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Compartment syndrome as a complication of femoral deep vein thrombosis

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•ompartment syndrome occurs when muscle compartments bounded by unyielding fascial layer has increased pressure leading to neurovascular compromise and subsequent necrosis of the tissue. Long bone fractures are the most common cause. Other causes include crush injuries, severe thermal injuries, penetrating trauma, prolonged limb compression or intramuscular hemorrhage. Non-traumatic causes include ischemia reperfusion injury, thrombosis, bleeding disorder, nephrotic syndrome, certain animal envenomation and bites and injection of recreational drugs. Compartment syndrome following venous thrombosis is rare with only few reported cases of acute massive venous thrombosis following an inferior venacava filter placement. Here we report a similar case of compartment syndrome following common femoral vein thrombosis. A 47 year old female with history of multiple recurrent deep vein thrombosis presented with three day history of extensive left lower extremity swelling and pain. She was previously on Coumadin which had been held for the past ten days due to supratherapeutic INR and paraspinal hematoma. On exam, vital signs were within normal limits except systolic blood pressures in 150s. The left lower extremity appeared erythematous with visible swelling up to mid-thigh, non-pitting firmness on palpation, severe tenderness and pain on passive dorsiflexion of left foot. Dorsalis pedis and posterior tibialis pulses were not palpable but were present on Doppler. She complained of paresthesia and numbness of lateral left lower leg. Remainder of the exam was unremarkable. Ultrasound of lower extremity demonstrated a new non-occlusive thrombus extending from the common femoral vein through the distal femoral vein. These findings were confirmed by CT of abdomen and pelvis. Patient did not have any history of recent trauma, fracture, prolonged limb compression, recreational drug use or recent long travel. Diagnosis of compartment syndrome was made clinically and therefore was taken to the operating room for emergent fasciotomy. She was appropriately anti-coagulated. Following the wound healing, autologous skin grafting was done with good results. Increased compartment pressure following venous occlusion due to extensive thrombosis of the lower extremity is a known process. The most severe form is termed phlegmasia cerulea dolens. Signs include venous hypertension, ischemia and necrosis of the limbs, systemic circulatory failure along with paralysis, contractures and limb loss. Though it is a rare complication of deep vein thrombosis, it is important to consider this syndrome when DVT presents with classical findings of compartment syndrome. (Five Ps: Pain, pallor, pulselessness, paresthesia & paralysis). Emergent fasciotomy is the mainstay of treatment for compartment syndrome. Other therapeutic options like thrombolytic therapy and venous thrombectomy is reserved for massive thrombosis.

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Developmental proteins in Fanconi anemia

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F of developmental malformations and a probensity to malignancies. Aplastic anemia due to bone marrow failure is frequent during childhood and there is a high predisposition for myelodysplasia (MDS), acute myeloid leukemia (AML) and solid organ cancers such as head and neck tumors. Developmental malformations in FA patients include short stature, radial ray anomalies, scoliosis, aberrant skin pigmentation, abnormal development kidney and urinary tract development. The responsible genetic defect is in one of the sixteen FANC genes encoding the proteins in FA/BRCA pathway which resolve DNA interstrand crosslinks (ICLs). FANC/BRCA pathway functions in cells including stabilization of replication forks, maintenance of stem cells, suppressing error prone repair and tumorigenesis. FA cells treated with ICLs agents show an increase in G2/M phase of cell cycle. The current knowledge regarding ICLs repair defect in FA cannot explain the bone marrow failure and malformations of patients. Early hematopoietic dysfunction coincides with skeletal and kidney malformations in FA patients suggest the possibility of FA protein interaction with developmental proteins. Previously published FANCD2i and FANCAi knockdown study and immunoprecipitation study with FANCC gave evidence for these interactions and other roles of FANC proteins. Our recent study seeking gene expression signature of homeodomain transcription factors HOX and their cofactors TALE in Fanconi anemia mesenchymal stromal/stem cells, also designates possible new interactors.

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