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CASE REPORTS

THERAPY-RELATED SPONTANEOUS PECTORAL MUSCLE HEMATOMA: A CASE REPORT AND REVIEW OF THE LITERATURE

To the Editor: Trauma to the chest wall or invasive medical procedures of the thorax are generally the cause pectoral muscle hematomas (PMHs), which is a rare condition.1,2 Occasionally, there may be no history of trauma or invasive medical procedure, with hematoma occurring spontaneously. Blood thinners can be the underlying etiological agent.2–5 This letter presents an unusual case characterized by spontaneous pectoral muscle bleeding in a woman undergoing warfarin therapy and reviews other spontaneous (nontraumatic) cases of PMH related to blood thinner medication.

A 79-year-old woman was hospitalized with the diagnosis of pneumonia and acute kidney failure. Piperacillin with tazobactam and furosemide infusion was initiated. Her medical history included Parkinson’s disease, Alzheimer’s disease, heart failure, and mitral valve replacement. She was taking memantine, warfarin, furosemide, lansoprazole, and a combination of levodopa, carbidopa, and entacapone. The day after she was hospitalized, a swelling approximately 5 cm in diameter developed in her right breast. Laboratory results were hemoglobin 8.6 g/dL (normal 12–16 g/dL), white blood cell count 9,510/µL (normal 3,500–10,500/µL), platelets 198,000/µL (normal 130,000–400,000/µL), international normalized ratio (INR) 7.74 (normal 0.8–1.2), alanine transaminase 28 U/L (normal 0–55 U/L), aspartate transaminase 51 U/L (normal 5–34 U/L), creatinine 1.67 mg/dL (normal 0.57–1.11 mg/dL). Ultrasonographic evaluation of the chest wall was consistent with PMH. She had no history of fall or trauma. The swelling grew over the subsequent few hours (Figure 1A). Computed tomography (CT) of the thorax showed a 14.7- by 14.2- by 8.6-cm PMH that was not in contact with the mediastinum or lung parenchyma (Figure 1B). The hematoma increased in diameter during follow-up. Tachycardia and hypotension developed consistent with the hemorrhagic shock. Four units of red blood cell suspension and three units of fresh frozen plasma were administered, leading to a decrease in INR to 1.3. Her hemodynamic condition improved, and there was no significant decline in repeated hemoglobin measurements. Right pectoral region pain was the only symptom. No symptoms or signs of new bleeding were detected during the rest of her hospitalization. Enoxaparin was recommended instead of warfarin for maintenance treatment.

Spontaneous PMH due to blood thinners is rare; four cases have been documented in the English-language literature,2–5 although more cases have been reported in other languages. Clinically insignificant bleeding may not have been presented as case reports,6 so the actual incidence may be higher. Table 1 presents a summary of the features of therapy-related spontaneous PMH cases. Various blood thinner medications may be the reason. The current case is

Figure 1. (A) Image of swelling of right anterior chest region caused by the pectoral hematoma. (B) Thoracic tomography image consistent with pectoral hematoma.
the second reported instance of spontaneous PMH with warfarin therapy and the first one with warfarin monotherapy.

All of the individuals reported were older adults. It has been suggested that old age is a risk factor for the bleeding side effects of blood thinner medications.7,8 Old age may be a risk factor for spontaneous PMH, possible because indications for and use of blood thinners are much more frequent in older adults. Interactions between drugs and changes in drug metabolism due to polypharmacy or liver or kidney failure more commonly seen in this population are other possible reasons.7–9 Two of the reported cases and the woman described herein had varying levels of kidney disease. Another reason might be the loss of elasticity in blood vessels, low muscle mass, and lack of muscle elasticity in older adults. Because of these changes, some movements that are not traumatic for younger individuals may be traumatic and hemorrhagic in the highly vascularized pectoral muscles of older adults.9,10

Acute development of swelling in the anterolateral chest wall may be indicative of PMH. Ultrasonography and CT are commonly used methods of definitive diagnosis. Hematoma volumes range from small without any signs or symptoms to as large as 25 by 15 by 10 cm and may present with hemorrhagic shock. In addition to the current case, one other case presented with hemorrhagic shock; both cases were associated with warfarin use and high INR (7.8 and 7.74).2–5 Classical management of spontaneous PMH is supportive therapy with fluid support and blood replacement. Surgical drainage has also been reported for treatment to reduce progressive pain and control hemorrhaging.4 PMH has a favorable prognosis, and there is no report of death due to hemorrhage.

Spontaneous PMH should be considered in the presence of clinical suspicion in older adults taking blood thinners. Bleeding may be severe, so follow-up and replacement of fluids and blood is critical and can be life-saving.

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A 68-year-old Chinese woman was admitted to the hospital for a 1-week complaint of painful and itchy lower limb rash. She had been diagnosed with Alzheimer’s disease 2 months before admission complicated by depression, making her behavior was severely distressing to herself and her family, she was started on quetiapine, an atypical antipsychotic. At admission, she was taking oral medications and her family, she was started on quetiapine, an atypical antipsychotic quetiapine and its clinical implications. At admission, she was taking an oral medication regimen of donepezil 5 mg/d and quetiapine 12.5 mg in the morning and 25 mg at night, started 6 weeks before the onset of rash. The dose of quetiapine had been gradually increased in the preceding weeks. She also had a nightly dose of fluvoxamine 100 mg that had been initiated 4 months before.

At admission, she was afebrile, and her vital signs were normal. An erythematous, raised, nonblanching rash was distributed across both shins, consistent with palpable purpura. Laboratory tests showed a borderline low leukocyte count (3.5 × 10^9/L). Eosinophil count, serum creatinine, transaminases, and C-reactive protein levels were normal.

History and physical examination was not suggestive of connective tissue disease, nor was she taking any new drugs. Tests for complement C3, C4, antinuclear antibodies, antidual-stranded deoxyribonucleic acid, anti-Ro, anti-La, anti-ribonucleoprotein, anti-Sm, anti-Jo1, anti-SCL 70, and antineutrophil cytoplasmic antibodies were negative.

Adjustments were made to her medications because worsening BPSDs were observed. Donepezil was withheld, and quetiapine dose was gradually increased to 50 mg twice a day. As the dose of quetiapine was increased, the purpuric rash appeared to worsen. Subsequent punch biopsy of the skin rash showed superficial perivascular lymphocytic infiltrate and focal red cell extravasation around upper dermal blood vessels. Direct immunofluorescence revealed granular deposits of immunoglobulin M and C3 within the walls of the upper dermal vessels, consistent with vasculitis.

The etiology of cutaneous vasculitis was postulated to be drug related, possibly due to quetiapine. Quetiapine was withheld, and the patient was given haloperidol and, later, risperidone. She did not receive corticosteroids. Her rash gradually improved and resolved by the seventh day after stopping quetiapine.