Pediatric Hematology Oncology Journal 1 (2016) 61–62

Contents lists available at ScienceDirect

Pediatric Hematology Oncology Journal

journal homepage: https://www.elsevier.com/journals/pediatric-hematology-oncology-journal/

Vincristine induced cortical blindness: An alarming but reversible side effect

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ARTICLE INFO

Article history:
Received 21 October 2016
Accepted 2 November 2016
Available online 4 November 2016

Keywords:
Amaurosis
Reversible blindness
Vinca alkaloids
Relapsed Wilms' tumor
Neuropathy

ABSTRACT

Vincristine is one of the commonest chemotherapeutic agents in the practice of pediatric oncology. Although peripheral neuropathy is a dose limiting adverse event, blindness secondary to vincristine is seldom reported. We describe a child with Wilms tumor who developed transient visual loss after administration of vincristine. The child underwent early surgery and vincristine was re-introduced at reduced doses and gradually escalated to full dose while closely monitoring for recurrence. Blindness is a distressing adverse event, and re-exposure to the offending agent involves a conscientious decision.

Vincristine is ubiquitous in the practice of pediatric oncology. Although vincristine induced neuropathy is a well-known dose limiting side effect, blindness is rarely reported [1,2]. Vincristine exposure can potentially lead to transient or permanent blindness by mechanisms including cranial neuropathy, optic nerve atrophy and cortical blindness [2,3]. We describe a child with transient, cortical blindness secondary to vincristine.

A 7-year old boy was diagnosed with left sided Wilms' tumor (WT). He was treated as per the International Society of Pediatric Oncology Wilms' Tumor protocol (SIOP-WT2001) [4]. Twenty-four hours following the first dose of neoadjuvant chemotherapy (NACT) comprising vincristine (1.5 mg/m^2, intravenous bolus) and actinomycin-D, he presented with sudden, complete loss of vision. There was no association with headache, vomiting, altered sensorium or seizures. On examination, patient was normotensive and fundus was normal. Pupillary reflex was intact. Neurological examination revealed no cranial neuropathy, sensory or motor deficits. He was euglycaemic, with normal serum electrolytes. Contrast enhanced computerized tomography of the head was normal. After a duration of 4 h, vision recovered completely. He experienced transient dizziness after the second dose of vincristine, administered after one week. The third vincristine was uneventfully administered at 50% dose, following which he underwent radical nephrectomy. Histopathological examination of the tumor revealed intermediate risk histology. He received 27 weeks of adjuvant chemotherapy (ACT) with vincristine and actinomycin-D as a stage II tumor [4]. He received 50% doses of vincristine in the first 4 weeks of ACT. Thereafter, doses were escalated gradually to achieve full dose by week 11. Two months following completion of therapy, he had a localized relapse. He was successfully salvaged with chemotherapy and local radiotherapy, based on the United Kingdom Children's Cancer and Leukemia Group protocol for relapsed WT [5]. He has been well and disease free for the last 1 year.

Vincristine can cause inhibition of microtubule polymerization, direct axonal injury in the retina, optic nerve ischemia and loss of neuro-synaptic activity [2,3,6]. The risk of neurotoxicity is increased in patients with liver dysfunction, pre-existing neurological disorders and malnutrition [1,7]. A wide range of ocular side effects, such as cranial neuropathy manifesting as ptosis, lagophthalmos and corneal hyperesthesia; optic atrophy; cortical blindness and nyctalopia has been reported in association with vincristine [2,3,6,8–11]. Blindness is an uncommon manifestation of vincristine induced neurotoxicity, described in few case reports and small case series [1,2,8–10]. Cortical blindness is characterized by complete loss of visual sensation, including perception of light; with the preservation of pupillary reflex and eye motility; and a normal retinal examination [1]. Vincristine induced cortical blindness is largely reversible with the discontinuation of the offending

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Peer review under responsibility of Pediatric Hematology Oncology Chapter of Indian Academy of Pediatrics.

http://dx.doi.org/10.1016/j.phoj.2016.11.001

drug [1,9,10]. Blindness secondary to optic atrophy may reverse, albeit to a lesser extent [2,8]. Re-challenge with reduced dose of vincristine is a conscientious decision which has to balance cure with the risk of recurrence of a potentially disabling adverse event. An early nephrectomy was done in the index patient after 3 weeks of NACT instead of the standard 4 weeks. Subsequently, gradually escalated doses of vincristine were administered without any recurrence of visual symptoms. However, the reduced doses may have contributed to relapse in this patient.

It is essential to be perceptive of vincristine induced blindness and withhold vincristine promptly on its occurrence. Re-exposure at reduced dose involves a cautious and individualized decision.

Source of funding

None.

Competing interest

None.

Conflict of interest

Nothing to declare.

Acknowledgements

The inputs for management from Prof. Kathy Pritchard-Jones (Cancer Section, University College London Institute of Child Health, London, UK) are gratefully acknowledged.

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