Acute Cerebrovascular Accident after Cisplatin Treatment in a Patient Taking Letrozole

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We describe a patient with left homonymous hemianopsia following the treatment with small cell lung carcinoma (SCLC) with cisplatin while also on letrozole.

Key Words
Vascular thrombotic events · Cerebrovascular accident · Cancer · Chemotherapy

Abstract
Vascular thrombotic events are common in patients with cancer and chemotherapy is considered a contributing factor. Venous thrombotic events are more common than arterial ones which are less documented. In this report, we describe a patient with right homonymous hemianopsia following treatment with cisplatin for small cell lung carcinoma while also taking letrozole. A brief review of the literature on arterial thrombotic events after chemotherapy follows.

Introduction
Vascular thrombotic events are common in patients with cancer. The hyperthrombotic state that accompanies malignancies was reported as long ago as the 19th century by Trousseau [1]. Chemotherapy can also be a risk factor for vascular events. Twenty-five years ago, Goldhirsch et al. [2] reported an acute cerebrovascular accident (CVA) in a patient receiving a cisplatin-based treatment. Other case reports followed [3–5].
This lesion was consistent with a thrombotic CVA. The lesion was not present in the brain MRI performed for staging 1 month before (fig. 1a). Doppler ultrasonography of carotids was normal.

Letrozole was stopped. Treatment with aspirin was started and the planned chemotherapy was continued every 3 weeks.

The neurological deficit remained stable and the patient had a partial response at the evaluation after 3 cycles of cisplatin/etoposide. MRI 2 months after the event showed a slight improvement of the left occipital lesion (fig. 1c). She received 2 more cycles of chemotherapy. No new thromboembolic events (TEs) happened until the patient died from progressive disease 10 months after the event.

**Discussion**

TEs occur frequently in cancer patients and constitute the second cause of mortality after cancer per se in these patients [6]. Conversely, about one fifth of patients with a venous thromboembolism have active cancer [7]. The mechanisms involved in the pathogenesis of TEs include aberrant activation of the coagulation cascade, defects in blood flow due to local tumor growth and the frequent presence of foreign bodies such as indwelling catheters in these patients and immobility due to cancer or surgery [6]. Furthermore, cancer treatment such as chemotherapy is an established risk factor for TEs in cancer patients in a variety of malignancies and many different treatment regimens. TEs are divided into venous (deep vein thrombosis and pulmonary embolism) and arterial (peripheral artery and cerebrovascular thromboembolism and myocardial infarction).

Several cases of ischemic cerebrovascular complications have been reported associated with the use of cisplatin-based chemotherapy.

A study that prospectively evaluated the incidence of major vascular events in 108 patients with non-SCLC receiving cisplatin and gemcitabine concluded that chemotherapy is a powerful risk factor. In this study, 10 of 22 recorded events were arterial including 1 patient who had an ischemic stroke [8]. Vascular events were detected between 4 and 234 days after the start of chemotherapy. The time to the first arterial event seemed to be shorter than the time to the first venous event (median 35 vs. 61.5 days) but it remains unclear if this observed numerical difference represents a true difference in the delay of these events, because it was not statistically significant. The role of gemcitabine as a contributing agent cannot be excluded [9]. A large retrospective analysis of 932 patients treated with cisplatin-based chemotherapy for various malignancies at a single institution confirms the high incidence of TEs in these patients. This analysis showed an incidence of 18.1% (169 patients) during or up to 4 weeks after chemotherapy. There were 18 arterial events, 10 of which were CVAs. Factors identified by multivariate analysis to increase the risk of TEs were advanced age, lower Karnofsky Performance Status score, the presence of a central venous catheter and higher Khorana score [10].

In another study of 179 patients with germ cell cancers receiving first-line platinum-based chemotherapy, 15 (8.4%) developed a TE; 3 of these TEs were arterial including 2 cerebral ischemic strokes [11].

Other case reports of CVAs in patients receiving cisplatin-based chemotherapy exist in the literature with various presentations. A patient with testicular cancer receiving cisplatin/vinblastine/bleomycin treatment was found to have left homonymous hemianopsia with encephalop-
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References


