branch. We finally concluded the damage at nose skin was due solely to hypoperfusion. Nose has a rich vascularization from facial artery in the lower part and from angular branch in the upper part. All these branches anastomose at the lateral sides. Nasal tip lacks anyway of a strong vascularization and it is more prone to hypoperfusion.

There are not standardized treatments for acute skin failure, maybe also because in a life threatening setting, treatment of skin lesions is not considered relevant. Minor lesions can regress after reset of blood circle, but established skin necrosis needs specific treatment. The nasal soft tissue envelope is composed of fat, muscle, overlying superficial musculoaponeurotic system, and skin. The caudal half of the nose contains also a higher density of sebaceous glands [6]. Surgical debridement in case of nose skin necrosis is commonly discouraged. The deeper sebaceous layers especially at the tip, have an important regenerative capacity, so conservative procedures are preferred. Many topical treatments are described in literature. Use of vasodilators such as ointment with nitroglycerine and systemic or topical treatment with pentoxifylline or prostaglandin E1 were used. Hyperbaric treatment is also described by some authors. The cost and the management of these treatments are not often suitable for critical illness patients. In our experience, we had good results in the topic use of heparin on burns and skin necrosis, that's why we routinely use it for cutaneous lesions that need conservative treatment.

Ng in his review of literature underlines that topic treatment with gel containing heparin improves or positively alter the microcirculation in normal skin at both deep and superficial capillary layers [7]. Also patients with microangiopathy and have a significant improvement after treatment with topical use of heparin. Heparin molecule is a long glycosaminoglycane. Apart its role in coagulation process, it has also anti-inflammatory property. For this reason heparin and its derivate are also used in the treatment of asthma, inflammatory bowel disease, cardiopulmonary bypass, and cataract surgery [8]. Many mechanisms are involved in the anti-inflammatory effect. Levels of

cytokines after heparin administration are decreased (IL 6, IL 8, TNF) and heparin also inhibits adhesion of leukocytes and neutrophils to endothelial cells.

The topical use of heparin on damaged skin has been long described in the treatment of burns by Saliba with excellent results in terms of healing; scarring and pain control [9-10]. At our advice the use of topical heparin is a valid treatment in case of skin damage due to vascular impairment. We recommend trying this before surgical debridement.

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