“More than meets the eye” non secretory myeloma presenting as cidp in a patient with longstanding diabetic polyneuropathy. a diagnostic and therapeutic challenge.

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“MORE THAN MEETS THE EYE” NON SECRETORY MYELOMA PRESENTING AS CIDP IN A PATIENT WITH LONGSTANDING DIABETIC POLYNEUROPATHY. A DIAGNOSTIC AND THERAPEUTIC CHALLENGE.

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ABSTRACT

Diagnosing chronic inflammatory polyradiculopathy in patients with pre-existing diabetic sensorimotor polyneuropathy is a diagnostic challenge. We present a case of a 69 years old who presented with weakness of legs for two months, he was diagnosed as having CIDP on the background of diabetic sensorimotor polyneuropathy, further and extensive workup revealed the final diagnosis of nonsecretory myeloma. Diagnosing non secretory myeloma is itself a diagnostic challenge and usually first line investigations for the workup of myeloma are negative as was the case in our patient. Our patient with CIDP had raised free light chains of kappa which made the final diagnosis of kappa associated plasma cell dyscrasia.

Key words: Chronic inflammatory polyradiculopathy (CIDP), Non secretory myeloma, Diabetic sensorimotor neuropathy

INTRODUCTION

Chronic inflammatory polyradiculopathy (CIDP) is clinically defined as ‘chronically progressive, stepwise or recurrent proximal and distal and sensory dysfunction of all extremities, developing over at least 02 months, with absent or reduced tendon reflexes in all limbs and sometimes with cranial nerve involvement’[1]. The diagnosis of CIDP in diabetes patients may be significantly more difficult than in non-diabetics, due to mild demyelinating changes associated with diabetic sensorimotor polyneuropathy (DSP), in the setting of poor glycemic control[2]. We present such a challenging case of 69 year old male from Islamabad where reaching to a final diagnosis was an arduous journey and in itself was a diagnostic challenge.

CASE REPORT:

A 69 years old male with 35 years long history of type 2 diabetes Mellitus with complications of diabetic neuropathy, nephropathy CKD III, retinopathy, diabetic foot ulcer presented with two months history of gradual but rapid worsening of weakness in both legs, he was independent with activities of daily living prior to two months and was driving. The weakness was asymmetrical and involved left leg more than right and involved both proximal and distal muscles equally and at the same time. He denied any symptoms in upper limbs. There was no significant backache or urinary symptoms. There was longstanding numbness in both his feet for more than six years which was unchanged. Examination revealed wasting of left leg, marked ischemic lower limbs skin changes, diabetic foot ulcer on right first metatarsal. There was prominent wasting of small muscles in hands as well. Power was 4/5 on left leg both proximally and distally. Power was 4+ on right leg both proximally and distally. He had normal power in upper limbs. Sensory system examination revealed impaired pin prick sensation in gloves and stocking distribution, impaired vibration and position sense in legs up to ankles. Investigations revealed HB of 10.5g/dL microcytic hypochromic. His renal functions revealed creatinine of 2.7mg/dL and urea of 40mg/dL. Serum electrolytes showed low sodium of 128mmol/L, normal potassium and low bicarbonate of 16. His HbA1c was 8.7. His nerve conduction studies and Electromyography revealed changes suggestive of sensorimotor predominantly polyneuropathy with demyelination changes as shown in figure 1&2. Was it just the rapid progression of Diabetic Sensorimotor...
A 69 years old male with 35 years long history of type 2 diabetes Mellitus with complications of diabetic sensorimotor polyneuropathy. Our patient had neurological deterioration in 02 months and had raised CSF proteins of 136.6mg/dl which further supported the diagnosis of CIDP and there was evidence of demyelination on nerve conduction studies, however...
A 69 years old male with 35 years long history of type 2 diabetes was admitted with symptoms of weakness in both legs, which rapidly worsened. The patient had a past medical history of diabetic sensorimotor polyneuropathy (DSP), in the setting of poor glycemic control. We associated this with diabetic sensorimotor polyneuropathy, a challenge.

We performed an arduous journey and in itself was a diagnostic challenge. We evaluated the patient, and the result was a diagnostic dilemma. Diagnosing CIDP and there was evidence of CSF proteins of 136.6mg/dl which further supported the diagnosis. We concluded that diagnosing CIDP in patients with diabetes is important due to the implications for therapy and prognosis. The finding of raised CSF proteins helped us in making the diagnosis of CIDP. In such difficult cases finding of raised CSF proteins without CSF leucocytosis further supports the diagnosis of CIDP and this is next important investigation to

**DISCUSSION**

Diabetes patients who have changes suggestive of demyelination on nerve conduction studies (NCS) are usually considered to have a superimposed immune-mediated polyneuropathy, such as chronic inflammatory demyelinating polyneuropathy (CIDP). The differentiation of CIDP from DSP in patients with diabetes is important due to the implications for therapy and prognosis. The finding of raised CSF proteins helped us in making the diagnosis of CIDP. In such difficult cases finding of raised CSF proteins without CSF leucocytosis further supports the diagnosis of CIDP and this is next important investigation to

**Figure 2.** Electromyography of the patient showing neuropathic changes in form of broad motor unit potentials, some showing high amplitude and reduced interference pattern.
A 69 years old male with 35 years long history of type 2 diabetes Mellitus with complications of diabetic neuropathy, nephropathy CKD III, retinopathy. His medical history included multiple hospital admissions due to thrush rendering him nil by mouth. Steroids and antifungal were offered but refused by patient. Patient was given prosthetic seats and had to use them regularly. He was fit and active and underwent an arduous journey and in itself was a diagnostic challenge. Diagnosing Polyneuropathy or this presentation suggestive of CIDP was the main diagnostic dilemma here. Diagnosing Diabetic Sensorimotor Polyneuropathy is just the rapid progression of Diabetic Sensorimotor Polyneuropathy. Our patient had sensorimotor predominantly polyneuropathy with demyelination changes as shown in figure 1&2. Was it due to mild demyelinating changes all limbs and sometimes with cranial nerve weakness right leg both proximally and distally. He had normal sensory system examination of small muscles in hands as well. Power was 4+ on left leg both proximally and distally. Power was 4/5 on his feet for more than six years which was unchanged. Examination revealed wasting of left leg, marked wasting of his legs, bilateral foot drop and marked tremor of feet. Sensory system examination of lower limbs. There was no significant backache or urinary difficulties with first line treatments. We underwent plasma exchange but refused by patient. Patient was given biological therapy but refused by patient. We undertook the second line investigations and further extensive workup offered but refused by patient. Patient was given biological therapy but refused by patient. We undertook the second line investigations and further extensive workup.

### INTRODUCTION

Chronic inflammatory polyradiculopathy (CIDP) is clinically defined as 'chronically progressive, stepwise improvement and exacerbation of neuromuscular disability, clinical impairment and motor nerve conduction velocities, although there may be rapid deterioration post exchange[9]. Seventy percent of patients of CIDP respond to one or another of the three standard therapies and probably 90% respond overall [1]. The lack of response to treatment and minimal electrophysiological progression over time should lead one to suspect the diagnosis and undergo secondlineinvestigations[1].

Figure 3. MRI lumbosacral spine of the patient showing mild to moderate lumbar spondylosis.
second line investigations and further extensive workup which revealed diagnosis of non secretory myeloma. Serum protein electrophoresis has only a 60% sensitivity for detecting paraproteins in the context of neuropathy, so immunofixation, 95% sensitivity along with bence jones proteins and serum free light chains should be performed[1]. Non secretory myeloma are one of the rare cause of myeloma found in only 1 to 5% of the cases of multiple myeloma[6]. They often present as diagnostic challenge[6].

CONCLUSION

We conclude that diagnosing CIDP in patients with background diabetic sensorimotor neuropathy is a diagnostic challenge. We also recommend diagnostic workup for myeloma in CIDP should not just include serum and urine electrophoresis but should include immunofixation as well as serum free light chains ratio especially if the response to first line therapy is poor. More than meets the eye, never stop at any step, keep on looking for the rare causes which can make a single unifying diagnosis of all symptoms, as in this case of non secretory myeloma

REFERENCES:


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Author’s contribution:
FarheenNiazi; Study concept and design, protocol writing, data collection, data analysis, manuscript writing, manuscript review
Arsalan Ahmed; Study concept and design, data collection, data analysis, manuscript writing, manuscript review
Huma Abdul Shakoor; Study concept and design, data analysis, manuscript writing, manuscript review
SajidBukhari; data analysis, manuscript writing, manuscript review
UzmaBatool; data analysis, manuscript writing, manuscript review