Case Report

Depression with Chronic Inflammatory Demyelinating Polyneuropathy

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ABSTRACT

Depression and chronic inflammatory demyelinating polyneuropathy (CIDP) both are chronic illness of different etiopathology and are usually not looked for together while screening a patient. However, both have got a long incubation period as well as have an overlapping symptom profile. Rarely, cases reported in literature talk both together. Here, we report a rare case with depression complicated by CIDP managed together to improve outcome of depression.

Key words: *Chronic inflammatory demyelinating polyneuropathy, comorbidity, depression*

INTRODUCTION

Depression and the autoimmune peripheral nerve injury condition called chronic inflammatory demyelinating polyneuropathy (CIDP) are both potentially disabling and have a long course and overlap of symptoms; So there is a association between the two diseases.

CIDP, chronic relapsing polyneuritis, steroid-dependent, demyelinating sensorimotor polyneuropathy primarily affect the limbs.[1]

Some clinicians consider CIDP to be the chronic form of the acute idiopathic polyneuropathy known as Guillain–Barre syndrome.[2-6] Both conditions are thought to be acquired autoimmune disorders. Separated from AIDP, or GBS, by Austin in 1958 on basis of a prolonged and relapsing course, enlargement of nerves, and responsiveness to corticosteroids.

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Features of CIDP include "progressive, sometimes relapsing, steroid-dependent, symmetric, proximal, and distal muscle weakness, variously accompanied by paresthesia, sensory dysfunction, and impaired balance." The symptoms tend to evolve slowly over 2 months or more.[7] The common CIDP variants include unifocal, multifocal, pure motor, pure sensory, sensory ataxic, and pure distal forms.[8] With the potential for such a variable clinical presentation, it is not surprising that diagnosis based solely on clinical signs and symptoms is difficult. The characteristic large fiber sensory loss and areflexia can suggest multifocal disease. CIDP may or may not have an associated pain component.[9] Majority of CIDP patients exhibit decrease in functional status, fatigue, and impairment. The duration of CIDP-related symptoms before

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diagnosis can range from 1.4 to 11.5 years.[10] This prolonged incubation time may negatively impact the ultimate clinical course for the patient, resulting in substantial physical dysfunction and a poor quality of life.[10-13] The need for immunosuppressive treatment, which often include long term use of corticosteroids and the uncertainties regarding the prognosis, present a special challenge to the patients, and dealing with these iatrogenic and situational problems can benefit from psychiatric consultation.[14]

Symptoms include muscle pain, irritable bowel syndrome, fatigue/tiredness, thinking or remembering problem, muscle weakness, headache, pain/cramps in abdomen, numbness/tingling, dizziness, insomnia, depression, constipation, pain in upper abdomen, nausea, nervousness, chest pain, blurred vision, fever, diarrhea, dry mouth, itching, wheezing, Raynaud's, hives/welts, ringing in ears, vomiting, heartburn, oral ulcers, loss/change in taste, seizures, dry eyes, shortness of breath, loss of appetite, rash, sun sensitivity, hearing difficulties, easy bruising, hair loss, frequent urination, painful urination, and bladder spasms.

Diagnosis of chronic inflammatory demyelinating polyneuropathy

In 1975, Dyke *et al*. were among the first to describe criteria for the diagnosis of CIDP, which included aspects of the clinical course (≥8 weeks' progressive weakness and other symptoms); the type of nerve fiber class affected (large nerve fibers) and the symmetry of distribution. Several more recent criteria have been developed for the diagnosis of CIDP, to include data from clinical manifestations, electrodiagnostic studies, imaging, cerebrospinal fluid (CSF) analysis, and/or pathology from nerve biopsy.[7,15] These studies were variously conducted and/or espoused by the American Association of Neurology (AAN), the European Federation of Neurological Societies, the Inflammatory Neuropathy Cause and Treatment study group, and the IGIV-C CIDP Efficacy study group.[16-21]

A relatively unusual approach used by one diagnostic criteria study was to seek a consensus of experts in the form of a Delphi exercise and then to define that consensus as the gold standard.[22] They justified their approach as follows: "Although this gold standard is fallible and vulnerable to criticism, in the absence of a reliable biological marker, this is currently the best surrogate of CIDP status." Subsequently, most authors have emphasized the value of objective electrodiagnostic and pathological findings in the diagnosis of CIDP.[22,23]

Diagnosis of depression

Diagnosis of depression was done using:

1. DSM 5 Depressive Disorders[24,25]
2. ICD 10 diagnostic criteria.[26]

CASE REPORT

A 53-year-old female a known case of depression for the past 15 years on serotonin reuptake inhibitor (SSRI) presented in the Outpatient Department of Psychiatry with history of pain in palm and sole region with difficulty in holding a bucket, inability to move few steps, and worrying about minor matters.

The patient, a known case of depression for the past 15 years, was maintaining well on SSRI till 5 months back, to start with she developed headache dull-aching continuous; burning type of pain moderate to severe, continuous throughout day. Complaints such as sleep disturbances, sadness, and incapacitation of day-to-day activities gradually increased in the last 5 months. The patient visited many physicians during this period for the above complaints.

Patient is nondiabetic, nonhypertensive without significant medical history.

Operated for anorectal surgery 5 years back. There was a positive family history of schizophrenia in brother.

The patient was admitted for detailed evaluation and investigation, and the patient was vitally stable. Neurological examination revealed generalized areflexia with diminished touch, pain, and vibration sense in upper- and lower-limb. Routine blood investigations including serum electrolytes and blood sugar were normal. Psychiatric evaluation was suggestive of low mood, appropriate affect, with coherent speech, intact memory, intelligence, insight, and judgment. No delusion or hallucinations were present. Patient was treated with pregabalin 150 mg + nortriptyline 20 mg/day at bedtime and escitalopram 10 mg/day (SSRI).

CSF was performed which was suggestive of raised protein (224 mg/dl) with normal glucose (54 mg/dl) and cell counts. Serum electrophoresis was negative for monoclonal band. Nerve conduction study was suggestive of sensorimotor, axonal + demyelinating polyneuropathy. This confirmed the diagnosis of CIDP. Autoimmune markers, ANA as well as ds DNA, were negative. Prednisolone 20 mg/day was added to existing treatment in consultation with neurologist.

Combined management in follow-up showed rapid reduction in the depressive symptoms and scores as various domains of depression were affected by the somatic and pain complaints and affecting the overall functionality [Table 1].

Table 1: Change in clinical presentation with time

HAMD 17	Baseline	Two Weeks	Six Weeks	Twelve Weeks	Twenty four weeks
score	*32*	27	*20*	10	**4**

HAMD: Hamilton Rating Scale for Depression

DISCUSSION

The physician's attribution of the patient's somatic symptoms to depression represented an inadequately-informed causal integration. Stresses in the lives of depressed patients are more numerous and more severe than in healthy normal people without depression as explained by the kindling hypothesis.

The stress-related physiological defect which actually pertains to depression is a hypothalamic-pituitary-adrenal (HPA) dysfunction which can be caused by:

a. In conjunction with amygdala activation leading to increased sympathetic tone which leads to increased sympathetic tone, which promotes the release of cytokines from macrophages, increase of abnormally elevated proinflammatory cytokines may be experienced as fatigue, loss of appetite, libido as well as hypersensitivity to pain[27]
b. Substance P levels in depression: Substance P is believed to inhibit the function of the HPA-glucocorticoid stress–response axis.[4,5]

Recognizing the potential variability of the presentation of each of the conditions relevant to this discussion, it would be expected that the presentation of depression/CIDP would be variable from one to another affected patient.

CONCLUSIONS

Depression and CIDP both are chronic illness of different etiopathology. Both have got a long incubation period, long course of illness, as well as an overlapping symptom profile.

Hence, the existence of a comorbid condition of different pathology complicating the outcome of depression should be considered before switching to another drug of different class when there is history of poor response to the same drug which showed adequate response and functional recovery earlier.

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Conflicts of interest
There are no conflicts of interest

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