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The evolution of the Helsinki frostbite management protocol

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ABSTRACT

Background: Severe frostbite can result in devastating injuries leading to significant morbidity and loss of function from distal extremity amputation. The modern day management approach to frostbite injuries is evolving from a historically very conservative approach to the increasingly reported use of early interventional angiography and fibrinolysis with tPA. The aim of this study was to evaluate the results of our frostbite treatment protocol introduced 3 years ago.

Methods: All frostbite patients underwent first clinical and then Doppler ultrasound examination. Angiography was conducted if certain clinical criteria indicated a severe frostbite injury and if there were no contraindications to fibrinolysis. Intra-arterial tissue plasminogen activator (tPA) was then administered at 0.5–1 mg/h proximal to the antecubital fossa (brachial artery) or popliteal fossa (femoral artery) if angiography confirmed thrombosis, as well as unfractionated intravenous heparin at 500 units/h. The vasodilator iloprost was administered intravenously (0.5–2.0 ng/kg/min) in selected cases.

Results: 20 patients with frostbite were diagnosed between 2013–2016. Fourteen patients had a severe injury and angiography was performed in 10 cases. The total number of digits at risk was 111. Nine patients underwent fibrinolytic treatment with tPA (including one patient who received iloprost after initial non response to tPA), 3 patients were treated with iloprost alone and 2 patients received neither treatment modality (due to contraindications). The overall digital salvage rate was 74.8% and the Hennepin tissue salvage rate was 81.1%. One patient developed a catheter-site pseudoaneurysm that resolved after conservative treatment.

Conclusions: Prompt referral to a facility where interventional radiology and 24/7 laboratory services are available, and the combined use of tPA and iloprost, may improve outcome after severe frostbite.

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1. Introduction

Cold injuries are comprised of peripheral cold injuries and systemic hypothermia [1]. In Finland peripheral cold injuries are not infrequent with an incidence of 2.5 cases per 100 000 inhabitants, and is more common in men [2]. The yearly incidence in Finland correlates with the environmental temperature and is often associated with alcohol and other psychosocial factors [2–5]. The distal extremities are most commonly affected (90% cases) and the severity of injury ranges from a mild, superficial, reversible condition known as 'frostnip' to a deep tissue freezing injury known as 'frostbite' [6–10]. Severe frostbite can result in devastating injuries leading to significant morbidity and loss of function from frequent distal extremity amputation. Management of frostbite includes rapid rewarming with adequate analgesia. Surgical management of frostbite is delayed to allow full demarcation and to allow maximal recovery of tissues. Traditionally in most institutions the approach has been very conservative with distal amputations an inevitable fate in severe cases and a consequent negative impact on quality of life [4] (Fig. 1). However, over the last 10 years a major therapeutic advance has been made in the early use of fibrinolytic agents to reduce the incidence and morbidity associated with distal extremity tissue loss [11–18]. In addition, the vasodilator drug, iloprost (a synthetic prostacyclin analogue) has been used with some success [19–21].

In an attempt to reduce the morbidity associated with frostbite, in 2013 we introduced the use of fibrinolytics and iloprost for severe frostbite cases at risk of amputation. We herein present our experience using our modified frostbite management protocol.

2. Patients and methods

This is a retrospective, observational cohort study of patients admitted with severe frostbite to the Helsinki Burn Centre between 2013 and 2016. Our modified frostbite management protocol is illustrated in Fig. 2. In short, patients with a distal extremity frostbite injury are assessed for severity of injury and clinical signs of ischaemia after initial rapid rewarming. Severe frostbite was defined as a single digit with clinical signs of ischaemia up to the level of, or proximal to, the proximal interphalangeal joint (PIPJ) or interphalangeal joint (IPJ) of the thumb. Involvement of multiple digits with more distal levels of ischaemia was also categorized as severe frostbite. Patients with severe frostbite presenting within 48 h from injury were then considered for fibrinolytic therapy and after screening for contraindications (as well as a platelet count $>100 \times 10^9$/L and a haematocrit >30% as laboratory parameter prerequisites) to fibrinolytics (Table 2), promptly underwent angiography. Severe hypothermia was considered as only a 'transient' contraindication as it can usually be reversed rapidly, permitting subsequent angiography and possible fibrinolytic therapy.

Diagnostic angiograms were performed via a femoral arterial port and if a clear perfusion defect was seen at the middle phalangeal level (or more distally in multiple digits), fibrinolysis would be indicated. Initially, 3 ml of the vasodilator, papaverine (50 mg/50 ml) would be administered slowly to reduce any possible vasospasm. The fibrinolytic drug, alteplase (Actilyse®, Boehringer Ingelheim, US) would then be given intra-arterially via the brachial or femoral artery just proximal to the antecubital or popliteal fossae respectively, at a rate of 0.5–1 mg/h and the dose divided by the number of limbs affected. A bolus of 5 mg per affected limb would be initially administered and the maximum duration would be 12 h and a check angiogram would be performed at this stage or earlier depending on the clinical situation. Unfractionated heparin was also concurrently infused intravenously to prevent the formation of new clots and the propagation of existing thrombi, and the rate adjusted to the Activated Partial Thromboplastin Time (APTT) accordingly. Patients were monitored on the Intensive Care unit. Following the completion of fibrinolysis, low molecular weight heparin (enoxaparin 40 mg bd sc) (Klexane®, Sanofi, Paris, France) would be commenced 2–4 h after removal of the arterial port.

Intravenous iloprost (Ilomedin®, Bayer AG, Leverkusen, Germany) was considered in the event of contraindications to

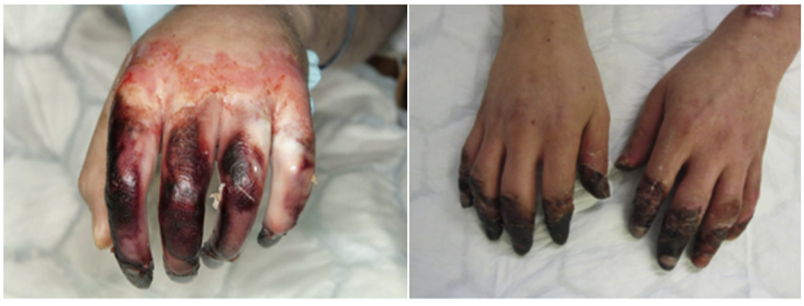

Fig. 1 – Two cases of severe frostbite with digital necrosis.