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IVIG INDUCED SPLENIC INFARCT.

Pharma

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ABSTRACT

Patient 65years came with chief complain of upper and lower limb weakness and on nerve conduction test diagnosing Guillen Barre syndrome. Patient was given intravenous immunoglobulin (IvIg) and presented with abdominal pain over left side, nausea and vomiting. On investigation HRCT showed splenic infarct. making this a contributor to splenic infarct and a rare complication of an approved method of treatment.

KEYWORDS

BACKGROUND

Splenic infarct is a wedge-shaped lesion due to lack of blood supply. The infarct is wedge shaped representing the segment supplied by concerned end artery. Drug induced isolated splenic infarction very rare, occurs in combination with myocardial, renal infarct The American Academy of Neurology guidelines suggest that treatment of GBS may consist of either plasma exchange or IVIg. IVIg has been noted to cause rare complications including venous thrombosis, myocardial infarction and stroke. In our patient, we describe the occurrence of splenic infarct provoked by the initiation of IVIg treatment in Guillain-Barre syndrome and a rare complication of an approved method of treatment.

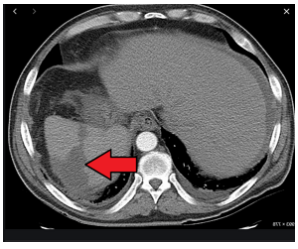
CASE PRESENTATION

Patient 65years old male came with chief complain acute attack of transient weakness of upper and lower limbs. After being admitted for 5 days patient was diagnosed with pneumonia, after being put on ventilator. He denied a recent travel history, local or international. He denied a family history of autoimmune diseases, communicable diseases or cancer. The patient was alert and oriented to person, place and time, with no signs of head or vertebral trauma noted. She had no facial asymmetry and no pronator drift, and performed an intact finger-to-nose test. Cardiovascular, pulmonary and abdominal examinations were unremarkable.

The patient was diagnosed with Guillain-Barre syndrome for which patient was started on IvIg (human normal immunoglobulin) 5gm/L infused over 2 hours. Treatment was continued till 30 g was infused over 2-3 hours for 5 days. Approximately 8 days after the completion of this regimen, the patient began reporting severe, sharp abdominal pain located at the left upper quadrant. This pain was accompanied nausea, vomiting and elevated white blood cell count.

INVESTIGATIONS

Neurological work up demonstrated an abnormal nerve conduction study with electrophysiological evidence of an acute demyelinating polyneuropathy. Owing to high suspicion of GBS, the patient was started on IVIg at 5gm/L over 2 hours for 5 days. Patient complains of severe, sharp abdominal pain located at the left upper quadrant, nausea, vomiting. Laboratory findings included Haemoglobin(11.0gm/dl), Total leukocyte count (15300/mm3), Platelet counts (711000/mm3). H.R.C.T showed in farcted lesions in splenic infract.

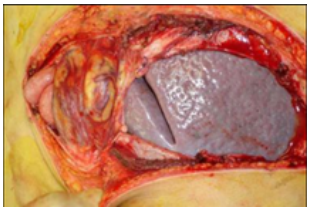


DIFFERENTIAL DIAGNOSIS

On initial presentation, the differential diagnoses for a 65year old man presenting acute attack of transient weakness of upper and lower limbs and inability to walk included stroke, acute inflammatory demyelinating polyneuropathy, transverse myelitis, multiple sclerosis, vitamin B12 deficiency (causing subacute combined degeneration of the spinal cord) and HIV infection. Along with a splenic infarct, patients who present with acute left upper quadrant pain should be evaluated for peptic ulcer disease with possible perforation, pancreatitis and pyelonephritis.

TREATMENT

HRCT imaging revealed a splenic infarct, patient has given supportive treatment, surgical opinion was taken for further treatment which was splenectomy



OUTCOME AND FOLLOW-UP

Patient has given supportive treatment, splenectomy was done, following which the patient recovered.

DISCUSSION

The patient's initial diagnosis of GBS was confirmed by nerve conduction studies, as well as by a strong clinical suspicion. The American Academy of Neurology recommendations for treatment of GBS includes the use of either plasma exchange or intravenous immunoglobulin[1]. IVIg is considered superior to plasma exchange for GBS, while also being the preferred treatment for GBS[2,3] Aside from its use in the treatment of GBS, IVIg has shown significant benefit in the treatment of other conditions as well, including idiopathic thrombocytopenic purpura, idiopathic inflammatory myopathies, ANCA-associated vasculitis, Kawasaki disease and chronic

inflammatory demyelinating polyneuropathy.[4] Generally considered a benign treatment, IVIg carries with it the rare, but serious, risk of thrombosis, which may manifest as a myocardial infarction, cerebral vascular infarct or even end-organ ischaemia. The cause of thrombus formation is likely multifactorial. Proposed mechanisms include increased viscosity post infusion, platelet aggregation, increased activation of specific coagulation factors, localised vasoconstrictive cytokine release and subsequent vasospasm[5,6]. It has been suggested that, along with a reduction of IVIg infusion flow rate, patients may benefit from prophylactic treatment with antiplatelet or anticoagulant therapy; however, there is not enough evidence to support the efficacy of these interventions. The signs and symptoms exhibited by this patient, including left upper quadrant pain and tenderness, fever and leucocytosis, are entirely consistent with splenic infarction, which was later identified through radiological imaging[7,8]. After tracing the sequence of events during the patient's hospital course, it was determined that the likely cause for this infarction was due in large part to IVIg infusion used in the treatment of GBS. To the best of our knowledge, this is the first report of splenic infarction caused by intravenous immunoglobulin infusion. According to Causality by WHO UMC Scale and Naranjo scale score comes to 3 that is between 1-4 hence possible.

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