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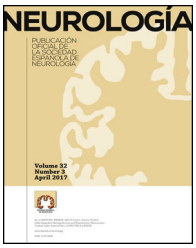

LETTERS TO THE EDITOR

Incidence of Guillain—Barré syndrome during Zika virus outbreak☆

Incidencia del síndrome de Guillain-Barré durante el brote del virus Zika

Dear Editor:

We read with great interest the article ''Incidence of Guillain—Barré syndrome (GBS) at a secondary centre during the 2016 Zika outbreak.''[1] Del Cario Orantes et al. conclude that ''cases of Guillain—Barré syndrome increased during the Zika outbreak, with increases in incidence and the number of cases per month; however, no direct causal relationship could be established between these 2 conditions.''[1] We would like to share some ideas and experiences on this subject. Firstly, the increased incidence of GBS may or may not be related to Zika virus infection. Several possible concurrent problems, such as other infections or vaccination during the study period, may give rise to increased incidence. In our setting in tropical Asia, where Zika virus is also endemic, increased incidence of GBS has not been observed.[2] Most cases of Zika virus infection in our setting are asymptomatic and present no complications. [3] Considering that Zika virus infection may be asymptomatic, the exact calculation of GBS incidence is difficult, and diagnosis of Zika virus infection related to GBS is problematic in any situation.[4]

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Horner syndrome secondary to cephalic paravertebral migration of local anaesthetic☆

Síndrome de Horner por migración paravertebral cefálica del anestésico local

Dear Editor:

Horner syndrome (HS), first described in humans in 1869 and in animals in 1852, is a possible complication associated with different techniques for controlling regional postoperative pain (intradural, epidural, or brachial plexus), with an incidence below 1.8%. We present a case of HS associated with the use of a thoracic paravertebral catheter to control postoperative pain after pulmonary resection by thoracotomy.

Our patient was a 58-year-old female smoker with drug-controlled arterial hypertension and insulin-dependent diabetes mellitus, receiving treatment with immunosuppressants to treat rheumatoid arthritis. She presented symptoms of diarrhoea and asthenia. A chest radiography revealed pulmonary consolidation in the middle lobe, which was diagnosed as pulmonary adenocarcinoma (clinical stage IA) after a CT-guided transthoracic needle biopsy. In the anaesthetic induction phase, a paravertebral catheter was placed to control postoperative pain, which had to be removed due to extravasation of blood through the device. During the procedure, we observed a haematoma fully dis-

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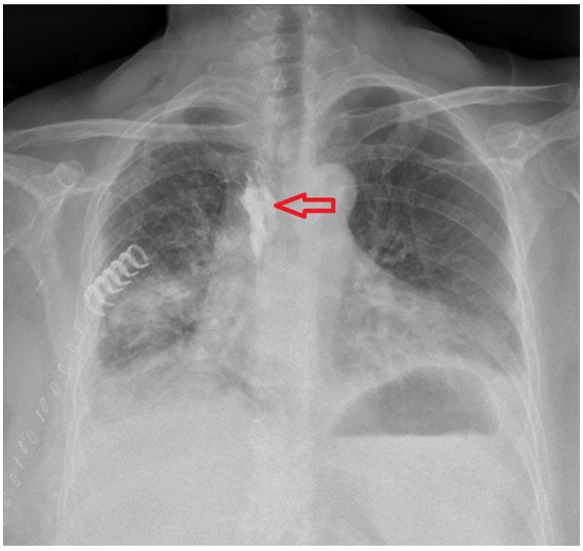

Figure 1 Posteroanterior chest radiography: paravertebral diffusion of iodinated contrast agent at the level of the third to the fifth intercostal space, administered through the catheter (arrow).

secting the paravertebral space and a second paravertebral catheter was inserted (at the level of the fifth intercostal space, advancing cranially to the third). We started continuous perfusion of bupivacaine 0.25% at 5 mL per hour for 24 hours following the procedure. During the clinical progression in the inpatient ward, we observed ptosis with non-reactive pupil which forced us to stop perfusion of local anaesthetic; these symptoms resolved fully in the following hour. We performed a chest radiography with a 5 mL bolus of iodinated contrast agent to verify that the paravertebral catheter was correctly placed (Fig. 1).[1] Six hours later, a single 5 mL bolus of bupivacaine 0.25% was instilled and symptoms reappeared, prompting the definitive discontinuation of the treatment. During the hospital stay, we observed no changes in cardiac rate, nor other accompanying neurological alterations.

Administration of local anaesthetic in the thoracic paravertebral space (including fat, the spinal intercostal nerves, communicating branches, and in the anterior portion, the sympathetic chain) (Fig. 2) may lead to diffusion of the drug to the cervical sympathetic chain, resulting in myosis due to blocking of the pupil dilator muscle; these symptoms are isolated, transient, and usually disappear with no sequelae within hours of local anaesthetic administration.[2] Exceptionally, HS may be accompanied by blockade of preganglionic sympathetic B-fibres[3] at the level of C4 and C5, with alterations in cardiac rate and cardiac contractile strength, or be associated with decreased motility of the corresponding hemidiaphragm due to the adjacent blocking of the phrenic nerve.[4]

The thoracic paravertebral catheter is an effective mechanism of pain control[5] in patients who have undergone thoracotomy, whether for pulmonary neoplasm resection or for any other pulmonary disease requiring the chest cavity to be opened.

Our patient showed cephalic paravertebral migration of a local anaesthetic reaching 5-8 dermatomes, facilitated by

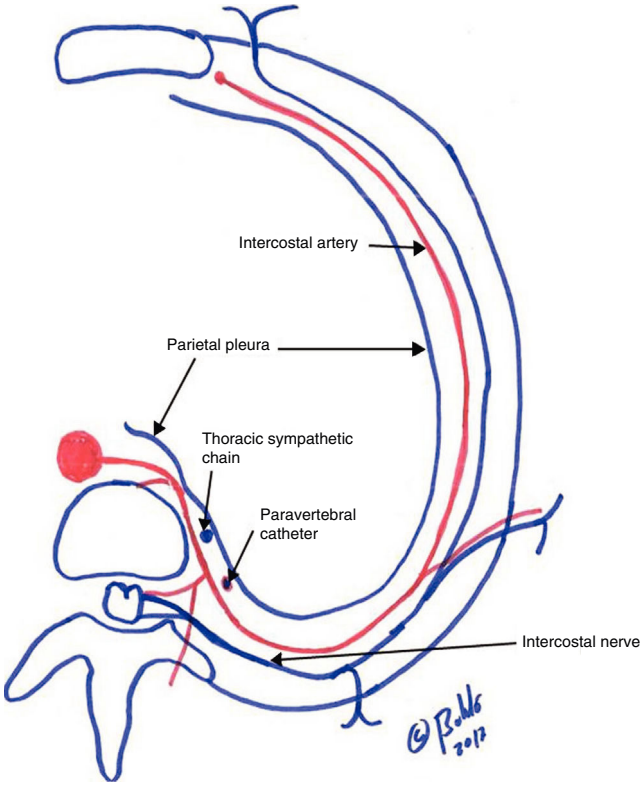

Figure 2 Location of the paravertebral catheter and its anatomical relationship with the sympathetic chain and the intercostal nerve.

the paravertebral haematoma, with involvement of the stellate ganglion leading to HS, but with no other clinical or haemodynamic consequences.

The presence of HS during the postoperative period after insertion of a paravertebral rather than an epidural catheter suggests blocking of sympathetic fibres at a higher thoracic level, which may be accompanied by severe neurological and cardiorespiratory manifestations. Under these circumstances, we should suspend administration of the local anaesthetic through the catheter and closely monitor the patient during the following 24-48 hours.

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Trends in the treatment of cerebral aneurysms: Analysis of a hospital series☆

Tendencias en el tratamiento de los aneurismas cerebrales: análisis de una serie hospitalaria

Dear Editor,

The interesting study by Lago et al.[1] transmits an important message: technological advances do not always represent an absolute improvement, since decreased mortality in the second period is cancelled out by an increase in dependence (mRS >3 in 13.3% of patients in the first stage vs 21.3% in the second stage) for patients with very similar severity and age, which are 2 key factors in determining outcome.[2]

Hospital mortality may not be the best indicator of healthcare quality; comparing therapeutic mortality associated with embolisation or surgery may lead us to mistakenly believe that surgery involves a higher mortality. Although there are very varied examples in the literature, we find no differences between the 2 periods, 1993—97 (no endovascular treatment) and 2008—2012 (when endovascular treatment was fully established).[3] Lagares et al.[4] have pointed to the variability in Spain regarding the use of guidelines for selecting the type of endovascular or surgical treatment; however, the type of treatment chosen did not have an impact on final progression. Furthermore, Horcajadas Almansa et al.[5] report that endovascular treatment is more costly than surgery for ruptured aneurysms, basically due to the price of embolisation materials, the rate of retreatment, and the necessary follow-up.

In specific cases, combined treatments with embolisation and surgery in a hybrid operating room (Fig. 1) may be a good therapeutic option; this way, neurosurgeons, who were previously the only specialists dedicated to aneurysmal subarachnoid haemorrhage (aSAH), may contribute to improving outcomes. Furthermore, the different specialists involved (neurosurgeons, neurologists, neuroradiologists) should be trained in endovascular treatment during residency; failure to provide this training will result in decreases in the number of trained/interested neurosurgeons, with only very difficult/complex cases considered by specialists as ''bad'' cases being treated surgically (Fig. 2). Furthermore, the industry advances rapidly and endeavours to find new materials for every situation, regardless of size, location, and dome-to-neck ratio. However, as with surgery, we should ask ourselves more frequently whether a determined aneurysm should be treated because it is possible or because it is necessary.

I have previously expressed my opinion regarding some organisational aspects of stroke treatment[6] and the obvious benefits of a multidisciplinary approach. However, only the specialist attending the patient in the emergency department, studying the CT scan and multimodal CT scan, can reconstruct images at a workstation and observe the characteristics of the aneurysm and its relation with other anatomical structures in order to decide the best approach for the patient (endovascular, surgical, or combined therapy [Fig. 1]), especially when he/she performs both techniques. Some surgeries can be performed with acceptable results using only CT angiography images, with no need for a preoperative angiography.[7] In light of this, I think it interesting that Lago et al.[1] report that emergency angiography for aSAH has been performed since 2014; however, we can deduce that treatment is not performed at the same time as the emergency diagnostic angiography. The multimodal CT scan used for ischaemic stroke should also be utilised to analyse some anatomical and physiological aspects of aSAH. I fail to understand why in some hospitals with on-call services for ischaemic stroke, the information provided by CT perfusion is not used for aSAH; some aneurysms might be treated more efficiently with surgery.

The consensus document[8] prepared by the societies of neurology, neurosurgery, neuroradiology, and the Spanish Group of Interventional Neuroradiology for training/accreditation in interventional neuroradiology represents a milestone in the history of neurosciences. The next step is probably to prepare another consensus document for vascular neurosurgeons, an endangered specialty, to obtain certified accreditation to perform bypasses, remove arteriovenous malformations, and (if copayment is established) to clip aneurysms, due to the high cost of embolisation materials for families with limited resources. The vascular neurosurgeon may first become an ornamental professional, an expert in clipping and bypass, mentioned on large hospitals' websites. Regarding the consensus document,[8] I would add that specialists trained in endovascular treatment should also be trained to place

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