LETTER TO THE EDITOR

Management of neuropathy musculoskeletal deficits is much more than general global exercises: physiotherapy-based programs for diabetes long-term complications

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TO THE EDITOR: We read with interest the Physiology in Medicine review by Allen et al. (1) on the role of the neuromuscular consequences of diabetic neuropathy (DPN). Three main points in the review deserve attention and should be further discussed.

First, DPN-related motor unit (MU) loss is suggested to be linked with the duration and severity of DPN. However, the literature shows that changes in motor function are observed in the early onset of diabetes (10, 12), and functional alterations are present despite the absence of structural alterations (7). A reduction in motor conduction velocity is observed even before the loss of muscle strength or early DPN sensory symptoms, suggesting that the progression of diabetes is coincident in neuromuscular and sensory systems (3, 5). Changes in electrical muscle activity during gait (11) and in isometric force (10) were described in diabetics without DPN, and in early DPN stages, proving that motor involvements are not related to DPN duration or severity, as stated in the review.

Second, the reason given for the decrease in skeletal muscle contractile properties deserves attention and revision. Authors affirm a preferential loss of type II MU; however, biopsy studies demonstrate a preferential loss of type I fibers in diabetic subjects (4, 6), which was corroborated with experimental animal models (2). Also, scientific evidence indicates a shift in fiber type proportion, confirming the maintenance of more type II fibers in diabetics (6). Corroborating these findings, studies of muscle fiber conduction velocity in isometric tasks point to a preferential loss of type I fibers in diabetic and DPN patients (3). Therefore, the statement that DPN causes a preferential loss of type II MU cannot be made. The supposedly late impairment of the motor system in the course of DPN and the maintenance of type I fibers seem inconsistent with the literature, and a review paper, such as this (1), should have drawn attention to this fact, reinforcing the need for further investigations.

Lastly, the review (1) discusses that exercise therapy appears to be a promising preventive strategy for patients with DPN. However, the authors included in their list only general exercise programs including aerobics, strength, or balance training for global musculatures focusing only benefiting glycemic control. The authors did not mention any of the published clinical trials that focus on improving patients’ functionality (8). The trials have proven that specific exercises targeting the severe musculoskeletal deficits of DPN can recover gait functionality, increase joint mobility, decrease joint stiffness, and decrease peak pressures during gait (8). Sartor et al. (9) presented therapeutic exercises for foot-ankle as a successful strategy to prevent tissue breakdown and to enable patients, for as long as possible, to have the residual biomechanical capability of interacting safely with the environment while walking. Thus, aside from cardiovascular and general strength training, specific therapeutic approaches have been shown to be effective for DPN management. We encourage the scientific community to carry out clinical trials to improve the level of evidence of foot-ankle–specific exercises and consequently strengthen the role of exercise therapy in the prevention and management of the long-term diabetes complications.

DISCLOSURES

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AUTHOR CONTRIBUTIONS

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REFERENCES


