O4V4M6); the follow-up transcranial Doppler studies were normal in the first two weeks; however, on the 16th day the patient presented transcortical motor aphasia and ideomotor apraxia. We carried out a TCD recording which revealed mean speeds of 165 cm/s compatible with moderate to severe vasospasm of the left middle cerebral artery; a CT perfusion showed an increase in the mean transit time in the left temporoparietal region in comparison with its right counterpart (6.39 versus 2.71 s), without any differences being found in the volume images and cerebral blood flow (fig. 1, Panel A; fig. 2, Panel A).

With a suspected diagnosis of delayed vasospasm, triple Ht therapy (hypertension, haemodilution and hypervolaemia) was instituted, with the neurological clinical signs disappearing in the next 24 h; insonorization of the left middle cerebral artery recorded mean speeds of 52 cm/s and a follow-up CT perfusion allowed observation of a normalization of the mean transit times for blood flow (3.41 left and 3.06 right). We also found a difference (6.39 s versus 3.41 s) when comparing the values of the mean transit time for blood flow in the left temporoparietal area in the first and second studies (fig. 1, Panel B; fig. 2, Panel B).

This confirmed the clinical, radiological and ultrasound disappearance of the vasospasm, so the patient was sent to an ordinary ward and later discharged.

Delayed vasospasm following SAH is an infrequent entity requiring clinical suspicion. Previously, diagnostic confirmation of cerebral vasospasm following an SAH required performance of an angiography,[2] but nowadays a transcranial Doppler study and CT perfusion allow a faster and less bloody diagnosis,[3-5] thus allowing the early start of safe treatment, the main practical application of this paper.

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Diagnosis and monitoring of delayed cerebral ischaemia and cerebral vasospasm in subarachnoid haemorrhage: When to modify the usual guidelines?

Monitorización y diagnóstico de la isquemia cerebral tardía y vasospasmo cerebral en la hemorragia subaracnoidea: ¿cuándo modificar las pautas usuales?

Dear Editor:

We have found the letter to the editor entitled "Diagnosis of delayed cerebral ischaemia and cerebral vasospasm in subarachnoid haemorrhage: How long should they be monitored?" to be of great interest. This document refers to certain aspects of the scheme published on the recommended guidelines for diagnosis of delayed cerebral ischaemia (DCI) and cerebral vasospasm in subarachnoid haemorrhage *(*Neurología. 2010;25:322-30*).*[1] Bearing in mind our discrepancies with respect to the aspects interpreted, we feel it is necessary to clarify these from a different perspective.

Since 1951, when cerebral vasospasm was described angiograms produced during the first 26 days following the bursting of saccular arterial aneurysms, there have been considerable controversies and inconsistencies in the diagnosis of vascular disorders and DCI.[2-4]

In the clinical trials and descriptive studies published to date, inconsistencies have been found in use of the definitions on DCI secondary to aneurysmal subarachnoid haemorrhage. The use of clinical vasospasm, symptomatic vasospasm and vasospasm-related ischaemia suggests that

DCI presents in combination with a radiologically confirmed narrowing of the artery. However, patients with subarachnoid haemorrhage may suffer a clinical impairment attributable to DCI in the absence of radiologically confirmed vasospasm and this ischaemia is probably due to other factors. The opposite situation, where the patient has angiographic vasospasm without developing any clinical manifestations of DCI, also occurs frequently.[1,5]

The typical manifestations of clinical impairment caused by DCI are the development of focal neurological signs (such as aphasia, hemiparesis, hemianopsia, neglect) and/or a reduction in the level of consciousness.[1,5] Evidently, the detection of these typical signs requires frequent meticulous neurological out-patient examinations, particularly during the period when there is a risk of this phenomenon occurring. In addition, it has recently been proposed that these neurological events attributed to DCI must have a duration in excess of one hour in order to reduce false positive diagnoses.[5]

In the neuroscientific literature, one of the critical subjects has been monitoring with transcranial Doppler (TCD) to detect cerebral vasospasm secondary to subarachnoid haemorrhage, in addition to clinical observation at intensive care units (ICU) or stroke units.[6] Signs of vasospasm can be detected through TCD and may indicate the need for intensive management prior to the start of cerebral ischaemia. Among the various guidelines described for the diagnosis and TCD monitoring of vasospasm, the most outstanding are:

- Frequently, and at least once daily during the first days after admission. If high speeds are not detected in the first 7 days, the frequency of the observations can be reduced.[7]
- In the first 24 hours and at least one other ultrasound examination between the 4th and 14th day of evolution of the subarachnoid haemorrhage, except where the first study is effected during this period and is normal. Ultrasound follow-up in the observation ward was performed every 48 hours but if the patient was admitted to the ICU, it was done daily.[8]
- A baseline study as early as possible (day 0) and then continuing once a day between days 3 and 10.[9]
- From the date of admission, with daily recordings until the resolution of the ultrasound or clinical signs of vasospasm, or until day 14 in those patients were not resolved.[10]

These monitoring guidelines essentially correspond to the normal pattern in the evolution of cerebral vasospasm in subarachnoid haemorrhage: it appears after 3-4 days, achieves its maximum incidence and severity between days 6 and 8, and then disappears after 12 to 14 days.[1-4] For this reason, it is desirable to have close monitoring, mainly through clinical and ultrasound examinations, in the first 7 to 10 days of the subarachnoid haemorrhage.[1]

In our review, it is also proposed that, when there is no suspicion of vasospasm/DCI, the frequency of the observations should be reduced until monitoring is withdrawn after 14 to 21 days.[1] This last deadline has been established since at least 4% of the neurological deficits attributable to cerebral vasospasm occur on or after day 13.[2] We agree with the authors of the letter that it its an infrequent form of debut and its diagnosis requires clinical suspicion. Furthermore, perfusion tests are a promising tool for diagnosing DCI, not only in patients with impaired consciousness, but also in patients with a good level of alertness.

Generally speaking, the risk time and the risk factors for vasospasm/DCI determine how long patients with subarachnoid haemorrhage should be monitored for. For example, it is appropriate to apply clinical and TCD observation more carefully and for longer than usual when there are dense collections of blood that give the impression of representing dense clots on the vertical or horizontal planes (grade III of Fisher's scale). Vergouwen et al.[5] add, as the best moment for conducting a follow-up structural neuroimaging test to detect cerebral infarction, once the risk period for DCI has passed and within 6 weeks following the start of the subarachnoid haemorrhage.

To sum up, we feel that research into the clarification of the definitions and characteristics of cerebral vasospasm and DCI must combine clinical monitoring with other diagnostic tests (particularly TCD, digital subtraction angiography and the multiple types of magnetic resonance and computed tomography) during the time interval indicated above. We are definitively grateful to the letter writers for their considerations regarding our review and for their valid case report.

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