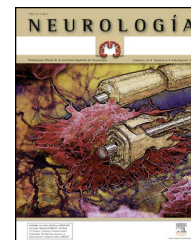




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LETTERS TO THE EDITOR

Neurological syndromes associated with medication use[☆]

Síndromes neurológicos asociados al uso de medicamentos

Dear Editor,

We have read with great interest the recently published study by Álvarez Soria et al. describing neurological adverse events associated with medication use from a patient safety perspective.¹

In recent years, numerous studies have shown that prevalence of adverse drug reactions is high and that many such reactions are potentially preventable.^{2,3} However, most of the available data are gathered from hospital settings and the general population. From our point of view, conducting this type of studies in specific populations or for specific adverse reactions, as in the publication by Álvarez Soria et al., provides more concrete information about adverse drug effects and their causes in specific populations. This serves to make healthcare professionals aware of the importance of detection and prevention, as well as promoting the development of initiatives aimed at improving drug safety.

Published studies on neurological adverse events associated with medication use are scarce. Some years ago, we completed a study⁴ whose aim was to analyse and determine the annual incidence of neurological adverse reactions to medication which required a neurological consultation. We concluded that there is a need for more studies on neurological syndromes caused by medication. We are delighted that this second study by Álvarez Soria et al. has been published, and furthermore, we feel it is appropriate to make readers interested in this topic aware of our own study, since it provides useful additional information, especially regarding possible causes and prevention measures. Additionally, we wished to comment on those findings which seem to be the most divergent between the two studies.

Firstly, it should be noted that the prevalence of adverse reactions is quite low (0.586%), a finding which Álvarez Soria et al. attribute to the study design and its possible biases.

Although the rates are not comparable due to differences in methodology, the annual incidence of neurological adverse reactions was 8.7% in our study; we included only those reactions that were directly responsible for an initial neurological consultation. The low percentage of preventable cases in this new study (21.9%) compared to the percentage reported by our study (91.6%) is also noteworthy. Such a difference in the percentage of preventability could be due to the methodological differences between the studies and to the fact that labelling an adverse reaction as preventable or non-preventable is largely a subjective judgement call.

Regarding the types of neurological syndromes observed, drug-induced parkinsonism was a frequent adverse reaction in both studies. However, the most frequent adverse reaction in our study was medication overuse headache (4.5%), whereas Álvarez Soria et al. found no cases. Since medication overuse headache is common, with a prevalence of about 1% in the general population,⁵ the absence of this neurological syndrome in all of the 17 896 patients assessed by this new study is striking.

Finally, in the study conducted by Álvarez Soria et al., adverse reactions were analysed by drug group instead of by separate active ingredients. Given the study's aim of characterising a particular type of adverse reaction (neurological reactions in this case), we believe the drugs involved should have been analysed separately. If not, researchers may fall victim to the fallacy of assuming that all drugs in the same class have a similar safety profile.⁶ If only one of the drugs is responsible for most of the adverse reactions, it will remain masked by the drug group and the trigger of these adverse reactions will be wrongly attributed to all drugs in the group. Furthermore, and more importantly from our point of view, the study does not clearly express the importance of selecting a specific drug from a group according to its safety profile.

We would like to use this opportunity to stress the need for similar studies, conducted in neurological patients, which will help raise awareness about the important healthcare consequences of drug-induced neurological disorders and promote the implementation of prevention measures.

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[☆] Please cite this article as: Ortín Castaño A, Otero MJ. Síndromes neurológicos asociados al uso de medicamentos. *Neurología*. 2014;29:573–574.

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Lymphocytic meningitis and spinal neurocysticercosis: A case report and literature review[☆]

Meningitis linfocitaria y neurocisticercosis espinal. A propósito de un caso y revisión de la literatura

Dear Editor,

Over the past decades, neurocysticercosis (NCC) has become the most frequent parasitic infection of the central nervous system (CNS) in our setting, as a result of immigration from developing countries.^{1,2}

Neurological manifestations depend on the location of *Taenia solium* larvae and on the immune response they trigger in the host. That response depends on the evolutionary stage of the larva, and it will be minimal during the vesicular stage, reach its peak during the colloidal stage, and decrease as it mineralises during the granular and calcified stages. Co-existence of cysts in different stages is not infrequent.¹ Neurocysticercosis can affect the brain, the spinal cord, or both parts of the CNS.

When cysts are located in the brain parenchyma, their main clinical manifestations are epileptic seizures (more than 70% of the cases),^{1,3} although they can present with any kind of focal neurological signs. Cysts located in the subarachnoid space or in the ventricular system may lead to meningitis, arachnoiditis or hydrocephalus due to CSF-flow obstruction in the subarachnoid space or ventricles.³ Cysts in these 2 locations elicit more severe clinical features and sometimes require surgery.

Although there are several published case series of NCC presenting with signs of acute meningitis,⁴ this presentation is not listed among the most common.¹ CSF analysis usually reveals moderate mononuclear pleocytosis and elevated CSF protein levels; and low CSF glucose lev-

els are detected in 12%-18% of the cases.⁵ The presence of eosinophils in CSF is characteristic, although infrequent (15% at onset). Complementary diagnostic procedures include ELISA for CSF and western blot for blood.¹ Most published cases of NCC presenting with meningitis included neuroimaging scans showing viable cysts, hydrocephalus, leptomeningeal enhancement or disorders other than calcifications.^{1,5,6}

The spinal cord is rarely affected even where the infection is endemic, and reported incidence is 1%-3% of all cases of NCC.^{2,6} Cysticercosis is classified as extradural, subdural, subarachnoid or intramedullary according to its location. Intramedullary NCC (less than 20%) and extradural NCC are rare,⁷ whereas extramedullary subarachnoid NCC is the most frequent type. Cysticerci migrate from the brain to the spinal subarachnoid space through CSF; intracranial subarachnoid NCC will also be present in most cases. The most frequent manifestations are radiculopathies.^{1,6} Isolated spinal involvement is very rare.^{8,9}

We present the case of a 29-year-old Bolivian woman with no relevant history who presented at the emergency department with progressive headache of 72 hours' duration, fever, and nausea. Her health was good otherwise and findings from the neurological examination were normal, with no signs of meningeal irritation.

A cranial CT scan showed multiple scattered cortical-subcortical calcifications measuring up to 2.5 mm and consistent with NCC in the calcified stage. A lumbar puncture (LP) delivered clear CSF with normal pressure, containing 265 white blood cells (76% mononuclear), 73 mg/dL proteins and CSF glucose level of 45 mg/dL (50% of glycaemic level). Doctors considered viral (non-herpetic) lymphocytic meningitis as the first aetiological possibility. The patient was admitted to hospital and symptomatic treatment was administered. Signs and symptoms varied during the first 2 weeks of treatment, so LP was performed two more times. Results from CSF analysis showed improvements in pleocytosis and CSF protein level. However, CSF glucose dropped to less than 20% of glycaemia. Doctors screened for different aetiologies for meningitis with low CSF glucose (carcinomatosis, subarachnoid haemorrhage, sarcoidosis, dermoid cysts, bacterial meningitis, meningeal tuberculosis, fungal meningitis and viral meningitis [choriomeningitis, varicella-zoster virus, cytomegalovirus, herpes simplex virus 1 and 2, human immunodeficiency virus, parotitis]). Results from

[☆] Please cite this article as: Abarrategui Yagüe B, García García ME, Orviz García A, Casas Limón J. Meningitis linfocitaria y neurocisticercosis espinal. A propósito de un caso y revisión de la literatura. *Neurología*. 2014;29:574–576.