Drug-induced neuropathy in a hospital setting

Neuropatía inducida por fármacos en un ámbito hospitalario

Sir,

Medication-induced peripheral neuropathy (PN) is an infrequent and potentially reversible complication. Although many drugs may cause neuropathy, it is most frequently caused by anti-neoplasia treatments. This neuropathy is generally axonal, distal and symmetric with a predominance of sensorial manifestations, particularly pain.1,2

This paper reviews the cases of medication-induced PN diagnosed between January, 2005, and December, 2009, at the University Teaching Hospital in Zaragoza, collecting medical records and identifying the patients where PN was medication-induced, department where the patient was admitted and that established the diagnosis, diagnostic tests, treatment and course. For this purpose, a search was carried out on the centre’s computing servers to identify all patients admitted to hospital during that period with a discharge report that included a diagnosis of PN and, through a review of these case files, those in which PN was medication-induced were selected.

Thirty cases of medication-induced PN were collected: 17 males and 13 females aged between 24 and 90 years of age. The department with the largest number of cases was haematology with 12, followed by internal medicine, oncology and digestive apparatus with 4 each, neurology, infective diseases and gynaecology with 3 each. All cases were diagnosed by the staff of the department in which the patients were admitted. In 20 patients, neuropathy was sensory in the form of pain with or without paraesthesias and was mixed in the other 10 cases. In 26 cases, it affected the lower limbs and in 3 the upper limbs, with one case of involvement of all 4 limbs; we have not found any cases of mononeuropathy or involvement of the cranial pairs. The most frequent medication was bortezomib with 12 cases (9 patients with multiple myeloma), other chemotherapy agents caused 13 cases and there was one case caused by atorvastatin, ethambutol, linezolid, isoniazid (these last three in patients with *Mycobacterium* infection) and phenytoin. In 17 cases, the pain was severe, forcing withdrawal of the medication.

If we divide the medication into anti-neoplasia treatments (group 1) and others (group 2), this gives a total of 25 cases versus 5. In the first group, 52% of treatments were suspended and 80% in the second. Symptomatic treatment was begun in 64% of the patients in group 1 and in 60% of those in group 2, with gabapentin being the drug most often used. The diagnosis was established on the basis of the electroneurogram in 20% of the anti-neoplasia cases and in 80% of the others; in the rest of the cases, it was established on the basis of a clinical suspicion and the history of exposure. In total, clinical signs remitted in 44% of the patients in group 1 and in 60% of group 2; in 8 cases, the clinical recovery was only achieved with withdrawal of the medication, while the rest required pharmacological treatment for the pain.

Although a large number of drugs may cause PN, the most frequent are those used in chemotherapy; the low number of diagnoses due to other medications leads us to think that either they have not been recognized as the causal agents or they have not been coded.

The handling of PN varies between the two groups, although the number of cases is too small for conclusions to be reached; the most striking finding is the lower percentage of electroneurograms (ENG) requested in group 1, probably because it is a relatively frequent side effect in patients treated with chemotherapy and the assumption by those responsible of a cause and effect relationship with administration of the treatment; in this sense, it should be mentioned that, at present, peripheral nerve toxicity is one of the most treatment-limiting conditions in oncohaematology. The fact that fewer treatments are withdrawn in group 1 might be due to the severity of the illnesses treated and the lesser availability of alternative medications.

References


M.A. Aibar Arregui a, M.P. Martín Fortea b, K. Laborda Ezquerra a, J. Cuesta Muñoz c

a Servicio de Medicina Interna, Hospital Clínico Universitario Lozano Blesa, Zaragoza, Spain
b Atención Primaria, Hospital Clínico Universitario Lozano Blesa, Zaragoza, Spain
c Servicio de Enfermedades Infecciosas, Hospital Clínico Universitario Lozano Blesa, Zaragoza, Spain

© Corresponding author.

E-mail address: Miguel_a_a_a@hotmail.com (M.A. Aibar Arregui)

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