Not an ordinary UTI: A case of multiple myeloma stage III with no manifestation of bone pain, hypercalcemia or osteolytic lesions

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A 63 year old AAM was admitted for dysuria and brown colored urine of 2 weeks' duration. He had no fever but had chills on and off. He reported a 23 lb weight loss and a decreased appetite. Workup on admission revealed anemia with a hemoglobin of 7.5 g/dL and renal insufficiency with creatinine as 1.67. He was treated with intravenous ceftriaxone which relieved his dysuria and discolored urine. However, his renal insufficiency persisted despite hydration and antibiotics. During his hospitalization, he was found to have worsening anemia, renal failure and weight loss. Due to his anemia and renal insufficiency, further workup was initiated. With the constellation of renal insufficiency stage III along with macrocytic anemia, multiple myeloma was suspected by the medicine team. Serum protein electrophoresis showed M protein of 4.4 g/dl with IgG kappa and free lambda on serum immunofixation. IgG was 6911 mg/dL. Kappa light chains were 622.3 mg/L with kappa/lambda ratio of 3.66. Subsequently, bone marrow biopsy showed 90% cellularity with 70-80% plasma cells that were kappa restricted. The following cytogenetics by FISH was reported: CCND1-IGH fusion, extra signal for chromosome 9 and loss of one copy of 13q14. Interestingly, the patient denied bone pain and had no lytic lesions on skeletal survey or MRI of the spine. He also did not have hypercalcemia; instead he actually has hypocalcemia with latest calcium level as 7.7 mg/dl. The patient was diagnosed with IgG kappa multiple myeloma, International Staging System Stage III, as his B2-microglobulin level is 10.3 mg/L.

Cardiac failure in a neonate-pulsating brain: Successful endovascular embolization of vein of Galen malformation

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A n inborn neonate, female, the third child of consanguineous Kuwaiti parents, 37 weeks gestation, uneventful pregnancy for the mother with no high risk factors was admitted into the neonatal intensive care at 24 hours of age, due to respiratory distress with tachypnoea, respiratory rate of 60/minute, cardiac rate of 110/minute, normal mean systemic blood pressure with a slightly widened pulse pressure of 25 mm of Hg, normal oxygen saturation and minimal hepatomegaly. There were no dysmorphic features, acyanotic in room air, normal cardiac examination with only a persistent gallop rhythm and a mild cardiomegaly on X-ray chest. The Hct was normal. The child was evaluated by cardiologists on three days and diagnosed as having high output failure without any structural congenital cardiac defect or myocardial dysfunction clinically and by echocardiogram. Aggressive management for cardiac failure was instituted. Endocrine work up showed no thyrotoxicosis. On the 3rd day of life, cranial ultrasound performed routinely by neonatologist showed a vein of Galen malformation, confirmed by angiogram. The child at one month of age received endovascular embolization of the vein of Galen malformation at the institute in Paris. The cardiac failure was controlled. At 6 years of age, the child has normal growth and development, both neurological and school performance. There is no hydrocephalus, only one episode of febrile seizure at 8 months of age with normal electroencephalogram repeated angiographic evaluation showed obliteration of aneurysmal malformation. This clinical report illustrates early recognition of vein of Galen malformation, as a cause of neonatal cardiac failure by the neonatal team and early endovascular embolization management with trans-arterial deposition of n-butyl cyanoacrylate into fistula sites shows improved outcome without complications in childhood. Treatment of refractory heart failure in neonatal VGAM with modern prenatal and pediatric neuro-endovascular care results in significantly improved outcomes with presumed cure and normal neurological development in most.