Bilateral paclitaxel-induced vocal cord paralysis

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Summary

We report the first case of a bilateral paclitaxelinduced vocal cord paralysis. The patient suffered from orthostatic hypotension, hoarseness and problems with swallowing, before coming to the emergency unit because of a progressive shortness of breath. Vocal cord paralysis is a rare complication of chemotherapy, usually unilateral and seen mostly in patients treated with vinca alkaloids. It should be considered in patients with hoarseness and swallowing difficulties and treated with antineoplastic agents interfering with the microtubules. (BJMO 2009;Vol 3;6:261-262)

Introduction

Vocal cord paralysis (VCP) is usually caused by trauma, malignancies, myasthenia gravis, and surgery such as thyroidectomy.¹ Chemotherapy as a cause for VCP is rare, and in most cases unilateral.² This oncocase reports the first case of a paclitaxel induced bilateral VCP. In addition, the possible pathofysiology of this life-threatening complication is discussed.

Patient history

A 52-years-old female had been diagnosed with a grade three, triple negative invasive ductal carcinoma of the right breast three years earlier. She had been treated with breast conserving treatment and chemotherapy (adriamycin 60 mg/m² - cyclophosphamide 600 mg/m² every three weeks for six cycles), with a total dose of 720 mg adriamycin. After lymph and bone metastasis were found, she was radiated (2 x 8 Gy) on the cervical-thoracal spine. In addition, treatment with paclitaxel (90 mg/m² day 1, 8 and 15) and bevacizumab (10 mg/kg day 1 and 15) every four weeks, was initiated. After the second cycle, the patient started to suffer from orthostatic hypotension, hoarseness and problems with swallowing, which were attributed to the local effects of radiotherapy. Two weeks later she came to the emergency unit because of a progressive shortness of breath. Physical examination detected a moderately ill woman with a progressively, inspiratory stridor. Laryngoscopy revealed a bilateral paralysis of the vocal cords, after which she was intubated. Physical examination did not reveal disease progression. Standard laboratory tests were normal. Ca15.3 had declined from 446 kU/l before the latest chemotherapy, to 97.9 kU/l. MRI and CT scans of the chest excluded mediastinal localisation of the tumour. Four weeks after undergoing permanent tracheostomy, she died at home due to an internal bleeding.

Discussion

Neuropathy is a common complication of treatment with chemotherapy. Besides polyneuropathy, mononeuropathies (such as peroneal and femoral neuropathy) and cranial nerve palsies (diplopia, facial nerve palsy, VCP, ophthalmoplegia and sensorineural hearing loss) can be seen.^{3,6} VCP from laryngeal neuropathy is a rare complication of chemotherapy. So far, only 32 cases have been reported in the literature, of whom seven were bilateral.^{1,2,5-8} All had received vinca alkaloids, except for one patient who had been treated with cisplatin and etoposide.¹ One patient recovered from a paclitaxel-induced VCP after which she had a relapse when receiving vinorelbine.⁶ In all patients, the VCP resolved gradually in

Key messages for clinical practice

- 1. Vocal cord paralysis should be considered in patients with hoarseness and swallowing difficulties, treated with chemotherapy.
- Chemotherapy induced vocal cord paralysis is almost exclusively seen in patients treated with vinca alkaloids.

a course of one to two months.

Taxanes block cells in the late G2 mitotic phase of cell cycle by polymerisation and stabilisation of microtubules. This results in an inhibition of fast axonal transport, causing peripheral neurological symptoms.³ The anti-tumour affect of vinca alkaloids is also related to interference of the microtubules. It binds to the dimeric tubulins, which prevents its polymerisation from soluble dimmers into microtubules.⁹

Conclusion

In conclusion, the possibility of chemotherapy induced VCP should be considered in patients with hoarseness and swallowing difficulties, especially for those treated with antineoplastic agents interfering with the microtubules.

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