Rapidly Spreading Deep Dissecting Hematoma Occurring One Month After A Minor Trauma: A Case Report.

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Case Report

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Abstract

Background: Deep dissecting hematoma (DDH) is a rapidly extending blood collection that splits the hypodermis from muscle fascia, constituting a medical surgical emergency. The natural history of this condition includes trauma (even minor physical injury) shortly before onset of the lesion, occurring in a patient with advanced dermatoporosis. A delay of several weeks between the appearance of a superficial haematoma following a minor trauma and its sudden decompensation into a rapidly spreading DDH has been scarcely mentioned in the medical literature.

Case presentation: We report the admission of a 70-year-old woman under anticoagulation to the emergency department of our hospital for the sudden appearance of a rapidly evolving hematoma one month after a negligible trauma to the right leg. A complete skin examination revealed clinical signs (spontaneous superficial skin haematomas, lacerations, wrinkles, stellate pseudo-scars) of advanced dermatoporosis, especially on the forearms. The initial biological testing disclosed an International Normalized Ratio of 3.15. The clinical aspect of the haematoma, its rapid extension and the cutaneous signs of dermatoporosis on the forearms allowed the diagnosis of DDH. Bedside ultrasound examination was used to eliminate differential or additional diagnoses and to assess the main features of the hematoma (dimensions, existence of blood supply). Due the extent of the lesion and the risk of extended skin necrosis, surgical debridement and hematoma drainage were performed. The operative report confirmed the diagnosis of DDH. Wound healing was obtained spontaneously after three months.

Conclusion: DDH is the most serious complication of dermatoporosis. Given its rapid horizontal extension and the risk of skin necrosis it induces, DDH is a medical-surgical emergency and must be diagnosed early. This observation emphasises that in patients with severe dermatoporosis, on the occasion of a Vitamin K Antagonist overdose, a limb-threatening DDH can develop suddenly, even several weeks after a minor impact.

Background

Deep dissecting hematoma (DDH) is the pooling of blood under pressure which rapidly dissects the virtual space between the fascia muscle and the hypodermis [1,2]. It represents the most serious stage of dermatoporosis. Prompt management is needed to minimize the risk of skin necrosis. DDH is caused by profuse bleeding from fragile superficial vessels. Like intramural hematoma in acute aortic syndromes, dissection provoked by blood under pressure between two splitting layers extends the lesion [1].

The diagnosis of DDH is clinical. It is classically a hematoma extending rapidly in a patient affected by dermatoporosis, arising forthwith after a trauma. Medical imaging (magnetic resonance imaging [MRI], computed tomography scan or ultrasonography) may be required to confirm the diagnosis [1,2], particularly if medical history fails to explain the onset of the lesion. Long periods of time between the initial trauma and the appearance of a hematoma, a common main feature of chronic expanding hematoma [3], have rarely been reported in DDH [4–6]. We present a case of a limb-threatening right-leg

pretibial DDH in a patient with secondary dermatoporosis that occurred one month after a minor trauma during an overdose of Vitamin K antagonist (VKA).

Case Presentation

A 70-year-old woman was referred to our emergency department with a sizeable painful pretibial hematoma located in the outer aspect of the back of the lower right leg, ending just above the ankle (fig. 1). She was treated by fluindione 20 mg per day for three years for paroxysmal atrial fibrillation and pulmonary hypertension. The patient's body mass index was 20.7 kg/m^2, blood pressure 147/96 mmHg, heart rate 101 bpm, and temperature 36.5°C. No clinical signs were in favour of erysipelas. Neither weakness nor paraesthesia was observed. Pedis and posterior tibial pulse palpation was limited due to hematoma. Skin examination revealed senile purpura, atrophy, large lacerations and superficial haematomas located in the forearms (fig. 2). There were no other findings on physical and neurological examination.

A month prior, the patient had collided her outside of her leg just above the malleolus with the leg of a chair, resulting in a limited superficial hematoma. On the day of admission to the emergency department in the morning, the initial superficial hematoma suddenly expanded, reaching the ankle and the calf, causing pain and preventing weight bearing on the affected limb.

In addition to long-term anticoagulant therapy, medical history included:

- sarcoidosis treated by prednisone 8 mg/day for more than ten years,
- chronic respiratory insufficiency treated with long term oxygen inhalation therapy,
- osteoporosis highlighted by dual-energy x-ray absorptiometry two years previously,
- hypothyroidism,
- smoking cessation several years previously.
- Other daily treatments included bosentan, furosemide, levothyroxine sodium, a fixed dose combination of fluticasone propionate and salmeterol for inhalation, potassium chloride and estradiol patches.

Blood testing revealed: white blood cell count, 12.10/µL (with neutrophils: 10.30/µL); platelet count, 366/µL; haemoglobin, 13.40 g/dl (haematocrit: 42.40 %); C-reactive protein, 7.50 mg/L and estimated glomerular filtration rate, 93 mL/min per 1.73 m^2. Fluindione was last taken the day before. Haemostasis tests showed a thrombin time of 13.2 seconds, an international normalized ratio (INR) of 3.15, a prothrombin ratio of 28 % and an activated partial thromboplastin time of 42 sec, (ratio: 1.42). The INR measured in the previous weeks was always between 2 and 3.

X-ray at admission showed no underlying bone fracture. An arterial and venous Doppler ultrasound (US) examination found that arterial axes in the lower limbs were patent without stenosis, and there was no evidence for recent deep vein thrombosis (DVT). Soft tissue study revealed a hypo/heterogenous well-

delineated mass located between skin and fascia (fig. 3). Initial medical management involved fluindione discontinuation, administration of 5 mg of vitamin K and analgesics.

Four hours after admission, the haematoma had spread to the upper part of the calf (fig. 4). Pain was not alleviated by usual analgesics. DDH extension went on and reached dorsum of the foot (fig. 5). Finally, DDH spontaneously opened, allowing pain's relief. The day after in the morning, surgical treatment consisted in debridement, and hematoma drainage in aseptic conditions under spinal block. A bulky hematoma (15 x 20 cm with 5-6 cm thickness) localized between the fascia muscle and hypodermis was noted, confirming the diagnosis of DDH.

The day following surgery, the patient had dyspnea and haemoglobin was 8.4 g/dl (haematocrit 27.1%), so two units of packed red blood cells were transfused. Postoperative management included analgesia and daily local care including mechanical debridement and dressings. Biological examination of surgical samples found numerous red blood cells and no leukocytes. Bacteriological analyses were sterile.

The loss of substance was very extensive (fig. 6). The hospital stay was thirteen days long. Clinical and biological follow-up revealed a 4.5 kilogram-weight loss (two months after surgery) and depressive mood, hypoalbuminemia (CRP-corrected albumin less than 30 mg/l) and chronic anaemia. Wound healing was obtained spontaneously after three months without surgery for a skin graft.

Discussion

Data concerning the prevalence and incidence of DDH are rare. A three-year single-centre retrospective study, that included 2092 patients over 75 years of age, seen in dermatology or plastic surgery consultations after trauma, found an incidence rate of 53‰ (95% CI = 44 - 64) per year, and among 112 hematomas, 26 were DDH [5]. The differential diagnoses include erysipelas, DVT, non-dissecting hematoma and Morel-Lavallee syndrome [2,4]. Uncommon aetiologies should not be overlooked [6,9]. DDH is the most serious complication of dermatoporosis, usually arising immediately after an injury, often a minor one [2,5,7–9].

Dermatoporosis is a chronic phenomenon where the skin becomes frail and tears easily [10]. A French cross-sectional study including 202 hospitalized patients aged 60 years and older found that the prevalence of dermatoporosis in their population was 32 % [11]. Primary dermatoporosis, resulting from chronological aging and long-term sun exposure, is the most commonly encountered type. Secondary dermatoporosis is due to exogenous factors weakening the skin, mainly chronic use of topical and systemic steroids.

Depending on the extension, age and whether or not the haematoma is closed, Fennira et al. described four types of DDH (early closed type, advanced closed, advanced type with necrosis and open type) and highlighted rapid increase in the volume and extent of the haematoma as signs of severity [2]. Furthermore, although it has been reported under direct oral anticoagulants [9], the association of skin

frailty and VKA - for which cutaneous complications are well documented (fluindione or warfarin) [8] - or antiplatelet drugs [4] remains the classical situation where DDH has been observed [1,5].

A skin biopsy is not always required, but it ascertains the diagnosis by confirming the presence of degenerative changes in the dermal and subcutaneous vessels, and can help rule out cutaneous amyloidosis, vasculitis, bullous dermatosis, or bullous pemphigoid. In our case, even it is quite clear that the clinical manifestations of dermatoporosis were secondary to long-term corticosteroid therapy, one of the limitations of the observation is the absence of histopathological analysis. Other differential diagnoses were eliminated by clinical examination, Doppler US and biological analyses.

According to Kayak et al., at advanced stages of dermatoporosis, where there is a loss of the skin's viscoelastic properties, a "fracture" of the dermis may occur after minor trauma, leading to DDH [10]. In our case, a negligible trauma caused a superficial hematoma which did not resorb (favoured by anticoagulation therapy) and decompensated spontaneously into DDH (due to dermis fragility) after a one-month quiescent phase. Eventually, after a few hours and favoured by overdose of VKA, an early-closed type DDH developed to an advanced closed type and then to an open type.

In Kaya et al., all patients had an MRI to assess the extension of the lesion, described as a bright structure between the muscular fascia and hypodermis [1]. MRI is a highly useful examination but with limited accessibility in emergency settings. US is widely accessible, but the results are dependent on the operator's skill level. In this case, management included a bedside ultrasound scan which provided valuable information (the depth of the hematoma, blood supply) allowing the medical team to rule out the differential diagnoses and to evaluate the prognosis for wound healing. Few studies have reported the utility of bedside ultrasound examination in this context [12].

Conclusion

DDH is the most serious stage of dermatoporosis. It constitutes a medical surgical emergency. Given the risk of rapid extension and subsequent skin necrosis, emergency department staff should be able to identify this type of hematoma and initiate appropriate management as soon as possible. A long interval between the initial minor trauma and the rapid extension of the lesion may exist, posing a diagnostic challenge for the treating physician. To the best of our knowledge, this issue of time has received little attention in the few studies focused on DDH. We report a case of DDH occurring one month after a minor trauma, complicating an iatrogenic secondary dermatoporosis in an acute mode during a VKA overdose, and requiring medical surgical management.

Abbreviations

DDH: Deep Dissecting hematoma; MRI: Magnetic Resonance Imaging; DVT: Deep Vein Thrombosis; VKA: Vitamin K antagonist; INR: international normalized ratio.

Declarations

Ethics approval and consent to participate: not applicable.

Consent for publication: Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available.

Availability of data and materials: not applicable.

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Authors' contributions: RH: collection of data, drafting and critical review of the literature and critical revision. PR: critical revision. NZ: critical revision. YM: critical revision. All authors read and approved the final manuscript.

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Figures

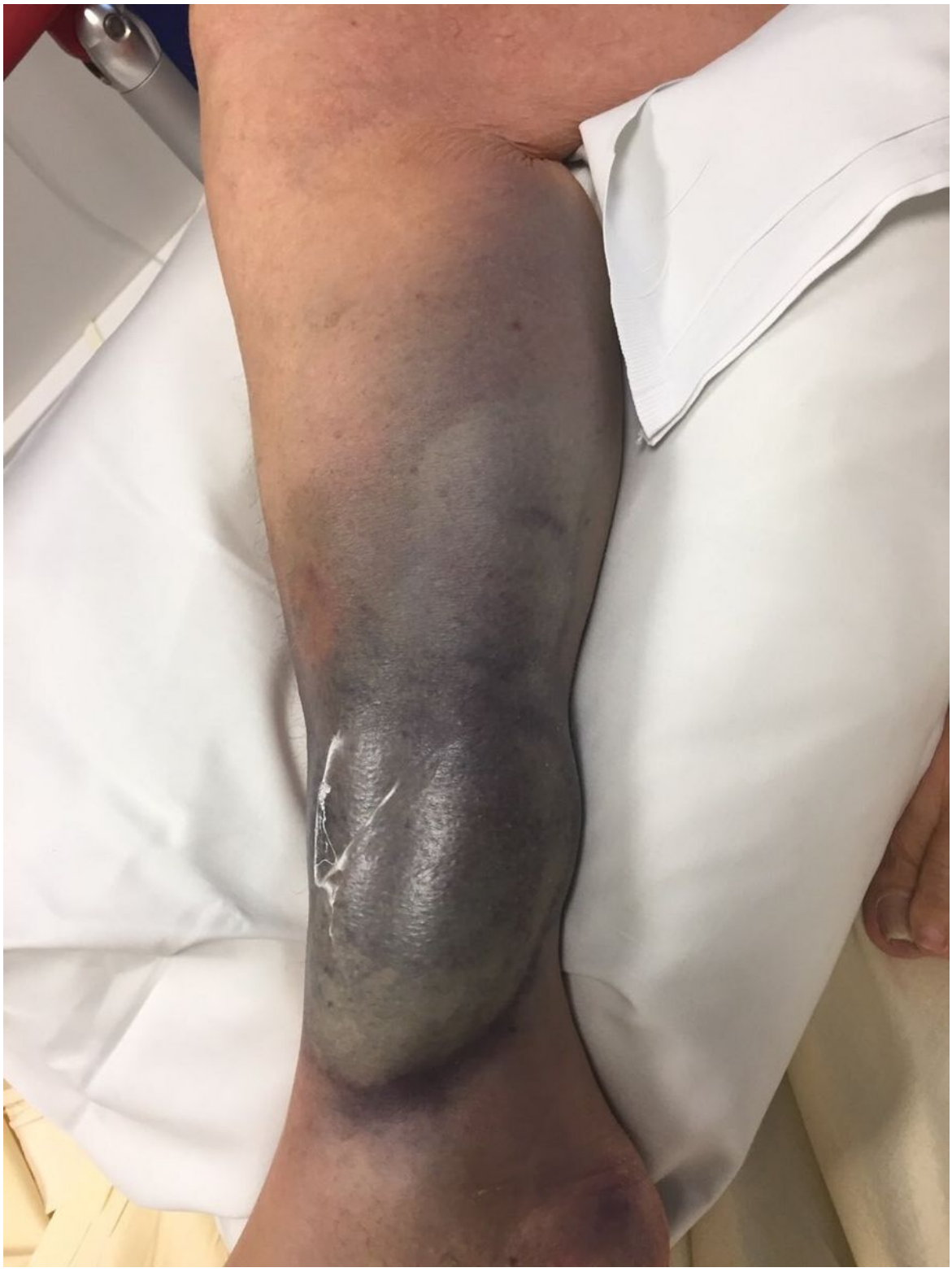

Figure 1

Clinical presentation of the patient on admission, demonstrating a right-leg deep dissecting hematoma.

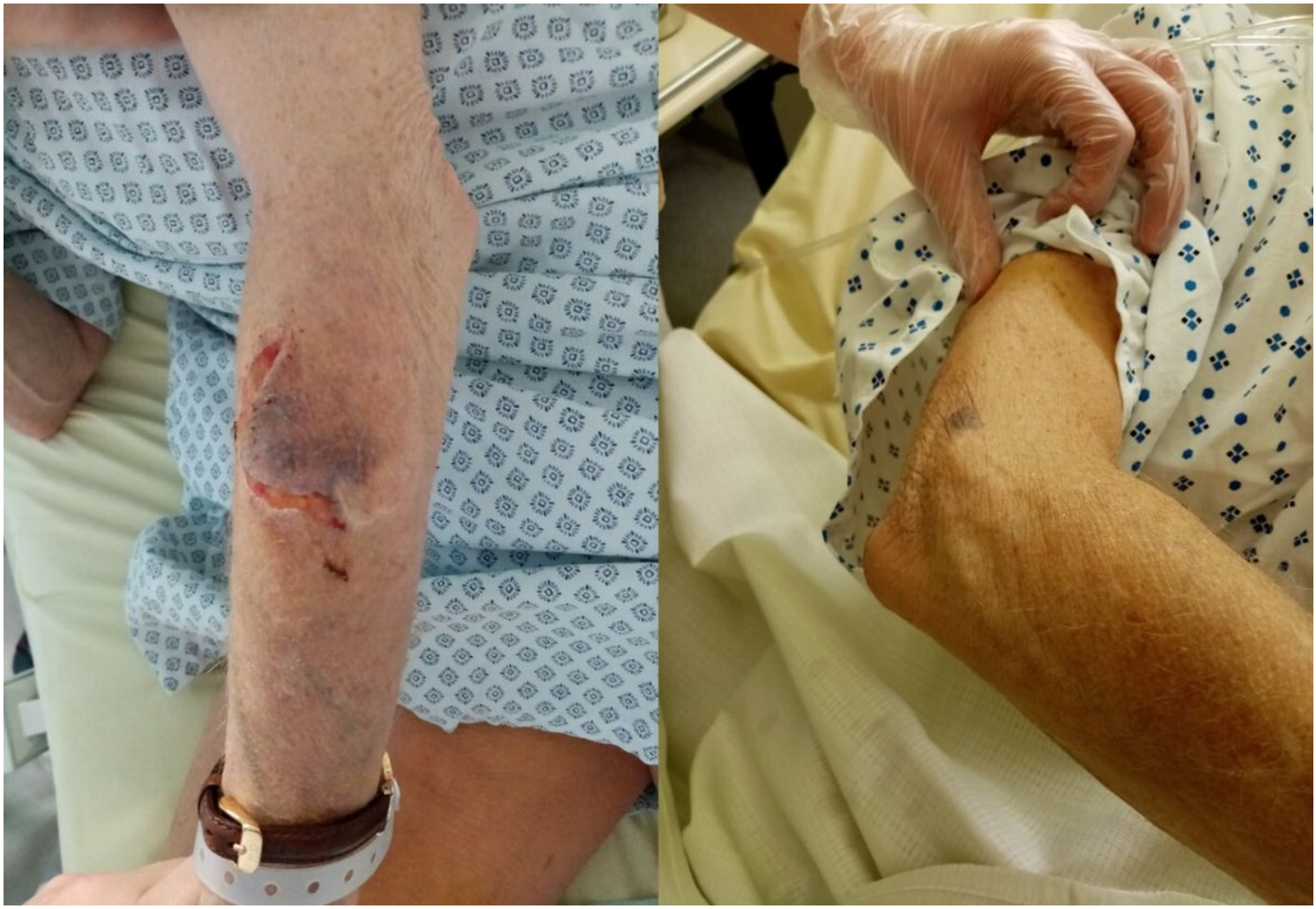

Figure 2

Lacerations and superficial hematomas in the forearms as signs of dermatoporosis.

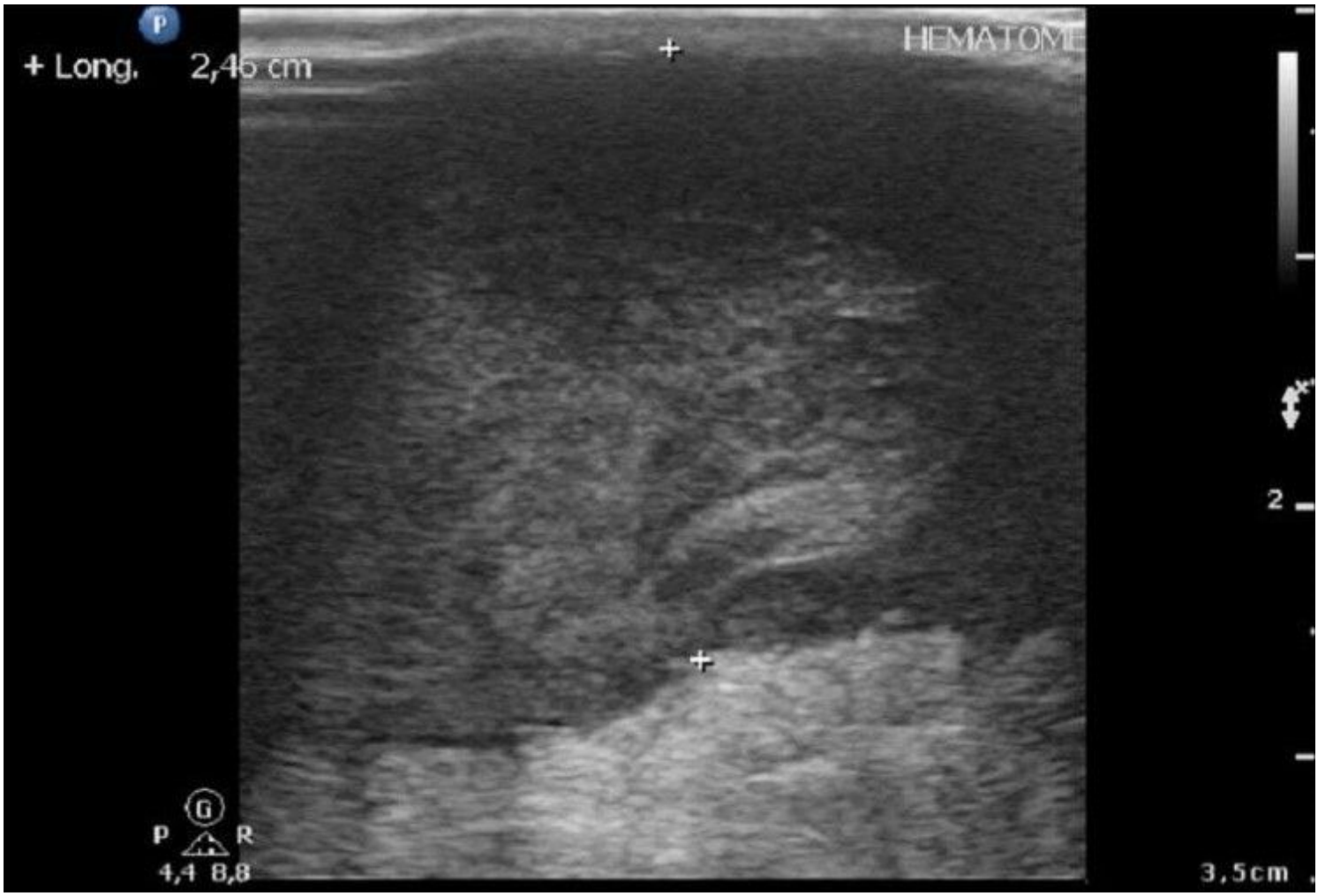

Figure 3

Transversal view of the deep dissecting hematoma during soft tissue sonographic study of the right leg.

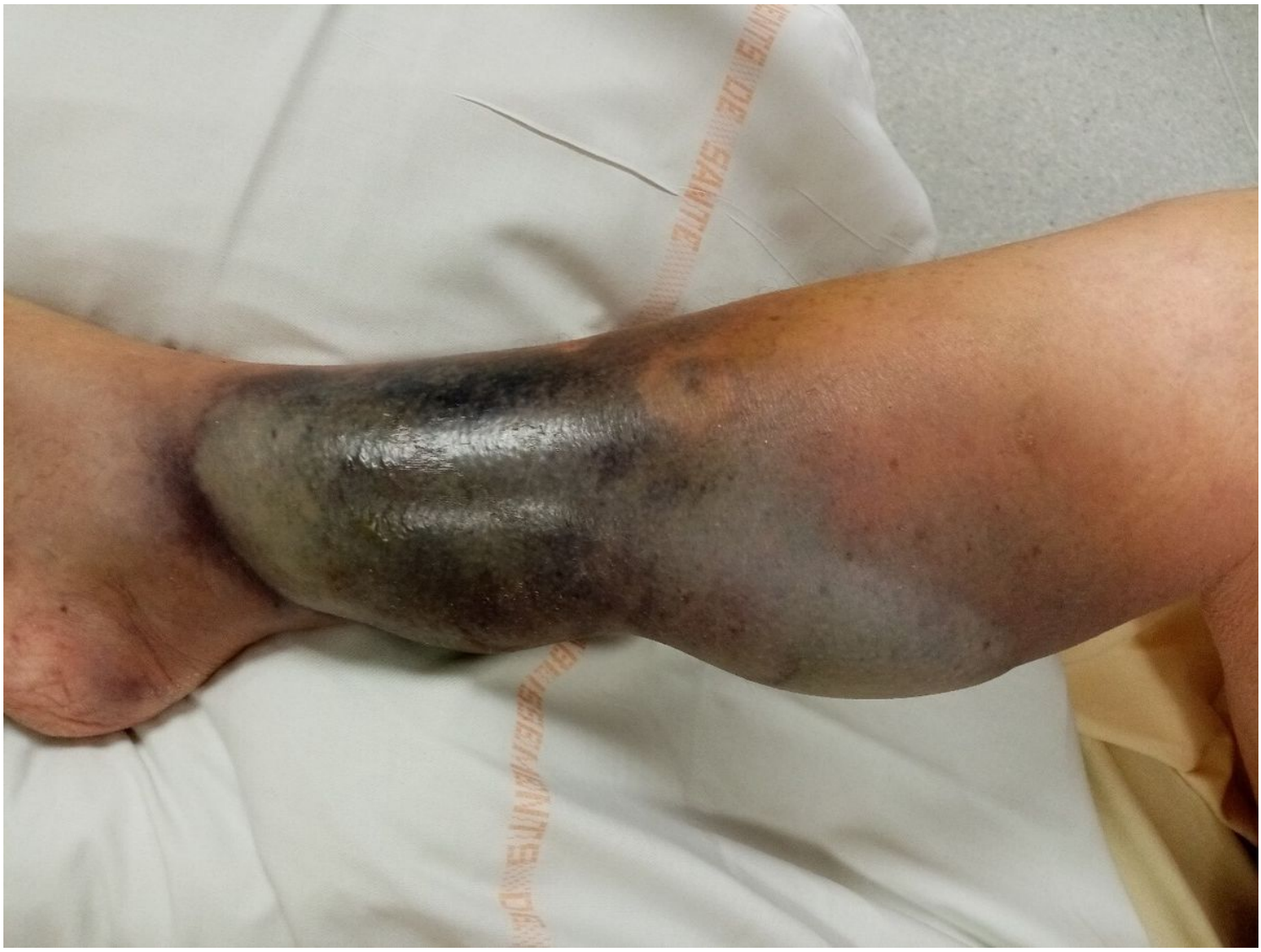

Figure 4

Right-leg deep dissecting hematoma four hours after admision at the Emergency Department, extending to the upper part of the calf.

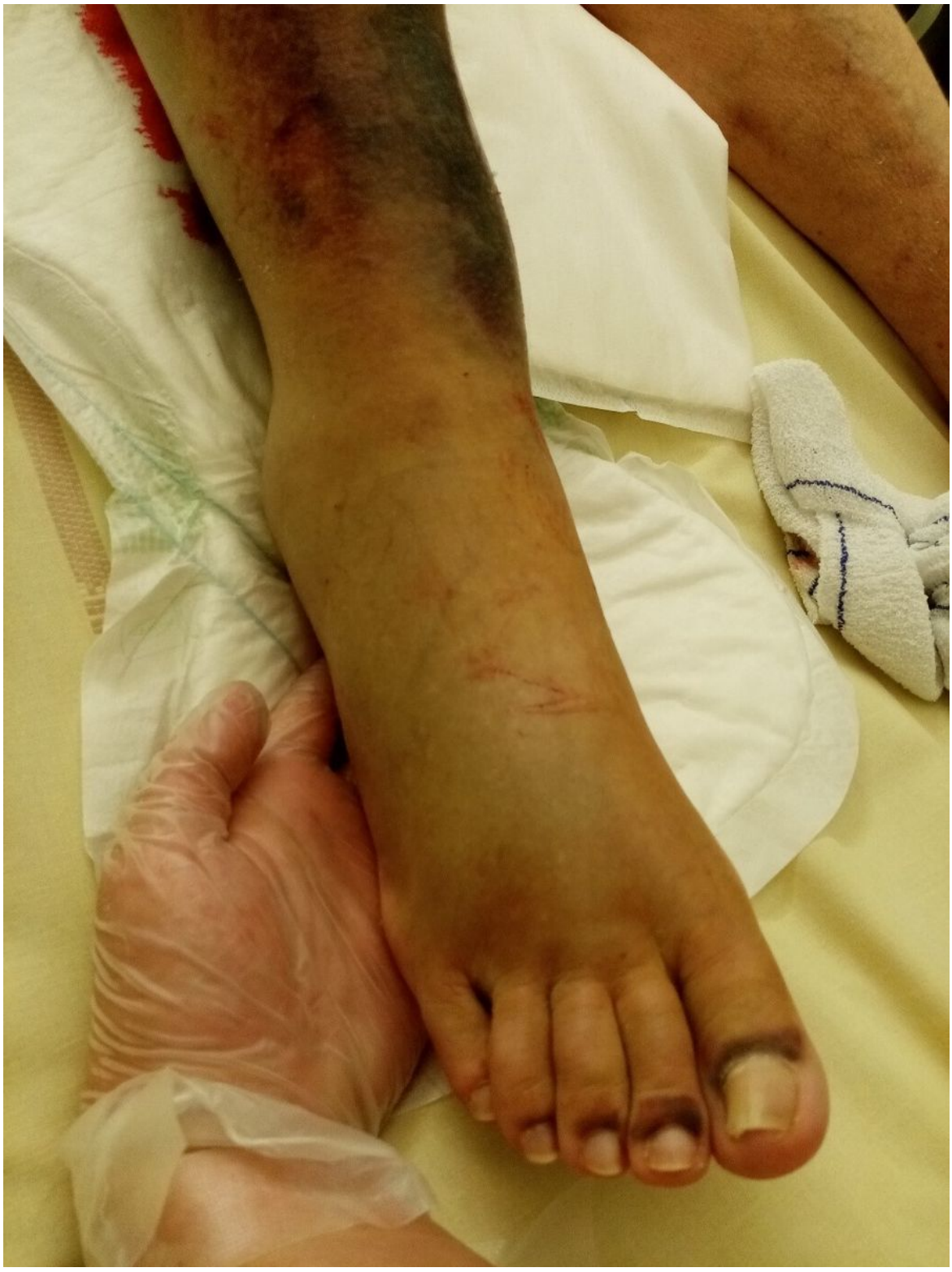

Figure 5

Right-leg deep dissecting hematoma spread to dorsum of the foot.

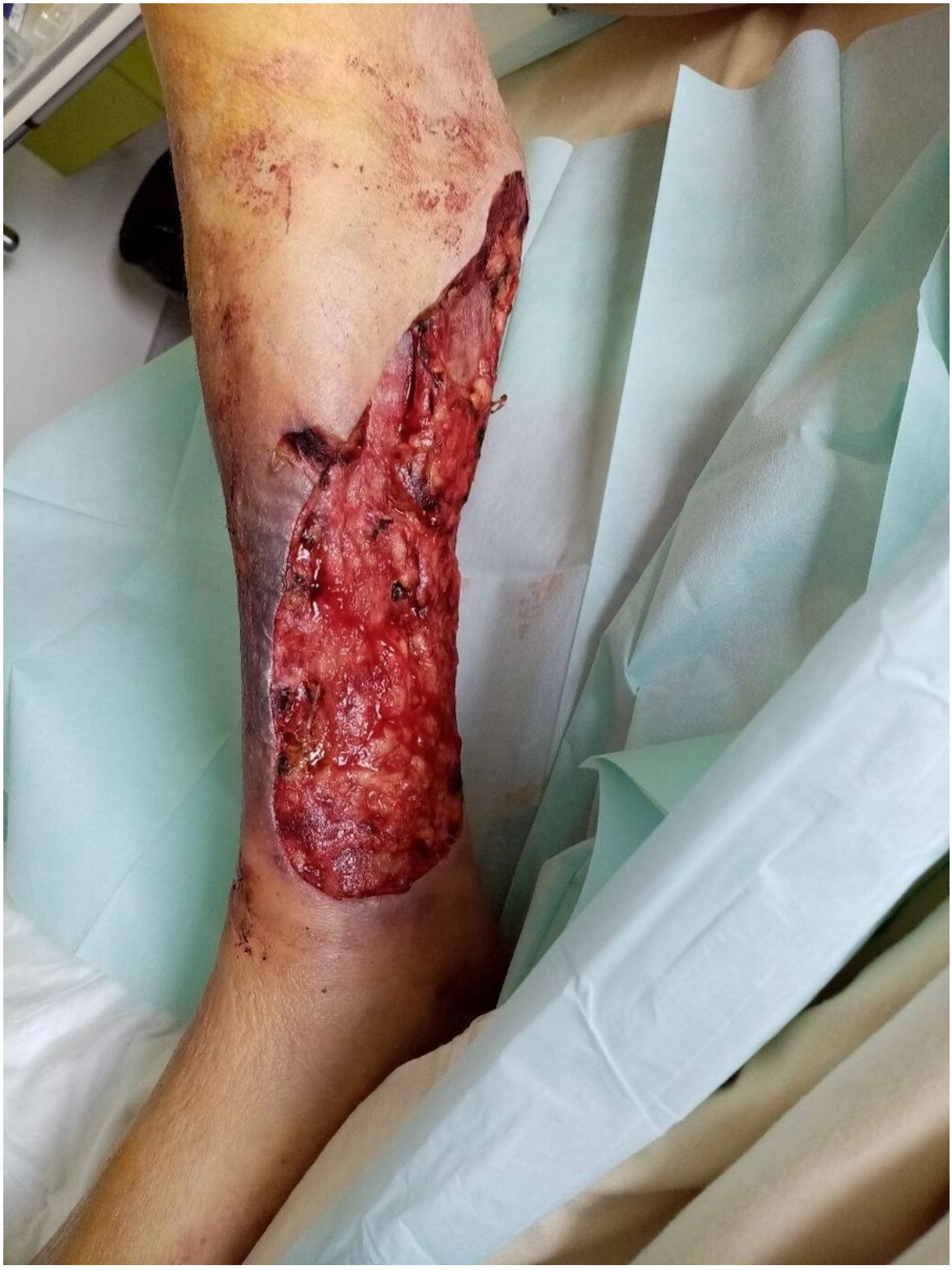

Figure 6

Right-leg wound one day after surgery.