technique: headache secondary to pneumocephalus and low CSF pressure headache.[7] The first of these usually manifests immediately after puncture or when the patient resumes a standing position. In our case, the onset of headache 2 days after epidural block was probably triggered by a postural change from lying to standing and caused by an air bubble in the subarachnoid space having migrated to the cranial cavity. Low CSF pressure headache has been reported as a consequence of accidental dural puncture as well as a cause of thunderclap headache, and an orthostatic factor is typically involved. Brain MRI scans in patients with low CSF pressure frequently indicate extradural CSF collection, diffuse meningeal uptake, engorgement of venous structures, pituitary hyperaemia, and downward displacement of the brain.[8] None of these signs were seen in our patient, making it unlikely that intracranial hypotension would be the cause of headache. Our patient did not undergo a lumbar puncture since neuroimaging studies revealed pneumocephalus as a possible cause of thunderclap headache. However, differential diagnosis of all the possible aetiologies of thunderclap headache is absolutely essential to rule out such severe entities as subarachnoid haemorrhage, other intracranial haemorrhages, cerebral venous thrombosis, cervical artery dissection, and reversible cerebral vasoconstriction syndrome. All patients with these headaches should undergo cranial CT and lumbar puncture, plus brain MRI and MRI angiography in selected cases.[9]

Such underlying processes as pneumocephalus and CSF hypovolaemia should be ruled out in patients experiencing thunderclap headache after undergoing an invasive procedure (for example, epidural anaesthesia with the LORA technique). This case and other published cases support using the 'loss of resistance to saline' technique to identify the epidural space. This method has been found to provide better analgesia with fewer adverse effects compared to the LORA technique.

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Myoclonus secondary to use of anti-flu drug☆

Mioclonías secundarias a fármaco antigripal

Dear Editor:

Drug-induced movement disorders are relatively frequent. In fact, a high number of drugs have been found to be linked to myoclonus. We present the case of a patient who developed myoclonus secondary to combined treatment with dextromethorphan and chlorphenamine, 2 widely used drugs. The literature describes one case of myoclonus associated with dextromethorphan use; no cases of myoclonus secondary to chlorphenamine use had been reported to date.

Our patient was a 64-year-old male smoker with no relevant medical history. He was receiving no medications and exercised regularly.

A few days previously, he presented general unease, fever (39 °C), holocranial headache, and nasal congestion. After a pharmacist identified the patient's flu-like symptoms, the patient began taking an anti-flu preparation containing paracetamol, dextromethorphan, chlorphenamine, ascorbic acid, and caffeine citrate.

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After taking the first tablet orally, our patient experienced intense restlessness and involuntary movements of the face and upper limbs which interfered with night-time sleep.

Upon arriving at the emergency department, he had no fever and his vital signs were within normal limits. He denied use of any other drugs or intoxicating substances. The physical examination, including auscultation and examination of the upper respiratory tract, revealed no abnormalities. The emergency blood analysis, chest radiography, and brain CT scan yielded normal results.

A neurological examination revealed sudden-onset short involuntary movements in the upper limbs, present during rest and exacerbating with movement. These movements were accompanied by decreased muscle tone and dropping of both upper limbs; after this, our patient was able to lift them immediately. Furthermore, he reported generalised restlessness and involuntary contraction affecting facial muscles on both sides. These contractions did not disappear with distraction movements. He displayed no alterations in strength or sensitivity and no signs of cranial nerve involvement. Likewise, he showed no signs of meningeal irritation, increased muscle tone, or regressive reflexes.

Suspicion of drug-induced generalised myoclonus led to treatment with intravenous benzodiazepines. After doctors discontinued the anti-flu drug and administered intravenous diazepam (3 doses of 10 mg every 8 hours), symptoms resolved completely and the patient became asymptomatic. He has experienced no recurrences and displayed no other neurological symptoms in the subsequent months. We recommended that he avoid such anti-flu drugs or their derivatives.

Myoclonus belongs to the spectrum of hyperkinetic movement disorders; it is described as sudden-onset quick involuntary jerking movements.[1] It is caused by either muscle contractions (positive myoclonus) or brief lapses of loss of muscle activity (negative myoclonus).

Depending on its various origins within the nervous system, myoclonus can be classified as cortical (multifocal, very brief duration, occurring especially during activity), subcortical (longer duration, occurring both at rest and during activity), or spinal (focal, longer duration, occurring especially at rest).[2]

In our patient, myoclonus seemed to be negative and of subcortical origin. Aside from myoclonus, drug-induced movement disorders include a wide range of symptoms such as dystonia, tremor, drug-induced parkinsonism, dyskinesia, akathisia, and even serotonin syndrome or neuroleptic malignant syndrome.

All of them share a direct temporal relationship between use of a certain drug and symptom onset, and between drug discontinuation and symptom resolution (except for late-onset symptoms).[3]

Drugs responsible for such syndromes have included levodopa, antidepressants, lithium, dopamine agonists, antiepileptics, opioids, antineoplastics, anxiolytics, and antibiotics.

In our patient, the drug included several active ingredients, which raises the question of whether myoclonus was caused by one of the components or rather by the synergistic effect of a combination of them.

Dextromethorphan is a widely used cough suppressant that can either be used alone or in anti-flu drug complexes. It has been linked to myoclonus in the context of renal failure,[4] as well as to serotonin syndrome in more severe conditions[5] and when combined with other drugs (especially serotonin reuptake inhibitors). Immediate discontinuation of the drug is recommended if either condition arises. Multiple drugs, including diazepam, have been suggested as treatment for myoclonus, although no specific indications have been established.

Chlorphenamine is a first-generation antihistamine with central nervous system side effects that included tremor, epileptic seizures, sedation, and somnolence. However, an association with myoclonus has not been described.

In our patient, myoclonus probably was an unexpected likely adverse reaction to the drug (according to the causality criteria for adverse reactions); as such, it was reported to the appropriate pharmacovigilance authority. However, we have no conclusive data on the underlying mechanism.

Although no studies have reported a higher risk of movement disorders in patients with a history of drug-induced myoclonus, it seems reasonable to think that these patients would have a higher risk of recurrences after using that particular drug or its derivatives.

To the best of our knowledge, no similar cases have been described in the literature to date.

Conflicts of interest

The authors have no conflicts of interest to declare. This study has not been presented elsewhere.

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