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Hyperbaric oxygen therapy in diabetic patients — comments on the paper by Karadurmus et al.

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To the Editor,

We read with great interest the article by Karadurmus et al. in which the effects of hyperbaric oxygen therapy on glycaemic control, inflammation markers and atherosclerosis were investigated in diabetic patients with foot ulceration [1].

We applaud their efforts to gain insight into the effects of hyperbaric oxygen therapy in diabetic patients. Hyperbaric oxygen therapy is an evolving treatment modality, and further studies are needed to better understand its beneficial effects.

However, we think that the authors should have provided more information on patients' wound characteristics and medical treatments to better interpret the changes observed in serum markers of glycaemic control, inflammation, and atherosclerosis.

Karadurmus et al. measured serum levels of high sensitive C-reactive protein (hs-CRP) and white blood cell (WBC) count to evaluate inflammatory processes. Both hs-CRP and WBC count were found significantly reduced after six weeks of treatment. The authors concluded that inflammatory markers were improved in patients receiving hyperbaric oxygen therapy, but they did not discuss this issue.

Our clinical experience, and many publications, suggest that infection develops in the majority of diabetic foot ulcers [2]. Moreover, 53.6% of the patients in this study had a 'Wagner grade 3' ulcer, which is defined as a deep ulcer with abscess or osteomyelitis [3]. In addition, the authors reported that mean initial WBC count (a well-known infection marker) was $11.2 \pm 3.0 \times 103/\mu L$, which is above the normal range ($4.0–10.0 \times 103/\mu L$). Taken together, it is clear that a significant number of the patients in this study had a wound infection and should have received antibiotic treatment as a key component of diabetic foot management.

We think that the improvements in inflammatory markers may be related to the resolution of infection with antibiotic treatment. In order to address this issue, Karadurmus et al. should have either excluded patients with infected ulcers from their study, or provided information on wound infections and antibiotic treatments of the patients. The authors also suggest that hyperbaric oxygen therapy, in itself, improves glycaemic control, which was assessed by fasting blood glucose (FBG), haemoglobin A_{1c} (HbA_{1c}), and homeostatic model of insulin resistance (HOMA-IR). The authors stated that the patients received only insulin treatment for glycaemic control throughout the six weeks study period, but unfortunately they failed to provide information as to whether the insulin doses changed during the study. The patients had poor glycaemic control, as can be understood from their high FBG (152 ± 37 mg/dL) and HbA_{1c} ($9.1 \pm 1.3\%$) values.

Diabetic foot infection is known to worsen glycaemic control [4, 5] and total daily insulin dose is usually increased to maintain adequate glucose levels. It is common clinical practice that diabetic patients with severe foot lesions are hospitalised for parenteral antibiotic treatments and strict metabolic control. Although this issue was not addressed in the paper, strict metabolic control, including an increase in total daily insulin dose, has probably had additional benefits in terms of glycaemic control.

In conclusion, it is hard to attribute all of the improvements obtained in the study solely to hyperbaric

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oxygen therapy. As outlined above, anti-microbial treatment and increased insulin dosage might have contributed to the changes reported in biochemical markers.

To truly determine whether glycaemic control, inflammation and atherosclerosis markers benefit from hyperbaric oxygen therapy, a well-matched control group not receiving hyperbaric oxygen therapy is mandatory.

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