his legs. His primary laboratory investigation showed anemia, monocytosis, eosinophilia and thrombocytopenia,. He also had elevated serum creatinine (4.4 mg/dl) and LDH(430U/L) values respectively. His prior creatinine level was normal. His urine analysis showed 28 erythrocytes/hpf. Calculated proteinuria was 750 mg/day. Urinary ultrasound examination was normal. Protein electrophoresis showed hyper gamma globulinemia 1.73 g/dl. Leucocytoclastic vasculitis was revealed by punch biopsy of skin. All serologic workup was normal except for low C3 levels (77 mg/dl). Bone marrow biopsy and renal fine needle biopsy was performed. Bone marrow biopsy showed peritrabecular and intertrabecular lymphoid nodules consisting of CD20, CD5, Bcl-1 positive atypical lymphoid cells regarded as neoplastic infiltration. Renal biopsy showed intravascular and interstitial atypical lymphoid cells as groups of 4–5 cells. These atypical lymphoid cells were CD20, CD5 Pax-5 and Bcl-1 positive. Pathological and immune-histochemical findings of biopsies were consistent with MCL. Positron emission tomography determined iliac, obturatory lymph nodes and avidity at renal hilum level. Patient was planned to receive 6 cures of Rituximab, cyclosporine, hydroxydaunorubicine, oncovine, prednisone. After first R-CHOP cure he suffered a massive pulmonary emboli and died. **Conclusions:** Infiltration of renal parenchyma by lymphoma cells is very rare and is rated in about 1 % of cases. There are only three cases of renal MCL infiltration with ARF. Our case is the fourth case of MCL presenting with ARF due to neoplastic cell infiltration while this is the first case that neoplastic MCL cells to be shown in the lumen of renal vessels, capillaries. The case demonstrates for the first time the possibility of intravascular renal infiltration by MCL. It also revealed the importance of the renal biopsy as a useful diagnostic choice in case of kidney impairment in lymphoma patients.

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Impact of an out-patient based strategy for the management of acute deep venous thrombosis in Saudi Arabia

Al eissi Salih, Hosny G.

Internal Medicine, King Abdulaziz Medical City, Central Region, Riyadh, Saudi Arabia

Introduction: In the past 20–30 years, management of acute DVT has been revolutionarised with the advent of low molecular weight heparin [1,2,3]. There is good evidence that management of DVT on an out-patient basis is safe, feasible and effective [5,6,7]. Also, in today's environment, of pressure on beds in acute hospitals, there is a need for admission avoidance strategies to facilitate savings in terms of bed days and money. **Methods:** We conducted a retrospective chart analysis of patients diagnosed with DVT between 2005 and 2012 to identify those suitable for out-patient management. **Aim:** Our aim was to evaluate the proportion of patients that would be eligible for out-patient treatment and the savings, in terms of bed days and money. **Results:** We found 190 patients diagnosed with DVT. 80 of these were eligible for out-patient management. Thus, 42.1% were eligible for out-patient treatment. Average length of stay was 7.88 days. 630 bed days would have been saved, or 78.75 bed days per year. Cost savings would be SR 945,000 or SR 118,125 per year. **Conclusions:** The percentage of patients eligible was below contemporary international levels, yet in keeping with the more conservative rates, which is appropriate for a new pathway. However, there are still cost savings and bed savings to be appreciated. It would be the first step to admission avoidance pathways in our institution.

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Distal mummification of all limbs — An odd presentation of multiple myeloma

J. Jacinto[a], A. Malheiro[a], A. Pestana[a], C. Gomes[a], M.I. Correia[a], M. Capelo[a], T.E. Freitas[a], P. Freitas[a], F. Jacinto[b], L.R. Brazão[c]

[a]*Department of Internal Medicine, Central Hospital of Funchal, SESARAM, Funchal, Portugal*
[b]*Department of Hemotology/Oncology, Central Hospital of Funchal, SESARAM, Funchal, Portugal*
[c]*Department of Internal Medicine (Chief/Director), Central Hospital of Funchal, SESARAM, Funchal, Portugal*

Introduction: Multiple myeloma (MM) is a plasmocytic malignant proliferation of a single clone resulting in an overabundance of monoclonal immunoglobulins. Frequently presents with bone pain or fracture, renal failure, susceptibility to infections, anemia, and hypercalcemia. Symptomatic blood hyperviscosity manifestations are uncommon, but may arise from this condition (particularly in IgA or IgG types). The Hyperviscosity Syndrome results of vascular stasis, reduced microcirculatory flow, hemostasis disorders and subsequent organ hypoperfusion. The clinical spectrum is broad, but more commonly presents with mucosal bleeding, visual and neurological changes (Bing–Neal syndrome), although constitutional and cardiorespiratory symptoms may also be present. The authors describe a highly unusual case of MM, which promptly presents with severe peripheral occlusive ischaemia, rapidly progressing to distal necrosis — dry gangrene of the nose and distal extremities of all four limbs. This catastrophic and particularly dramatic presentation of an otherwise fairly common disease – Multiple Myeloma – is almost unprecedented, with only a few cases reported worldwide. **Case presentation:** An 80 year-old woman was admitted to the Emergency Department presenting with quadrigangrene of limbs and nose, and variable signs of severe peripheral ischaemia and distal necrosis. Medical history of ischaemic heart disease, atrial fibrillation and chronic vertiginous syndrome. Polymedicated and anticoagulated on warfarin for several years. Despite limited information, the authors learnt that the patient was partially-dependent on her daily life activities, but overall lucid. About two months earlier she started complaining of "bluish", painful cold fingers of hands and feet, and severe intermittent Raynaud phenomena, accompanied by worsening vertigo and blurred vision. She then sought private medical assistance and prescribed with oral corticosteroids, as warfarin was replaced with enoxaparin. However, it rapidly unraveled to severe acrocyanosis, permanent Raynaud, resulting in peripheral ischaemic lesions and ulcerations, distal necrosis and progressive mental deterioration. On arrival, she was stuporous, moaning and not responsive to interrogation. Severely dehydrated, pale, cachectic, but hemodynamically stable. No tachypnea, fever, nor lymphadenopathy. Cardiopulmonary auscultation: arrhythmia (HR around 80); basal bilateral fine crackles. Dry gangrene was observed on all four distal limbs and nasal pyramid (complete mummification of the 5th finger of both hands, both first toes and gangrenated tip of the nose). Several areas of peripheral erosive lesions and livedo reticularis were also seen in both forearms, forelegs and auricles. All distal pulses were palpable and symmetrical. The Doppler showed vascular permeability. Laboratory findings: anemia, WBC 55,100 cells/mm3, normal platelets and increased C-reactive protein. Peripheral blood smear with 2% plasma cells and roleaux formation. INR 1,43 and D-Dimer 11898. Remarkably, the metabolic panel showed no significant derangement. Globulin gap = 7,18. Serum protein electrophoresis demonstrated monoclonal gammopathy, confirmed by bone marrow aspirate that also revealed plasmacytic infiltrate of 21% - compatible with Multiple Myeloma. Despite the immediate aggressive treatment with plasmapheresis, steroids and broad-spectrum antibiotics, the outcome was fatal only 96 hours after admission, rendering impossible further investigation.

Conclusion: Similar case reports are remarkably scarce in the literature, ascertaining the oddity of this multiple myeloma clinical presentation and compelling the authors to share knowledge.

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Hemolytic uremic syndrome — A challenging case of hemolytic anemia

M. Fonseca[a], M.F. Barros[b], R. Vaz[b], A. Praça[b], J. Frazão[b]

[a]*Internal Medicine, Hospital de Braga, Braga, Portugal*
[b]*Nephrology, Centro Hospitalar de São João, Porto, Portugal*

Background: Hemolytic Uremic Syndrome (HUS) is a disease of nonimmune (Coombs negative) hemolytic anemia, low platelet count and renal impairment. Anemia is severe and microangiopathic in nature, with fragmented red blood cells (schistocytes) in the peripheral smear, high serum lactate dehydrogenase (LDH), circulating free hemoglobin and reticulocytes. In children, the disease is most commonly triggered by Shiga-like toxin (Stx) producing E. coli and manifests with diarrhea, often bloody. Non-Stx associated HUS comprises a heterogeneous group of patients in whom an infection by Stx producing bacteria could be excluded as cause of the disease. It can be sporadic or familial. Collectively, non-Stx-HUS forms have a poor outcome. **Methods:** The authors present a case of a 32-year-old black man, resident in Angola where he works for the last 4 years, with history of Systemic Lupus Erythematosus (SLE) since he was 15-year-old. Medicated with plasmoquine until 1 year ago, having abandoned both his medical appointment and his medication for economic issues. Paternal history of Sickle Cell Anemia. He presents to the Emergency Room (ER) complaining about asthenia, Raynaud-like phenomenon and arthralgias for as long as one month, and he refers abdominal pain, vomiting and one episode of diarrhea since last week. Without reference to fever, myalgias or chills. His physical examination was irrelevant besides icteric sclerae and mucosal pallor. **Results:** On admission to the ER he presented with hemoglobin 5.4 g/dL, normal WBC count, thrombocytopenia less than 10,000/μL. Positive direct Coombs (9/12), numerous schistocytes described, reticulocyte production index > 2.5, LDH: 1540 U/l, decreased serum haptoglobin, total bilirubin: 2.3 mg/dL, indirect bilirubin: 1.6 mg/dL. Acute kidney injury (Creatinine: 2.2 mg/dL, BUN: 53.3 mg/dL), non oliguric. Urinalysis revealed hematuria and hemoglobinuria. Research of Plasmodium in the blood was negative. Complement was in the normal range. Thoraco-abdominal-pelvic angiography CT-scan was unremarkable. He began a regimen consisting of daily plasmapheresis, corticosteroids (1 mg/kg per day) and transfusions of red blood cells and platelet concentrates. He showed initial recovery of thrombocytopenia and improvement in parameters of hemolysis, such as serum LDH levels. On the tenth day of hospitalization was observed worsening of thrombocytopenia and therefore plasma exchange was intensified by increasing the volume of plasma replaced to 1.5 plasma volumes per exchange. He was hospitalized for a total of 40 days, having performed 25 sessions of plasmapheresis, eight of which on alternate days. At the time of discharge, he had normal platelet count and LDH. Renal function was normalized. **Conclusion:** The authors present a case of hemolytic anemia secondary to HUS, highlighting the variety of other potential differential diagnosis in an individual with a history of SLE, sickle cell anemia and who lives in an endemic area for Malaria. The availability of effective treatment has created an urgency for establishing the diagnosis of HUS. The platelet count seems to be the most important parameter on which to base treatment decisions. Persistence and patience with plasma exchange may be the most effective practice. Prolonged courses of plasma exchange treatment, with frequent exacerbations when treatment is tapered or stopped, are characteristic of patients with idiopathic HUS.

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Acquired hemophilia A: A critical bleeding syndrome in the elderly

A.R. Oliveira[a], A. Raposo[a], A.A. Santos[b], M.J. Diniz[b], C. Marques[a], J. Almeida[a]

[a]*Medicine Unit 1.2, Lisbon Central Hospitalar Center, Lisbon, Portugal*
[b]*Immunohemotherapy Unit, Lisbon Central Hospitalar Center, Lisbon, Portugal*

Acquired hemophilia A is a rare and serious bleeding disorder characterized by autoantibodies directed against circulating coagulation factor VIII (FVIII). It has a bimodal age distribution, with a first peak of incidence occurring in young women in the postpartum period and a second major peak in the elderly, frequently associated with malignancy, drugs or autoimmune diseases. Other rare causes including infections or inflammatory bowel diseases have been identified. Our patient, a 79-year-old man with controlled hypertensive cardiac disease and benign prostatic hypertrophy presented with multiple spontaneous haematomas of the lower limbs and of iliopsoas muscles, without previous episodes or family history of bleeding disorders. Laboratory investigations revealed severe anaemia (haemoglobin 6.1 g/L) and normal platelet count. Coagulation tests showed prolonged activated Partial Thromboplastin Time (aPTT) with normal Prothrombin Time (PT) and INR. APTT was not modified after mixing test, and the screening for lupic anticoagulant was negative. Clotting factor measurements showed isolated low FVIII level (1.8 %; normal 50–150%) and FVIII inhibitor was 2.0 BU/ml, consistent with Acquired Haemophilia A (AHA). Treatment was instituted with red cell transfusion, bypassing agents and corticosteroids, achieving control of the acute bleeding and clotting abnormalities. He was discharged after 12 days on prednisolone 1 mg/kg/day, gradually reducing every month, without recurrence of bleeding. No identifiable cause was found after investigation and the patient remains stable 5 months after presentation. In approximately 50% of the cases of AHA no underlying condition is identified, but there is an important risk of solid tumours or hematologic malignancies associated to the disease at this age, so our patient will be maintained under close surveillance. With this case we aim to raise awareness of this uncommon but potentially life-threatening cause of a bleeding diathesis. A learning point is that the pattern of bleeding in these patients differs from that in inherited Haemophilia A and tends to occur in soft tissue, muscle, retroperitoneal space, and gastrointestinal or genitourinary tracts. In contrast with patients with inherited Haemophilia A, haemarthroses are rare. In this case early recognition enabled prompt immune suppression and a successful outcome. In any patient who presents with recently-onset severe or deep tissue bleeding and an unexplained isolated prolonged aPTT, acquired Hemophilia A should be considered.

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