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CLINICAL SPOTLIGHT

Warfarin Anticoagulation and Spontaneous Pectoral Haematomas

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Warfarin is the oldest and most commonly used anticoagulant in the outpatient setting. Major bleeding events remain as the most life threatening complication of this medication. Bleeding into enclosed structures and body cavities can be fatal in acute scenarios or cause continuous exsanguination if left unnoticed. Pectoral haematomas are an unusual presentation of bleeding diathesis, and are also seldom reported in the literature. We present three cases of patients with development of spontaneous pectoral haematoma during therapy with warfarin alone or with heparin bridging in the treatment of atrial fibrillation and thromboembolism.

Keywords Pectoral haematoma • Warfarin • Heparin bridge • Anticoagulation

Introduction

Since the discovery of the agent bishydroxycoumarin from spoiled sweet clover silage in haemorrhagic cattle disease and its subsequent uses as rodenticide, the bleeding property of warfarin has transformed it into an anticoagulant now used by more than 1.5 million people in the US today [1]. Warfarin is the most commonly used maintenance anticoagulant in the outpatient setting; its indications of use include venous thromboembolism, mechanical valves and atrial fibrillation. Close monitoring of the international normalised ratio (INR) has been the standard of care for patients on warfarin therapy. Yet, bleeding remains as one of the most significant and serious complications of warfarin use. Short duration of bleeding into the enclosed structures of the head and pericardium is fatal, but severe haemorrhage can go unnoticed for longer periods in various body cavities and muscular compartments [2]. Haematoma formations have been reported in various muscle groups, including the quadriceps, psoas and pectoral muscles. Pectoral haematomas have been infrequently reported in the literature, either due to lack of recognition or reporting. Here we present three case reports of spontaneous pectoral haematomas noted in patients on warfarin therapy alone and also on heparin bridge to warfarin therapy.

Case Summaries

Case 1

A 66-year-old male with a history of coronary artery disease, systolic heart failure (ejection fraction 23%, with implanted ICD), hypertension, atrial fibrillation (on warfarin), diabetes Type 2, stage 3 chronic kidney disease, ischaemic stroke with left hemiparesis and recent left above knee amputation two months prior for superinfected left ulcer, presented to the emergency department (ED) from home with weakness, lethargy and hypotension on a home blood pressure monitoring device. His family also noted left-sided ecchymosis on his chest that morning. The patient was found to have systolic blood pressure of 60 mmHg initially in the ED. He was aggressively volume resuscitated with normal saline, but he became unconscious, and was noted on monitor to have pulseless ventricular tachycardia. His ICD fired twice without effect, and he was then externally cardioverted twice, intubated, started on intravenous pressor medications and

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lidocaine drip. His initial laboratory results revealed a haemoglobin of 7.8 g/dL, significantly less from 11.4 g/dL on outpatient laboratory work several days prior. His INR was also noted to be 5.25, and serum creatinine 2.45 mg/dL. He received two units of packed red blood cells (PRBCs), two units of fresh frozen plasma (FFP), and vitamin K 5 mg. Initial chest radiograph showed only mild costophrenic angle blunting. The patient was then transferred to the medical intensive care unit (MICU) for hypovolaemic shock secondary to haemorrhage. On the second day, the left chest wall ecchymosis was noted to have increased in size. He underwent chest computed tomography (CT), which found a hyperdense collection on the left chest wall, measuring 11x7x12 cm, posterior to the ICD device and compatible with a haematoma. He subsequently required an additional two units of PRBCs transfusion and vascular surgery and interventional radiology were consulted. The recommendations were for continual surveillance and conservative management. His serial haemoglobin and haematocrit had stabilised without further decrease and hence was not intervened. The patient was extubated five days later and transferred out of the MICU. Warfarin was restarted for thromboembolic stroke prophylaxis, as his calculated $CHADS_2$ score was 5, but his new INR goal was targeted at a narrower range around 2. Subsequent history obtained from the family revealed that the patient was actively engaging in home physical therapy for recent left knee amputation. It was further surmised that overuse of the pectoral muscles may have been a factor especially with use of crutches after lower limb amputation in this case.

Case 2

An 80-year-old female with a history of coronary artery disease, atrial fibrillation (on warfarin), diabetes Type 2 and ischaemic stroke with left hemiparalysis who is wheelchair bound, presented to the ED from home with bright red blood per rectum for three days. She was found to be in atrial fibrillation with rapid ventricular response with a heart rate of 140-150 beats per minute and systolic blood pressure of 80 mmHg on admission, and was given normal saline intravenous fluid bolus and intravenous diltiazem injections twice without slowing down of her rate. She was then started on diltiazem drip at 5 milligrams per hour with adequate control of her heart-rate. Her laboratory work on admission included a haemoglobin/haematocrit of 13.2 g/dL, 37.6% respectively, INR of 1.08, and sodium of 124 mEq/L. In total, she received three litres in fluid boluses and her systolic blood pressure had stabilised in the 90-100 mmHg range, with normalisation of serum sodium on the second day of hospitalisation. She was weaned off diltiazem drip and started on diltiazem orally for control of her heart-rate. Her healthcare proxy (HCP) declined colonoscopic workup for the initial complaints of bright red blood per rectum, as he felt that the patient was too high risk to undergo the procedure. She was then restarted on anticoagulation for her atrial fibrillation, using heparin infusion to bridge with warfarin as the INR was not therapeutic. On the third day of bridging therapy, she had an acute drop in her haemoglobin from 10.1 g/dL to 6.4 g/dL, and also noted her left chest wall to be significantly more swollen. The INR at that time was 2.19, and heparin infusion had already been discontinued for one day, but significantly for that the aPTT were supratherapeutic for two readings, 191.5 sec and 128 sec, four days prior to the event. She was transferred to the MICU for increasing lethargy and for observation. She received five units of PRBCs and four units of FFP with adequate response and no further drop in her blood count. Emergent chest CT showed a large left retropectoral haematoma measuring 14x7.9x18 cm in size with surrounding haemorrhage into the left chest wall and breast. Vascular surgery was consulted but the HCP declined to pursue further intervention, and the patient was managed conservatively with serial blood monitoring and compressive dressing. She was stable and transferred out of the MICU after three days of observation. It is notable that family revealed that the patient had been to the ED six days prior to this admission after having accidentally fallen out of her wheelchair. She had mild abrasion over her forehead and was sent home after negative CT head imaging during that visit. The possibility of wheelchair transfers in this case may have lead to pectoral muscle overuse and contributed to the retropectoral haematoma.

Case 3

A 71-year-old male presented with a history of coronary artery disease status post bypass grafts, mitral regurgitation, antiphospholipid syndrome with deep vein thrombosis (DVT) and pulmonary embolism (status post inferior venous cava filter), superior vena cava syndrome (on warfarin), stage IV chronic kidney disease, recent clostridium difficile colitis, and dysphagia (status post percutaneous endoscopic gastrostomy {PEG} placement). He presented from his rehabilitation facility with complaints of nausea, vomiting, diarrhoea, abdominal pain and blockage of his PEG tube. He was admitted on the workup of diarrhoea and treatment for pneumonia. His hospital course was complicated by respiratory failure requiring intubation; he was also placed on temporary haemodialysis via left internal jugular Shiley catheter. The patient was bridged with heparin to warfarin for his history of DVT. Three days into this therapy, the aPTTs were supratherapeutic for three readings: >200 sec., 166 sec., and 160 sec.; however, his INR was still subtherapeutic at 1.52. On day five of heparin bridge to warfarin, the patient complained of right-sided chest pain, but there was no noticeable swelling on physical exam. His chest pain was not relieved by analgesics; therefore chest CT was performed that found an enlarged right pectoralis muscle with high attenuation compatible with haematoma formation. Serial imaging with ultrasound two days later showed a 3.8x1.5x1.2 cm intramuscular haematoma at right upper chest anteriorly. Serial ultrasound imaging of his right chest was performed weekly for two weeks but no intervention was pursued as the patient remained haemodynamically stable without any acute drop in his haematocrit/haemoglobin. It was later determined that the patient had his second physical therapy session for