(2), the latter was the main bladder abnormality in our patient, presumably reflecting the brainstem atrophy. Previously, bowel dysfunction has only rarely been documented in WFS (3,4). In our case, loss of SPRCs and sphincter weakness were the main bowel abnormalities. The SPRCs are likely to reflect the intrinsic neuronal activities of the pacemaker cells in the myenteric plexus, which can be damaged in peripheral neuropathies that involve small fibers. Sphincter tone is maintained by the extrinsic somatic nerve for the external sphincter and sympathetic nerve for the internal sphincter, respectively, which can also be damaged in peripheral neuropathies. These bowel dysfunctions need specific management to maximize the quality of life in patients with WFS.

ZHI LIU, MD
RYUJI SAKAKIBARA, MD
TOMOYUKI UCHIYAMA, MD
TATSUYA YAMAMOTO, MD
TAKASHI ITO, MD
SHOICHI ITO, MD
YUSUKE AWA, MD
TAKEO ODADA, MD
TAKETO YAMAGUCHI, MD
TAKAMICHI HATTORI, MD

From the 1Department of Neurology, Chiba University, Chiba, Japan, the 2Department of Urology, Chiba University, Chiba, Japan, and the 3Department of Gastroenterology, Chiba University, Chiba, Japan. Address correspondence to Ryuji Sakakibara, MD, Neurology Department, Chiba University, 1-8-1 Inohana Chuo-ku, Chiba 260-8670, Japan. E-mail: sakakibara@faculty.chiba-u.jp.

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References


Objective Evidence for the Reversibility of Nerve Injury in Diabetic Neuropathic Cachexia

Diabetic neuropathic cachexia is an acute complication of diabetes marked by such extreme pain and weight loss that, although exceptionally rare, it imparts major challenges in management and diagnosis. Little is known of the fundamental pathophysiologic features of the peripheral nervous system in this condition; for example, the symptomatic resolution that is classically observed may arise from either complete destruction of pain-transmitting nerve fibers or from their repair. To reconcile this issue, we report the first case study to our knowledge that the nerve dysfunction is reversible. A 36-year-old woman presented with subacute hyperglycemic symptoms. Soon after institution of insulin therapy and the decline of HbA1c from 14.9 to 5.5%, she developed severe lancinating pain and profound weight loss associated with anorexia, amenorrhea, insomnia, and dehydration. On examination, alodinia was so pronounced that a light touch to her shoulder would cause her to weep. Profound loss of subcutaneous adipose tissue and loss of muscle bulk was evident, such that her weight had decreased from a baseline of 58.3 to 41.8 kg (corresponding to a decrease in BMI from 21 to 15.7 kg/m²). Pain, temperature, and light touch sensation were abnormal in the hands and feet.

Blood count, chemistries, and thyroid and cortisol levels were normal. Further tests for malignancy and malabsorption, including serum immunoelectrophoresis, computed tomography, bone scan, and endoscopy with biopsies, were normal. Titer for antinuclear antibodies and viral etiologies were negative. Nerve conduction studies demonstrated impaired conduction velocity (indicative of impaired myelin sheath function) and impaired amplitude potentials (indicative of impaired nerve axon function) of both sensory and motor nerves.

Hydration, oral nutritional support, and opiate therapy were provided during an 8-week hospitalization. She subsequently received symptomatic therapy with amitriptyline and gabapentin; a year later these analgesic therapies were discontinued, and her weight had returned to baseline. The aberrations in nerve function had normalized; most dramatic was the improvement in nerve axon function, represented by a doubling of amplitude potentials. For example, in the sural nerve the conduction velocity improved by 30% (from 36 to 47 m/s) and the sensory amplitude potentials doubled (from 3.4 to 7.5 μV) from baseline. Similar changes were seen in the median and peroneal nerves for these parameters and also for F-wave latencies and vibration perception thresholds.

The profound weight loss, the symmetrical sensorimotor polyneuropathy associated with dramatic painful paresthesias devoid of weakness, the temporal relation with insulin therapy, and the chronic course are in complete accordance with the diagnosis of diabetic neuropathic cachexia (1). This report emphasizes the need for vigilant symptomatic therapy of diabetic neuropathic cachexia while expediting investigations for eliminating alternate causes. Unique to this report, however, is the objective finding of nerve dysfunction that was dramatically reversed after symptomatic recovery; it supports the proposed hypothesis that paradoxical hypoxic injury occurs at the initiation of insulin therapy (2,3). This case fundamentally suggests that the reversal of painful symptoms is associated with repair of functional aberrations in nerve fibers induced by hypoxia rather than their irreversible ischemic destruction.

JASPREET GREWAL, MD
VERA BRIL, MD
GARY LEWIS, MD
BRUCE A. PERKINS, MD, MPH

From the 1Division of Neurology, Department of Medicine, University of Toronto, Toronto, Canada; and the 2Division of Endocrinology and Metabolism, Department of Medicine, University of Toronto, Toronto, Canada.

Address correspondence to Bruce A. Perkins, MD, MPH, FRCPC(C), Endocrinology and Metabolism, Assistant Professor, University of Toronto, Staff Physician, University Health Network, Toronto General Hospital, 200 Elizabeth St., Room EN-12-217, Toronto, Ontario, Canada M5G 2C4. E-mail: bruce.perkins@uhn.on.ca.

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References

Response to Alfenas and Mattes

In their recent article, Alfenas and Mattes (1) conclude that the glycemic index values of individual foods do not predict glycemic response to mixed meals, nor influence measures of hunger. Because the observed glycemic response did not differ between diets, the lack of effect on appetite is not surprising. Thus, the potentially important aspect of the effect on appetite is not studied. Hence, their conclusion merits study. To advance the discussion of chronic ad libitum consumption of mixed meals (1), this conclusion is unnecessary.

Michael A. Wahl, MD, PhD
University of California, Irvine
Irvine, California

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