

Rapidly spreading deep dissecting hematoma occurring 1 month after a minor trauma: A case report

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Abstract

A deep dissecting hematoma is the most serious complication of dermatoporosis, consisting of a rapidly expanding blood collection that splits the hypodermis from the muscle fascia. A several-week time lapse between a minor trauma-induced superficial hematoma and its sudden evolution into a rapidly spreading deep dissecting hematoma is unusual. We report the case of a 70-year-old woman with long-term oral anticoagulation and dermatoporosis who suddenly developed a rapidly spreading right-leg deep dissecting hematoma 1 month after minor trauma, for which a surgical debridement and drainage were performed. Only local care and absorbent dressings were used to manage the post-operative wound, and within 4 months, the wound had healed. In this report, we emphasize the importance of preventing deep dissecting hematoma in patients who are at risk as well as the need to weigh the benefits and risks of anticoagulants when dermatoporosis cutaneous signs are present. A limb-threatening deep dissecting hematoma may develop suddenly, even weeks after a minor impact. In order to prevent skin necrosis from occurring, caregivers, patients, and carers must be able to identify this condition early on.

Keywords

Emergency medicine, dermatology, surgery, deep dissecting hematoma, anti-vitamin K

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Introduction

Dermatoporosis is a chronic condition with a female predisposition where the skin becomes frail and tears easily,¹ with a prevalence in France of 32% among hospitalized patients over 60.² 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.¹ Deep dissecting hematoma (DDH) represents the most serious stage of dermatoporosis. It refers to the rapid pooling of blood under pressure, dissecting the virtual space between the muscle fascia and the hypodermis. The horizontal extension of the hematoma results in a large detachment of the overlying skin, which can then become necrotic. Prompt surgical management is needed to prevent skin necrosis.^{3,4}

The diagnosis of DDH is clinical. It is classically a hematoma extending rapidly and arising forthwith after trauma in an elderly woman affected by dermatoporosis. Long periods of time between the initial trauma and the appearance of a hematoma, a common main feature of other types of hematomas,⁵

have rarely been reported in DDH.⁶ We present a case of limb-threatening right-leg pretibial DDH in a 70-year-old woman with secondary dermatoporosis that happened 1 month after minor trauma, during an overdose of fluidione, a vitamin K antagonist (VKA).

Case

A 70-year-old woman was referred to our emergency department with a sizable, painful pretibial hematoma located in the back of the right leg, ending just above the ankle (Figure 1).

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A month prior, the patient banged her leg into a chair, resulting in a limited superficial hematoma. On the day of the admission, in the morning, the hematoma suddenly expanded, reaching the ankle and the calf, causing pain, and preventing weight bearing.

She was treated with fluindione, 20 mg per day for 3 years for paroxysmal atrial fibrillation. The International Normalized Ratio (INR) measurements were always between 2 and 3 in the previous weeks. Fluindione was last taken the day before. In addition to long-term anticoagulant therapy, the patient's medical history included sarcoidosis treated for more than 10 years with prednisone 8 mg/day, chronic respiratory insufficiency under long-term oxygen inhalation therapy, osteoporosis, and hypothyroidism. Bosentan, furosemide, levothyroxine sodium, a fixed dose combination of fluticasone propionate and salmeterol for inhalation, potassium chloride, and estradiol patches were used on a daily basis. Smoking had been stopped for several years.

The body mass index was 20.6 kg/m² (weight: 61 kg); the blood pressure was 147/96 mmHg; the heart rate was 101 beats per minute; the temperature was 36.5°C; and the pain was severe (a numerating rating scale of 8–9).⁷ There was no argument for erysipelas. Neither weakness nor paraesthesia was noted. Due to the hematoma, palpation of the pedis and posterior tibial pulses was limited. On skin examination, senile purpura, atrophy, large lacerations, and superficial hematomas were found in the forearms, indicating severe dermatoporosis (Figure 2).

At the time of admission, the white blood cells (WBC) were 12.10/L (neutrophils: 10.30/L); the platelets were

366/L; the hemoglobin was 13.40 g/dL (hematocrit: 42.40%); the C-reactive protein was 7.50 mg/L; the creatinine was normal; and the INR was 3.15. X-ray showed no underlying bone fracture. A Doppler US examination ruled out a deep vein thrombosis (DVT) and revealed a



Figure 1. Right-leg deep dissection hematoma at admission.



Figure 2. Lacerations and superficial hematomas in the forearms as signs of dermatoporosis.

well-defined hypo/heterogeneous mass between the skin and muscle fascia (Figure 3).

Initial medical management involved fluid intake discontinuation, administration of 5 mg of vitamin K and analgesics. Four hours after admission, while the pain was not alleviated by usual analgesics, the hematoma had spread to the upper part of the calf (Figure 4(a)), went on, reached the dorsum of the foot (Figure 4(b)) and eventually self-opened with pain relief. The day after, surgical treatment consisted of debridement and drainage in aseptic conditions under spinal block. A bulky hematoma (15 × 20 cm with a 5–6 cm thickness) localized between the muscle fascia and hypodermis was noted, confirming the diagnosis of DDH. Antibiotic prophylaxis with amoxicillin/clavulanic acid for 7 days was

started. Intraoperative samples contained a large number of red blood cells (RBC) without WBC or germs on direct examination, and were sterile after culture.

The day following surgery, the patient had dyspnea and hemoglobin was 8.4 g/dL (hematocrit: 27.1%), so two units of packed RBC were transfused. The post-operative wound was extensive, deep, exudative and painful (Figure 5(a)). After cleansing with saline, a calcium alginate dressing was applied and the leg was bandaged. From the second post-operative day, as the pain intensity decreased, daily wound care included saline lavage, paraffin Tulle Gras dressing, a secondary absorbent dressing, compresses, and leg bandaging. The patient was discharged on Day 12. She was prescribed physiotherapy, preventive anticoagulation (enoxaparin sodium 40 mg daily), and wound care (cleaning, detersion, application of a primary hydrofiber foam, leg bandaging) to be performed twice a week by nurses. A follow-up medical consultation was planned every 2 weeks. At the first follow-up consultation, the wound was fibrinous and highly exudative, so the hydrofibers were switched to calcium alginate dressings, and nursing care became daily. Budding was quickly achieved, and, after 1 month, calcium alginates were switched to tulle gras dressings. Complete re-epidermisation was achieved within 4 months (Figure 5(b)) without any infection or bleeding.



Figure 3. Transversal view of the deep dissecting hematoma during soft tissue sonographic study of the right leg.

Discussion

DDH usually appears immediately after an injury, which may be minor. A single-center retrospective study that included 2092 patients found an incidence rate of 53% (95% CI=44–64) per year, and among 112 hematomas, 26 were DDH.⁸ A skin biopsy is not always required, but it ascertains

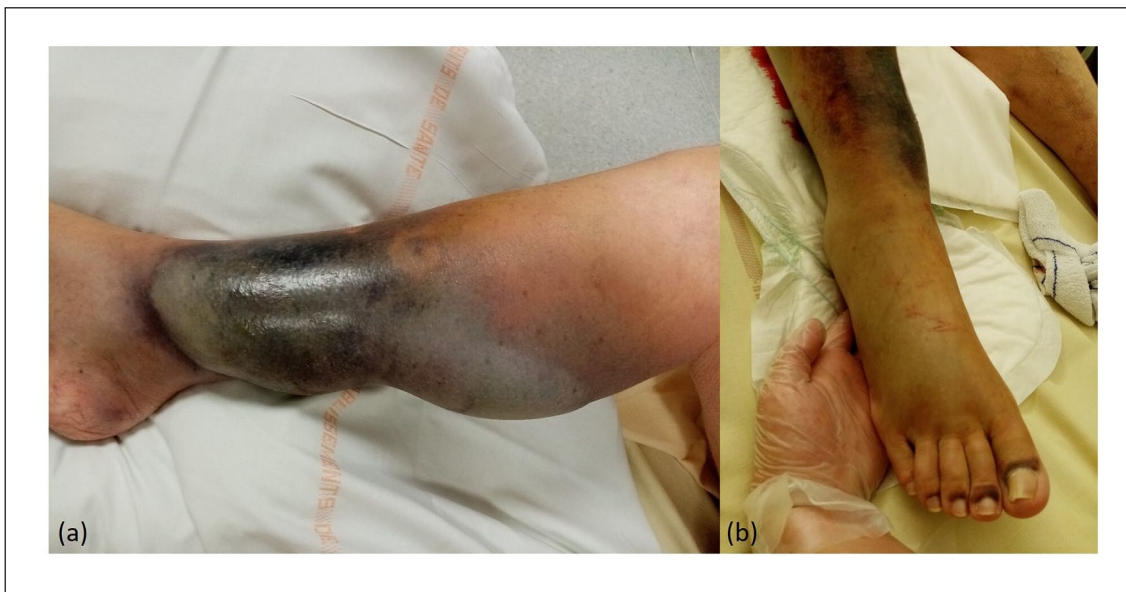


Figure 4. Right-leg deep dissecting hematoma 4 h after admission at the Emergency Department, extending to the upper part of the calf (a) and dorsum of the foot (b).



Figure 5. Significant loss of substance after surgical debridement and drainage of the right-leg deep dissecting hematoma (a). Right-leg wound 4 months after surgery (b).

the diagnosis by confirming the presence of degenerative changes in the dermal and subcutaneous vessels.^{3,4} The main differential diagnoses are erysipelas, DVT, non-dissecting hematoma, and Morel–Lavallee syndrome.^{4,6} According to Kaya and Saurat,¹ 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. Depending on the extension, age, and whether or not the hematoma is closed, Fennira et al.⁴ described four types of DDH (early closed type, advanced closed, advanced type with necrosis, and open type) and highlighted rapid increases in the volume and extent of the hematoma as signs of severity. Concerning our patient, a negligible trauma caused a superficial hematoma which did not resorb (favored by anticoagulation therapy) and decompensated spontaneously into DDH (due to dermis fragility) after a 1-month quiescent phase. An early closed type DDH eventually developed into an advanced closed type and then into an open type in a matter of hours.

The treatment of DDH comprises an invasive procedure (deep incision or surgical debridement) followed by conservative treatments.^{3,4,9} Negative wound pressure therapy (NWPT) is recommended by some authors when skin grafting is being considered,⁹ and the majority of articles on DDH mention the use of NWPT,^{3,10–12} despite the low level of evidence supporting its value.^{13,14} Post-operative wound management was limited to local care and dressings in only two reported DDH cases.^{6,15} In our case, rapid healing deemed skin grafting unwarranted, so

NWPT was not proposed. When our patient's wound was exudative, we opted for a calcium alginate dressing to remove excess exudate, prevent maceration, and promote granulation.

Management of severe dermatoporosis involves prevention, particularly against trauma in the population identified as being at risk of DDH, and entails the risk of falls (by adapting furniture in the home, layout of the carpets), visual disorders' correction, and lower limbs' protection.^{4,9,10,11,16} The signs of dermatoporosis and its complications, such as DDH, must also be early recognized by caregivers, carer, and patients affected by dermatoporosis, because the earlier the hematoma is removed, the less likely it is that the patient will sustain severe skin damage.^{4,9}

Although it has been reported under direct oral anticoagulants, the association of skin frailty and VKA-for which cutaneous complications are well documented¹⁶ or antiplatelet drugs⁶ remains the classical situation where DDH has been observed.^{3,4,8} Hence, our report raises the issue of bleeding risk assessment in anticoagulated patients who may have dermatoporosis. A detailed and frequent skin examination should look for dermatoporosis clinical signs. Like polypharmacy, a high risk of falling or social isolation, skin fragility caused by aging (or, as in our case, by prolonged exposure to corticosteroids) should make one reconsider the risk–benefit ratio of an anticoagulant treatment. When an anticoagulant treatment is necessary, the dosage must be adapted and carefully monitored, especially in elderly patients who are at risk of renal failure and drug interactions.^{4,16}

Conclusion

DDH is the most serious dermatoporosis complication and is a medical surgical emergency. A major issue is preventing DDH by creating a secure environment, dressing appropriately, and education. Moreover, a skin examination for signs of dermatoporosis should be performed when anticoagulant therapy is being considered. In this case, we highlight that a long time may pass between a minor trauma and a DDH's rapid growth, especially if the patient is taking anticoagulants. While this presents a diagnostic challenge for the treating physician, to the best of our knowledge, there have not been many DDH-specific studies that address this time-related issue. Because of the risk of rapid extension and subsequent skin necrosis, caregivers should be able to recognize a DDH and initiate appropriate management in order to protect patients and enhance skin outcomes.

Author contributions

RH: collection of data, drafting and critical review of the literature and critical revision. PR: critical revision. NZ: critical review of the literature and critical revision. YM: collection of data, critical revision. All authors read and approved the final manuscript. Ms. Suzanne Rankin is to be thanked for proofreading this document and making the necessary corrections. The authors also acknowledge Research Square that the article has been accepted for publication as preprint.

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Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

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