TITLE: HYPEREOSINOPHILIC SYNDROME PRESENTING AS STROKE IN ELDERLY; A RARE CAUSE
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ABSTRACT: Stroke is the leading cause of neurological disability and mortality worldwide affecting more of elderly population. Hypertension and diabetes are deadly duo responsible for most of the cases of stroke. Hypereosinophilia is reported in the literature as an uncommon cause of stroke. Hypereosinophilic syndrome is rare and true prevalence is unknown. In one study the estimated prevalence was between 0.36-6.3 per 100000. Stroke incidence in hypereosinophilic syndrome has been estimated to be around 12%.

METHODOLOGY: A 60 year lady, housewife presented with sudden onset weakness of left side of body for 2 days. There was no h/o fever, headache, vomiting or seizure. There were no symptoms or signs of sensory deficits, dysphagia, dizziness, chest pain, palpitations or dyspnea. No past history of respiratory illness or any skin condition was present. No h/o T2 DM /hypertension or any significant family history. Her general examination showed pulse of 80/min, regular, all peripheral pulses palpable, blood pressure 130/82 mmHg, respiratory rate of 18/min, SPO2- 96% at RA, absence of pallor, icterus, clubbing, cyanosis, lymphadenopathy, or oedema. She was conscious, oriented with normal higher mental function, with intact cranial nerves. Her motor system findings showed normal nutrition, spasticity in the left upper and lower limbs (Modified Ashworth Scale-1), power- 2/5 in LUL and LLL (Medical Research Council-MRC scale), brisk deep tendon jerks on LUL &LL, Plantar- Right flexor and left extensor, normal right side with normal sensory system, cerebellar, and rest of nervous system examination. Her other systems were unremarkable.
Her investigations revealed hemoglobin 11.69g/dl, leucocyte count 1,51,300 cells/cumm with neutrophils-8.5%, lymphocytes-6.3%, eosinophils-83.86%, PCV- 3.9 milllion cells/ml, peripheral smearshowing no hemoparasite seen, no atypical cell seen, normocytic normochromic picture with eosinophilia, absolute eosinophil count- 92880 cells/cumm, platelet count- 2.6 lac /cumm, serum bilirubin total-0.64mg/dl, SGOT- 153 IU/L, SGPT-178 IU/L, ALP-308 IU/L, prothrombin time 15.5, INR1.16, normal thyroid, renal profile, B12, homocysteine, folate,HB1C, IgE levels and vasculitic profile. Her chest xray, ultrasound abdomen, CT thorax were unremarkable. Her electrocardiogram revealed NSTEMI features, however her echocardiogram was normal with Serum Trop T 1.25 which increased to 1.55 after 6 hr and serum Pro BNP -13500 levels. MRI Brain showed infarcts in multiple vascular territories with normal angiogram.Bone marrow examination revealed hypercellular markedly increased eosinophils and its precursors, without immature cells or parasite/granuloma. Stool for ova and parasite was found to be negative. Genetic testing for primary leukemia was negative(FISH for MPN panel – BCR/ABL, PDGFR,PDGFRB,JAK2 and FGFR1 rearrangements).

DISCUSSION: Hypereosinophilic syndrome is defined as association of hypereosinophilia (absolute eosinophil count > 500), with eosinophil mediated organ damage, provided other causes of organ dysfunction has been ruled out. In our case absolute eosinophil count was 99000/cc and we ruled out all possible causes of hypereosinophilia and hence diagnosis of idiopathic hypereosinophilic syndrome kept. Patient was put on steroids , hydroxyurea and supportive treatment. The mechanisms by which eosinophilia can cause neurological dysfunction are multi-factorial, 1) due to embolism from a focus of endomyocardial fibrosis or 2) through endothelial dysfunction mediated by hypereosinophilia. Eosinophils cause direct cytotoxicity through the local release of toxic substances including cationic proteins, enzymes, reactive oxygen species, pro-inflammatory cytokines, and arachidonic acid derived factors. The degree of end-organ damage is heterogeneous, and usually there is no correlation between the level or duration of eosinophilia and the severity of organ damage.

CONCLUSION: Hypereosinophilic syndrome is a rare disorder in itself and its presentation in elderly, is usually uncommon . The element of hyperviscosity involved ,meets the Virchow triad for the pathogenesis of thrombus formation that might lead to stroke and myocardial infarction in this case. So, as strokes can cause significant residual impairments, some of which are refractory to medical management, it is imperative for clinicians to be aware of this uncommon but treatable etiological factor for stroke.