A 44-year-old woman was hospitalized with syncope involving a serious fall. Twenty-eight years earlier, she had undergone cervical radiotherapy for Hodgkin’s lymphoma. For 6 months, she had presented numerous episodes of syncope, always during daytime after getting up and all preceded by significant clinical vasoplegia. On examination, her blood pressure was 112/77 mmHg lying down with a regular pulse of 62/minute and no orthostatic hypotension. Massage of the carotid sinuses did not cause bradycardia but massage of the left carotid sinus, just before the patient got up, led to severe vasoplegia with generalized redness, a pulse that could not be taken and syncope followed by a sudden fall. Blood pressure and heart rate values during this period are shown in Fig. 1. Syncope was reproduced in the same way by rotating the head to the left. The electrocardiogram, 24-hour rhythm recording, cardiac ultrasound and cerebral magnetic resonance imaging were normal. Owing to the cervical antecedents and clinical history, we decided to perform a left carotid glomectomy. Surgery went ahead without any complications and the anatomopathological examination showed a normal carotid body. Since the glomectomy, and after a 4-year follow-up period, the patient has been totally asymptomatic.

The cells of the carotid body, a small vasculonervous formation containing chemoreceptors, are essential to cardiovascular and respiratory homeostasis [1]. Although these cells do not directly control the baroreflex, their proximity to the carotid sinus explains why a carotid body tumour compressing the sinus leads to a feeling of faintness and even syncope due to sinus hypersensitivity. This carotid sinus hypersensitivity is common in patients with syncope and falls, particularly in elderly subjects [2,3]. In 1933, the first treatment for carotid sinus hypersensitivity was denervation of the sinus [4]. In the 1990s, a team suggested performing a glomectomy in combination with a presinusoidal lymphadenectomy [5]. In our patient, the presence of syncope could have indicated carotid

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sinus compression by an adenopathy (history of Hodgkin’s lymphoma) or some other tumour [6]. Another possible aetiology was compression of the carotid sinus by osteosynthetic material. However, imaging and perioperative explorations did not support these hypotheses. We therefore retained the hypothesis of major vasoplegia with a non-tumoural anomaly of the left carotid body. This led us to suggest a carotid glomectomy that allowed the total recovery of the patient. The normality of the anatomopathological examination did not suggest anomaly of carotid body innervation or severe carotid sinus hypersensitivity. In any case, this functional anomaly was cured by surgical nervous deafferentation.

In view of our observation, a glomectomy can be considered, even in the absence of a tumour, when faced with incapacitating carotid body hypersensitivity with significant syncopal vasoplegia.

**Disclosure of interest**

The authors declare that they have no conflicts of interest concerning this article.

**References**


