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Diagnostic criteria of chronic inflammatory demyelinating polyneuropathy in diabetes mellitus

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Objective – The possibility of co-association between diabetes mellitus (DM) and chronic inflammatory demyelinating polyneuropathy (CIDP) has long been a focus of interest as well as of clinical significance. As CIDP is a potentially treatable condition, it is diagnosis in the context of DM is of great importance. However, diagnostic criteria to identify CIDP in patients with diabetes are not available. We propose a diagnostic tool that should help clinicians to decide what is the probability that a patient with diabetes might have CIDP. *Methods* – We list several clinical, electrophysiological, and laboratory parameters that, when combined, have the power of discriminating an immune-mediated neuropathy in patients with DM. By summing the points assigned to each of these parameters, we define four levels of probability for a patient with diabetes to have CIDP. To analyze the validity of the diagnostic toll, we applied it in three different patient populations: (i) Patients with diabetes with peripheral neuropathy, (ii) Patients with CIDP without DM, and (iii) Patients with diabetes with CIDP. *Results* – The scores of patients with diabetes without CIDP ranged from −7 to 2, while those of patients with DM–CIDP ranged from 2 to 20. The scores of non-diabetic patients with CIDP were similar to those of patients with DM–CIDP and ranged from 6 to 16. The mean score of patients with DM–CIDP was 9.083, while the score of patients with CIDP was 11.16 and that of patients with diabetic polyneuropathy was −3.59. *Conclusions* – These results show that this diagnostic tool is able to identify patients with diabetes with overlapping CIDP.

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Introduction

Chronic inflammatory demyelinating polyneuropathy (CIDP) is a symmetric, mainly motor, proximal, and distal, demyelinating peripheral neuropathy of a progressive or relapsing course. First described in detail by Dyck et al. in 1975 (1), the condition is considered an immune-mediated disorder, where inflammation is directed against peripheral nerve epitopes located mainly in the myelin sheath of peripheral nerves. This understanding is supported by electrophysiological and pathological data, as well as the beneficial response to immunomodulation and immunosuppression.

Although no specific predisposing factors have been clearly identified, several conditions can favor CIDP. This may include immunogenetic background, association with other dysimmune disorders such as connective tissue diseases (systemic lupus erythematosus, Sjogren syndrome, rheumatoid arthritis), sarcoidosis, thyroid disease, myasthenia gravis, lymphoproliferative conditions, and acquired immunological abnormalities (2, 3). Of note here, however, is the possible association of CIDP with diabetes mellitus (DM).

DM is the most prevalent cause of peripheral neuropathy (4). Its pathogenesis is not completely understood, but may include metabolic derangement of neuronal metabolism with resultant

axonal damage, disturbances in vascular supply by the vasa nervosum to peripheral nerves, and damage to nerve sheath and Schwann cells (4, 5).

Based on anecdotal experience, it was proposed that DM may herald CIDP. The evidence relies mainly on retrospective studies and on small cohorts of patients. In some, the methodology is not clear and the criteria for the diagnosis of CIDP vary. This may account for the conflicting conclusions (6–11).

Diagnosis of CIDP in the context of DM is not only significant to outline possible association, incidence, and eventual pathogenesis, but has important therapeutic implications. By and large, unlike diabetic neuropathy, CIDP is a treatable condition. Moreover, steroids, with proven beneficial effect in CIDP, are relatively contraindicated in DM. Introducing steroid therapy in a patient with diabetes should be based on convincing reasons and evidence.

However, the ability to identify CIDP in the context of DM is an immense challenge, as both conditions share not only similar symptoms and findings but may have identical electrophysiological abnormalities. In fact, there is no accepted electrophysiological framework for the diagnosis of CIDP, and there are at least 16(!) different sets of proposed criteria for CIDP diagnosis (12–24).

Thus, for example, even histology of peripheral nerve obtained by biopsy, that might be of help and has an important role when showing inflammatory demyelination, on many occasions is nonspecific. Unfortunately, negative findings do not rule out CIDP (23–25).

We therefore developed a diagnostic tool that may help clinicians who face patients with DM and peripheral nerve disorder, to approach this diagnostic dilemma in a systematic and logical scheme, in order to determine the probability of an immune-mediated neuropathy.

To validate the proposed diagnostic tool, we retrospectively analyzed 12 patients with a diagnosis of DM and CIDP, who are being followed up in our neuromuscular–neuroimmunological clinic, and compared their score at the time of CIDP diagnosis, with that of 18 patients diagnosed with CIDP (according to either the AAN criteria or the criteria proposed by Van den Bergh and Piéret- 14, 16) and 27 patients with diabetes who meet the clinical and electrophysiological diagnostic criteria of diabetic polyneuropathy (5). The diagnosis of CIDP in the DM–CIDP group was made according to the AAN criteria or the criteria proposed by Van den Bergh and Pie'ret (14, 16). All patients were treated with either corticosteroids, immunoglobulins, or plasmapheresis as a first-line therapy, which was effective in stabilizing or improving the clinical course in 10 of 12 patients. Two patients, who showed marked motor deterioration during the initial treatment, underwent sural nerve biopsy to confirm the diagnosis of CIDP and were then treated with cyclophosphamide, reaching clinical stabilization.

The method

Our approach is based on clinical, electrophysiological, and laboratory parameters. As there is an overlap between CIDP and diabetic neuropathy, we assembled a list of factors that are more likely to be present in CIDP and assigned points to each of them. We then list factors that are less probably associated with CIDP and are more common in diabetic neuropathy. When present, they reduce the likelihood of CIDP. Each point present is added or subtracted and the sum can rule out or suggest possible, probable, or definite diagnosis of CIDP in the context of DM.

When assigning the number of points for each parameter, those that are more specific and distinguishing are scored as 3 points, and those less characteristic of CIDP are scored 2 or 1 point according to their possible power of separating the two conditions.

Clinical parameters supportive of CIDP

Progressive\relapsing motor weakness that develops over a period of <6 months – Diabetic neuropathy is usually a slowly progressive condition, while CIDP has a more rapid course (Table 1). CIDP is a motor greater than sensory neuropathy (1) while diabetic neuropathy tends to be more a sensory condition (4).

Significant proximal motor involvement – Distal symmetric polyneuropathy (DSP) accounts for such a large proportion of peripheral nerve manifestations of DM that the terms DSP and diabetic neuropathy are used interchangeably (4). Proximal involvement in diabetic neuropathy is unusual, present with uncommon conditions such as diabetic amyotrophy or mononeuritis (4, 5). Thus, proximal motor involvement is evocative of CIDP.

Significant upper limb involvement, either symmetrical or asymmetrical – DM tends to be symmetrical and distal. Involvement of the limbs in DM is of an ascending type affecting more the lower than the upper extremities. Although CIDP tends to be ascending as well, the condition may

Table 1 Supportive parameters of CIDP

Parameter	Score
1. Clinical	
A. Progressive\relapsing motor weakness of 2–6 months	+3
B. Significant proximal motor involvement	+3
C. Significant upper limb involvement, either symmetrical or asymmetrical	+3
D. If sensory symptoms predominate, deep sensation > superficial	+2
E. Recent onset DM, relatively well controlled	+1
2. Electrophysiology	
A. Motor distal latency prolongation ≥50% above upper limit in at least two nerves	+1
B. Reduced motor conduction velocities ≤30% below lower limit in at least two nerves	+1
C. Prolonged F wave latency ≥20% above upper limit in at least two nerves	+1
D. Partial motor conduction block (at least 50% reduction in proximal negative peak CMAP relative to distal, if distal negative peak CMAP at least 20% of lower limit of normal values) or abnormal temporal dispersion of the CMAP (>30% duration increase between proximal and distal negative peak CMAP) in at least two nerves	+3
E. Distal CMAP duration (interval between onset of the first negative peak and return to baseline of the last negative peak) of ≥9 ms in at least one nerve and at least one other demyelinating parameter in ≥1 other nerve	+3
3. Ancillary studies	
A. CSF protein elevated ≥2 times the upper normal limit	+2
B. Associated serological biomarkers suggestive of other dysimmune inflammatory conditions (ESR, CRP, ANA, dsDNA, anti-Ro, anti- La, RF, protein electrophoresis, etc.)	+1

first involve the upper extremities, unlike DM (26).

If sensory symptoms predominate, deep sensation is more affected than superficial sensation – The sensory involvement in diabetic polyneuropathy is generally associated with damage to small, unmyelinated fibers leading to numbness, tingling sensations, and neuropathic pain in a stocking and glove distribution (4) while proprioceptive loss due to damage of the large myelinated fibers is much more indicative of CIDP (27, 28, 32).

Recent onset DM, relatively well controlled – Diabetic polyneuropathy usually develops on a background of long-standing chronic hyperglycemia and the associated metabolic derangement (4). Recent onset, relatively well-controlled DM is only rarely accompanied by progressive debilitating peripheral neuropathy.

When the later is present, it is more likely to be due to CIDP.

Electrophysiological parameters supportive of CIDP

The electrophysiological workup can sometimes enable to distinguish between a primary demyelinating damage and peripheral disorders that mainly involve the axons. The first is characteristic of the inflammatory, immune-mediated conditions such as CIDP, and the later has a much higher probability of being associated with diabetic neuropathy. When examining the various sets of electrophysiological criteria elaborated over the past three decades for the diagnosis of CIDP, it is evident that the establishment of a strict set of criteria has been challenging, mainly because demyelinating and axonal types of injury often coexist and overlap (18). Most of the proposed electrophysiological criteria of CIDP consider the presence of reduced conduction velocities, prolonged distal latencies, prolonged F wave latencies, and conduction blocks and/or temporal dispersion as indicators of primary demyelination. Thus, criteria repeatedly require their presence to establish the diagnosis of CIDP (15–18, 29, 30).

However, none of these parameters is a specific discriminator between CIDP and diabetic neuropathy. In fact, nerve conduction studies of patients with typical diabetic sensorimotor polyneuropathy often reveal reduced motor nerve conduction velocities and prolonged F waves latencies (4, 31, 32). Abnormal conduction studies in at least two nerves might be considered sufficient to fulfill the diagnostic criteria of CIDP (21, 24), but may also be found in other conditions, such as multifocal pressure neuropathies or radiculopathies (33). As both these conditions have been reported to be more frequent in diabetic polyneuropathy than in the general population, these findings may simply reflect the underlying diabetic neuropathy and not necessarily indicate a demyelinating process of immune-mediated pathogenesis (33). Still, the presence of at least two of the above mentioned parameters is a more significant indicator of a primarily demyelinating process (34).

It was also proposed that the duration of the distal CMAP is a parameter favoring demyelination (19).

All these parameters were incorporated in our scheme, as they seem more specific for primary demyelinating conditions rather than diffuse nerve injuries caused by metabolic states (19, 20). On the other hand, the presence of significant reduction in motor and sensory action potential amplitudes, with normal or slightly reduced conduction velocities, is the main distinguishing characteristic of primary axonal damage and therefore considered contradictive for the purpose of our score.

Ancillary studies

In CIDP, the cerebrospinal fluid contains elevated proteins and is acellular, but this finding is

Table 2 Contradictive parameters of CIDP

Parameter	Score
1. Clinical	
A. Slowly progressive course	−2
B. Predominant sensory symptoms suggestive of small fiber involvement	−2
C. Cranial nerves (except facial) and\or autonomic involvement	−3
D. Preserved DTRs	−3
2. Electrophysiology	
A. Reduced CMAP amplitude disproportionate to motor conduction velocities	−3
3. Histology	
A. Axonal degeneration without evidence of demyelination	−2
B. Evidence of other causes of neuropathies that may mimic CIDP (vasculitis, sarcoidosis, amyloidosis)	−3

not specific and may be present also in DM (35). Nevertheless, CSF protein levels in patients with diabetes are generally mildly increased and do not reach levels higher than 100 mg/dl. We therefore added this parameter as contributing to CIDP diagnosis when CSF protein levels are above twice the upper limit of normal levels.

Dysimmune conditions with immunogenetic background have a tendency to co-occur. There are also serological parameters which suggest an abnormal immunological response in CIDP. Such abnormalities, though not specific, may also be indicators for CIDP.

Additional evidence

Histology – This is reserved for selected cases of suspected CIDP. Sural nerve biopsy consistent with demyelination and/or remyelination by electron microscopy or teased fiber analysis and inflammation by H & E stain, *per se*, is sufficient to establish definite diagnosis of CIDP.

Therapy – Favorable response to immunomodulatory and/or immunosuppressive therapy can be evaluated on clinical grounds (arrest of disease progression, improvement of sensory symptoms, and objective regaining of muscle strength), electrophysiological improvement, and reduction of CSF protein levels. When present, it establishes definite diagnosis of CIDP.

Contradictive parameters – There are several features consistent with diabetic neuropathy. These include a chronic relentless course of more than a year, involving predominantly small fibers sensory disturbances, autonomic nervous system dysfunction, cranial nerve involvement, and preserved deep tendon reflexes (DTRs) (Table 2). Likewise, reduced cMAP potentials and histology suggesting of primary axonal damage are more indicative of diabetic neuropathy.

How to use this scale? – By applying points to each of the clinical, electrophysiological, and biochemical parameters, a total score that ranges between

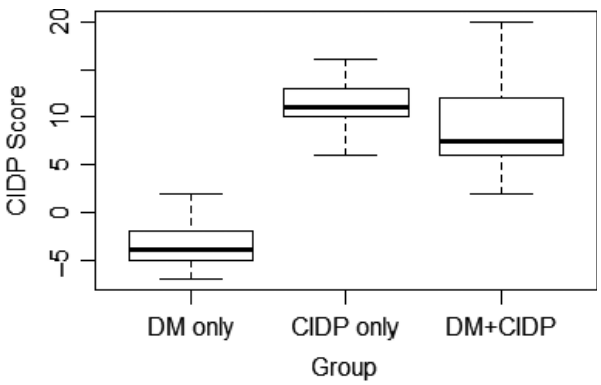

Figure 1. CIDP mean score among patients with DM, CIDP and DM with CIDP. No significant difference in the CIDP score was demonstrated between diabetic patients with CIDP and non-diabetic patients with CIDP. A Fisher's least significant difference (LSD) *post-hoc* test revealed lower score among diabetic patients without CIDP compared to diabetic patients with CIDP and compared to non-diabetic patients with CIDP.

Table 3 Main characteristics of the study population

Variable	ONLY DM (n = 27)	ONLY CIDP (n = 18)	DM+CIDP (n = 12)	Total (n = 57)
Age (Mean ± SD)	70.11 ± 10.89	58.39 ± 18.26	65.00 ± 10.39	65.33 ± 14.28
Male Gender (%)	59.3%	72.2%	66.7%	64.9%
CIDP SCORE				
Mean ± SD	−3.59 ± 2.70	11.17 ± 2.38	9.08 ± 4.80	3.74 ± 7.71
Range	(−7) to 2	6–16	2–20	(−7) to 20

One-way analysis of variance between the three study groups demonstrates a statistically significant effect of age [F(2,56) = 4.037, P = 0.023], with lower age among patients with CIDP compared to patients with diabetic polyneuropathy (P = 0.006). No significant differences in age are evident between patients with DM–CIDP and the two other groups (only DM and only CIDP). In addition, there are no significant differences in gender distribution between the three comparison groups.

minus 18 and plus 24 points can be assembled for each patient in whom a diagnosis of CIDP is considered.

According to the total score, we define 4 levels of certainty regarding the diagnosis of CIDP in DM:

1. Above 11 points: definite.
2. Between 5 and 10 points: probable.
3. Between 2 and 4 points: possible.
4. Below 2 points: unlikely.

For patients who meet a total score compatible with a diagnosis of definite or probable CIDP, we recommend a therapeutic trial with one of the first-line CIDP immunotherapies. For those with possible CIDP, it is appropriate to seek further reinforcing demyelinating histological features on sural nerve biopsy and consider immunotherapy accordingly. At last, those patients who have a score of <2 points most likely do not have an immune-mediated neuropathy.

Statistical analysis and results:

We analyzed the validity of this diagnostic toll in 3 different patient populations: (i) Patients with diabetes with peripheral neuropathy, (ii) Patients with CIDP without DM, and (iii) Patients with diabetes with CIDP.

Statistical analysis was carried out using SPSS-20 software. Proportions and means were used to describe the main characteristics of the study populations (gender and age, respectively). One-way between-subjects analysis of variance (ANOVA) was used to evaluate differences in CIDP scores between the three study groups.

Table 3 illustrates the main clinical characteristics of the three study populations. While patients with diabetic polyneuropathy tend to have an older age at diagnosis, there is not a statistically significant effect of age among patients with CIDP compared to patients with DM and CIDP. No significant differences were evident in gender distribution between the three comparison groups. The scores of patients with diabetes without CIDP ranged from −7 to 2, while those of patients with DM–CIDP ranged from 2 to 20. The scores range of non-diabetic patients with CIDP was similar to those of patients with DM–CIDP and ranged from 6 to 16.

As demonstrated in Fig. 1, the mean score of patients with DM–CIDP was 9.083, while the score of patients with CIDP was 11.16 and that of patients with diabetic polyneuropathy was −3.59. These results validate our approach and shows that this diagnostic tool is able to discriminate between patients with diabetes with overlapping CIDP.

Discussion

Unraveling CIDP in patients with DM is challenging, as several features of diabetic polyneuropathy may overlap with CIDP. However, as previously discussed, the diagnosis of CIDP in patients with diabetes has significant clinical and therapeutic implications.

The tool described here combines clinical and electrophysiological parameters that enable structured and logical approach and can quantitatively provide the likelihood of diagnosis.

We validated our approach on a relatively small number of patients, and further studies on larger cohorts are required to confirm our findings

Once established, along with its potential clinical utility, it may also enable to recruit patients to clinical studies and therapeutic trials and may pave the way to elucidation of the pathogenesis of CIDP in the context of DM.

Acknowledgment

None.

Conflict of interest

None.

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