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A RARE ASSOCIATION OF CHRONIC INFLAMMATORY DEMYELINATING POLYNEUROPATHY WITH ULCERATIVE COLITIS

Medicine

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ABSTRACT

Chronic inflammatory demyelinating polyneuropathy (CIDP) is a rare complication of Ulcerative colitis and it is uncertain whether it is associated with Ulcerative colitis itself or with its treatment. We describe a case of CIDP-like neuropathy as an initial symptom of Ulcerative colitis. A 17 years old male patient presented with symmetrical weakness of both lower extremities with paresthesia of both hand and feet. With impression of AIDP patient was treated with plasmapheresis and discharged on tapering steroid therapy. After 3 months again presented with bilateral weakness of all four limbs associated with diarrhea and fever. Patient was diagnosed with Ulcerative colitis ,so considering previous episode as 1st episode of CIDP and with rare association with Ulcerative colitis . Patient was treated with high dose of steroids and immunosuppressive therapy. Neurological and gastrointestinal symptoms remarkably improved after treatment.

KEYWORDS

CIDP, Ulcerative Colitis ,IBD,Steroids

INTRODUCTION

Chronic inflammatory demyelinating polyneuropathy (CIDP) is an acquired, immune mediated neuropathy affecting peripheral nerves characterized by relapsing-remitting or progressive course. CIDP has a rare association with Ulcerative colitis.

CASE REPORT :

We reported a case of a 17-year-old male admitted with complaint of ascending weakness of both lower extremities that had progressed for about 3-4 days. General Physical examination was normal. On neurologic examination, he had symmetric weakness of both lower extremities with power MRC grade 2 & paresthesia of both hands and feet. Deep tendon reflexes were absent in both upper and lower extremities. Routine laboratory investigations, including complete blood count, biochemistry, urine analysis and C-reactive protein, were normal. Cerebrospinal fluid (CSF) protein level was 30mg/dL without pleocytosis. Other laboratory investigations including, thyroid function, anti-nuclear antibodies, viral markers, were negative (Table:1). Motor nerve conduction study (NCS) showed demyelinating type of polyneuropathy. With the impression of AIDP, Patient was treated with 5 cycles of plasmapheresis and after improvement patient was discharged with tapering course of steroid. On discharge, the patient had power MRC grade 4 in both lower extremities.patient was on regular follow up.

TABLE :1

CBC	Hb-13.4 g/dl, WBC-10700/cmm, platelet 375000/cmm
RFT	Creatinine-1.06 mg/dl , urea-29.30mg/dl S.Sodium-136.3mg/dl S.Potassium-4.2mg/dl
LFT	SGPT-25.7IU/L Direct bilirubin -.08mg/dl Total bilirubin -1.24mg/dl
CRP	Negative
Urine Analysis	NAD
Thyroid Function Test	S.TSH-0.764 uIU/mL S.free t3-2.17pg/ml S.free t4-0.99ng/dl
Viral Markers	HIV, Hepatitis B& C nonreactive
CSF Protein	30 mg/dl without pleocytosis
ANA	Negative
Nerve conduction studies	Demyelinating type of polyneuropathy
MRI Brain (P+C) with whole spine screening	Normal

After 3 months patient presented with gradually progressive bilateral lower limb followed by upper limb weakness with power MRC grade 2, associated symptoms of fever and diarrhea mixed with blood & mucus . Patient was investigated with routine investigations including heamatological and biochemistry which were with in normal limits ,and special investigation Stool Routine and microscopic examination, USG Abdomen and pelvis,CECT abdomen, colonoscopy (with colonic biopsy)(Table :2) . These were suggestive of inflammatory bowel disease and NCS was suggestive of demyelinating type of polyneuropathy. So in view of 2nd episode of symmetric motor weakness without sensory and bowel / bladder involvement after 3 months of previous episode, the first episode was considered as 1st episode of CIDP with rare association of Ulcerative colitis. Patient was treated with high dose of steroid prednisolone 60mg daily for 2 weeks followed by tapering dose and mesalazine 2gm/day orally and also put on immunosuppressive agent azathioprine 100mg/day. Patient improved clinically with marked neurological improvement. On discharge, patient had power of MRC grade 4 in all the extremities. Also there was improvement in gastrointestinal symptoms including Diarrhea. Motor nerve conduction study after two months showed improvement of electrophysiologic parameters and waveforms.

TABLE 2

CBC	Hb-12.7 g/dl, WBC-9400 , platelet-350000
RFT	Creatinine-1.0 mg/dl , urea-23.50mg/dl S.Sodium-139mg/dl S.Potassium-4.6mg/dl
LFT	SGPT-22.9IU/L S.BILIRUBIN Direct-.06mg/dl Total-1.4mg/dl
CRP	Negative
Urine Analysis	NAD
Thyroid Function Test	S.TSH-0.784 uIU/mL S.free t3-2.22pg/ml S.free t4-0.95ng/dl
USG	Few of small bowel loops prominent and fluid filled measuring 24-26 mm in external diameter
Abdominal CECT	Diffuse circumferential oedematous wall thickening involving caecum, ascending colon, hepatic flexure, transverse colon, splenic flexure, descending colon, sigmoid colon, rectosigmoid junction and rectum with mural stratification and mucosal enhancement with loss of haustration
Biopsy	Colonic cryptitis and crypt abscess and transmucosal lamina propria infiltration with plasma cells and neutrophils

DISCUSSION

CIDP is characterized by a symmetric, motor predominant peripheral neuropathy that causes both proximal and distal weakness and sensory impairment that is usually greater for vibration and position sense than for pain and temperature sense and areflexia.

Following points may help in diagnosis of CIDP over GBS :
(1) Deterioration after 8 weeks from onset of weakness
(2) Deterioration occurs more than 3 times
(3) No loss of independent ambulation
(4) Absence of cranial nerve involvement

IBD is a relapsing inflammatory disorder that can affect the entire gastrointestinal tract from the mouth to the anus. It is considered a systemic disease because it is frequently accompanied by extraintestinal manifestations, including uveitis, arthritis, ankylosing spondylitis, primary sclerosing cholangitis and erythema nodosum, among others. Neurologic complications can also occur; however, the exact prevalence of neurologic involvement associated with IBD, including CIDP, is unknown.

Concerning the peripheral nervous system, neuropathy is a recognized complication of both UC and CD and usually is present as an acute form, such as Guillain-Barrè syndrome, or as a chronic inflammatory demyelinating polyneuropathy. Sometimes, it manifests as sensorimotor axonal polyneuropathy or as single axonal mononeuritis. The main pathologic lesions reported in literature are represented by perineuritis, axonal damage, or demyelination. The incidence of neuropathy in adults is about 1%.

The pathogenesis of peripheral nervous system damage in inflammatory bowel disease is yet to be elucidated, although it seems to be related to immune mechanisms; therefore, treatment with immunotherapy is recommended. In addition, CIDP may appear as iatrogenic-related disorders associated with several drugs used in controlling inflammatory bowel disease activity; finally, CIDP may also be caused by micronutrient deficiencies secondary to malabsorption-related disorders.

Pathophysiology of IBD is believed to involve inappropriate inflammatory responses to intestinal microorganisms in genetically susceptible hosts. The components of the type 17 helper T-cell pathway are features of this inflammatory response, which involves proinflammatory mediators produced by immune cells in the intestines such as tumor necrosis factor-α (TNF-α), interleukin-1β, -6, -12,-23 and chemokines. These factors overlap with those responsible for the pathogenesis of CIDP and may modulate T-cell activation involving the peripheral nervous system to generate inflammatory lesions. The early improvement and subsequent worsening of grip strength after PE in our patient suggests that pathogenic humoral factors are crucial for the development of CIDP associated with IBD.

Studies done by Lossos A, River Y, Eliakim A et[9] show assessing 638 inflammatory bowel disease(IBD) patients found that 10 CD patients and 9 ulcerative co-litis (UC) patients presented neurologic complications with an incidence of about 3%. In particular, 6 UC patients were affected with peripheral nerve disorders, 5 CD patients with my-elopathy,3 CD patients with myopathy, and I CD patient with myasthenia gravis. Cerebrovascular disorders occurred in 2 UC and 2 CD patients.

Study executed by Larrode etal[10],to a peripheral sensorimotor neuropathy developed tn 4 patients with a parallel course to IBD

The present case shows the young patient with CIDP during course of Ulcerative colitis . the neurological complication of Ulcerative colitis concern with central nervous system but it may be associated with peripheral nervous system.

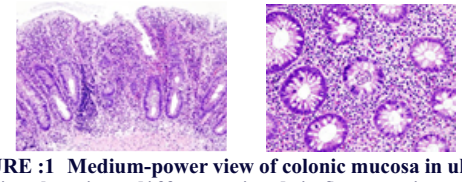

FIGURE :1 Medium-power view of colonic mucosa in ulcerative colitis showing diffuse mixed inflammation, basal lymphoplasmacytosis, crypt atrophy and irregularity,and superficial erosion. These features are typical of chronic active ulcerative colitis.

CONCLUSION

We report a case in which CIDP-like neuropathy was associated with Ulcerative colitis. The origin of CIDP is still unclear, but it is widely accepted that it is an autoimmune disease with underlying immunopathology involving autoreactive T cells and B cells. CIDP is associated with systemic illnesses, SLE, and other collagen vascular disorders, thyroid disease, nephrotic syndrome and inflammatory bowel disease.

Among extraintestinal complications of Ulcerative colitis, neurologic involvement is rare which includes cerebrovascular accidents, seizures, myelopathy, myopathy, and peripheral neuropathy. In our patient we can hypothesize that the neurologic involvement is the result of an autoimmune mechanism secondary to many factors like immunocomplex depositions, autoantibodies, or unknown toxic substances. Further studies are necessary to better understand these pathogenic mechanisms.

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