MORNING REPORT

A Postoperative Pulmonary Embolism? Fat Chance

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A 55-year-old female with a past medical history of hypertension, post-partum deep venous thrombosis (DVT), obesity, and cardiac murmur since childhood is admitted for elective bilateral total knee arthroplasties. Her medications prior to surgery include losartan, hydrochlorothiazide, meloxicam, and nebivolol. She denies any tobacco or alcohol use. She discloses a strong family history of coronary artery disease but denies any familial thrombophilic propensity. Prior to this procedure, she felt well and had no complaints. She is taken to surgery and tolerates the procedure without any obvious complications. Approximately two hours after surgery, while still in the post-anesthesia recovery unit, she develops shortness of breath and substernal chest pain. The chest pain is exacerbated by deep inspiration, but there is no radiation. She reports no nausea, vomiting, or diaphoresis. Examination of the patient reveals blood pressure 110/50 mm/Hg, pulse 62 beats per minute, respiratory rate 16 breaths per minute, oxygen saturation 93% on 3L/min nasal cannula. She has clear breath sounds, normal S1/S2, a II/VI crescendo-decrescendo murmur, and bilateral knee drains in place with moderate amounts of sero-sanguinous fluid. Electrocardiogram (ECG) shows nonspecific T-wave abnormalities. Chest x-ray demonstrates no acute disease. Serial troponin and other laboratories are unrevealing.

Post-operative chest pain requires immediate evaluation. Given her history of DVT, obesity, and post-operative state, she is at elevated risk for pulmonary embolism (PE). Total knee arthroplasty patients are particularly high risk for DVT/PE (40% to 80% for calf DVT, 10% to 20% for proximal DVT, 4% to 10% for non-fatal PE, and 0.2% to 5% for fatal PE).[2] Our patient's sudden onset of symptoms and pleuritic nature of the pain are consistent with PE. However, postoperative PEs rarely develop sooner than a few days after surgery.

Myocardial infarction (MI) would be another perioperative concern, especially with her history of hypertension, obesity, and family history. The non-specific ECG and normal serial troponins render MI much less likely.

Her symptoms last approximately 45 minutes and then spontaneously resolve without any specific intervention. She remains asymptomatic overnight. The following day, however, the patient is noted to be in atrial fibrillation on telemetry monitoring, with concomitant chest palpitations and mild chest heaviness. ECG confirms atrial fibrillation, along with nonspecific diffuse T-wave inversions. Basic chemistries, as well as cardiac markers, are normal. To further elucidate her cause of atrial fibrillation, she undergoes an echocardiogram (ECHO). The ECHO reveals right-sided cardiac enlargement with moderate-to-severe pulmonary hypertension (pulmonary artery systolic pressure of ~72 mmHg).

Acute atrial fibrillation may be seen in up to 4.1% of patients following non-cardiac surgery, typically within the first three days.[5] Sometimes the inciting cause of new onset postoperative atrial fibrillation is acutely elevated pulmonary artery pressures, such as in the case of an acute PE. Our patient's ECHO findings of right-sided cardiac enlargement and pulmonary hypertension are concerning for the possibility of an acute PE.

Subsequently, a computerized tomography (CT) of the chest with contrast is obtained. No large thromboemboli are identified. However, there is bilateral and diffuse, prominent upper lobe, ground-glass mosaic attenuation consistent with small vessel miliary artery disease from micro fat emboli.

Fat emboli are extremely common following orthopedic procedures, especially after long-bone or pelvic fracture repair, though the vast majority is clinically undetectable. The high prevalence of fat emboli has been suggested not only by post-fracture autopsies but also by intraoperative echocardiography, revealing presumed emboli, a phenomenon that seems to lessen with bone vacuuming intraoperatively.[6] Despite the high prevalence of fat emboli, only 1% to 3% of patients will develop serious manifestations of the dreaded "fat emboli syndrome." This syndrome is characterized by the triad of pulmonary dysfunction, altered mental status, and petechial rash, usually occurring 24 to 72 hours after an inciting event, though sometimes demonstrating cardiopulmonary compromise almost immediately.[3] Hematologic/coagulopathy changes are also common. The symptoms are thought to occur from deposition of fat in the lungs, brain, and skin. The diagnosis is typically clinical and of exclusion; there exists a Gurd's criterion, which suggests major and minor criteria for diagnosis. Diagnosis of fat emboli syndrome requires one major criteria (hypoxemia, CNS depression, petechial rash, or pulmonary edema) and four minor criteria (tachycardia, pyrexia, retinal emboli, fat in urine or sputum, thrombocytopenia, or decreased hematocrit).

The patient is then placed on high flow oxygen (15L/min nasal cannula) for treatment of fat embolization and has resolution of her symptoms. She remains hemodynamically stable during her entire hospital course. She is discharged without any ongoing

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symptoms, with planned repeat echocardiogram and cardiology follow-up in two weeks.

Therapy remains supportive, including oxygen, positive end-expiratory pressure (PEEP), and proper fluid volume management. Steroids have been marginally studied and are seemingly helpful when given prophylactically in long-bone fracture,[1] suggesting that they may have an even more pervasive role in non-prophylactic and non-fracture scenarios. Nonetheless, there are almost no proponents that recommend their routine use as prophylaxis or treatment because most patients recover very well from this syndrome with supportive therapy alone, and the use of steroids poses an obvious infection risk. With conservative therapy alone, some studies report proposed mortality rates to be as high as 5% to 15%,[3] while some suggest rates closer to 1.2%.[1] Aside from orthopedic procedures and bone fractures, rarer causes of fat emboli have been cited and include burns, liposuction, chest compressions, severe soft tissue injuries, bone marrow harvesting, bone marrow transplant, diabetes mellitus, pancreatitis, osteomyelitis, corticosteroids, sickle cell anemia, alcoholic liver disease, and lipid infusion.[6] There are varying theories as to how fat emboli occur. One school of thought is that of embolization from the vasculature, which can enter arterial circulation, either via a patent foramen ovale (PFO) or directly through the microcirculation. Another theory is that C-reactive protein (CRP), in highly inflammatory states, induces calcium-dependent agglutination of lipids/chylomicrons already in the serum.[4] Finally, there is a theory of free fatty acid liberation, which when liberated through existing fat stores induces endothelial inflammation.[6] Whatever the case, this common phenomenon is often underrecognized.

Take Home Points

- Fat embolism is common in orthopedic patients—not only those with long-bone or pelvic fracture but also those with elective total joint arthroplasty due to intramedullary instrumentation.
- Fat embolism is a clinical diagnosis (Gurd's criterion) and one of exclusion, as there is no confirmatory laboratory test.
- In severe cases, fat embolism syndrome can develop. Diagnosis requires one major criteria, such as hypoxemia, CNS depression, petechial rash, and pulmonary edema, and four minor criteria, such as tachycardia, pyrexia, retinol emboli, fat in urine or sputum, thrombocytopenia, and decreased hematocrit.
- Treatment of fat embolism is supportive oxygen, positive airway ventilation, and possibly corticosteroids.

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