autoimmune phenomena.[5,8,9] Finally, another theory would establish an association between IgA deficit and alterations on the part of T cells in the regulation of peripheral tolerance, hence provoking the autoimmune process.[8,14] Regardless of the cause for the association, what is clear is that IgA deficit patients have a higher risk of associated autoimmune diseases.

This is the first case in the literature reporting an association between IgA deficiency and MS. Although there are several hypotheses, the exact role of IgA deficiency in the genesis of the autoimmune phenomenon has not been elucidated to date. Future research will clarify the precise role of this association.

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# Diagnosis of delayed cerebral ischaemia and vasospasm in subarachnoid haemorrhage: How long should they be monitored?

## Diagnóstico de la isquemia cerebral tardía y el vasoespasmo en la hemorragia subaracnoidea: ¿hasta cuándo monitorizar?

*Dear Editor:*

We have reviewed with great interest the article published by Rodríguez-García et al. regarding the diagnosis of delayed cerebral ischaemia and cerebral vasospasm in subarachnoid haemorrhage (SAH), which appeared in the previous issue of your journal (Neurología. 2010;25:322-330).[1]. We believe this to be a document of the greatest importance and extremely timely, as well as being very well developed, and we should like to contribute a few comments.

Rodríguez-García et al. report, on the basis of a review of the literature, that delayed cerebral ischaemia is a major cause of morbidity and mortality among patients presenting SAH and that this ischaemia is secondary to delayed cerebral vasospasm, therefore proposing monitoring, mainly through clinical and ultrasound examinations, for the first 10 days following SAH before reducing and eventually suspending monitoring.

We present here a case of delayed vasospasm and its clinical, radiological and ultrasound correlation, manifested on the 16th day after the SAH.

Female, 52 years old, hypertensive without treatment, presenting a diffuse SAH at basal cisterns, WFNS I, Fisher 3, with a Glasgow score of 14 on admission, without neurological focality. On the same day, a cerebral arteriography was performed and revealed a 3 x 2 mm aneurysm of the right posterior cerebral artery, a 3 x 4 mm aneurysm in the anterior communicating artery and an infundibular dilatation of the right posterior communicating artery. The first aneurysm was treated endovascularly. The following day, the aneurysm in the anterior communicating artery and the dilatation of the right posterior communicating artery were clamped.

The patient progressed favourably, with scant stiffness in the neck and a good level of consciousness (Glasgow 14,

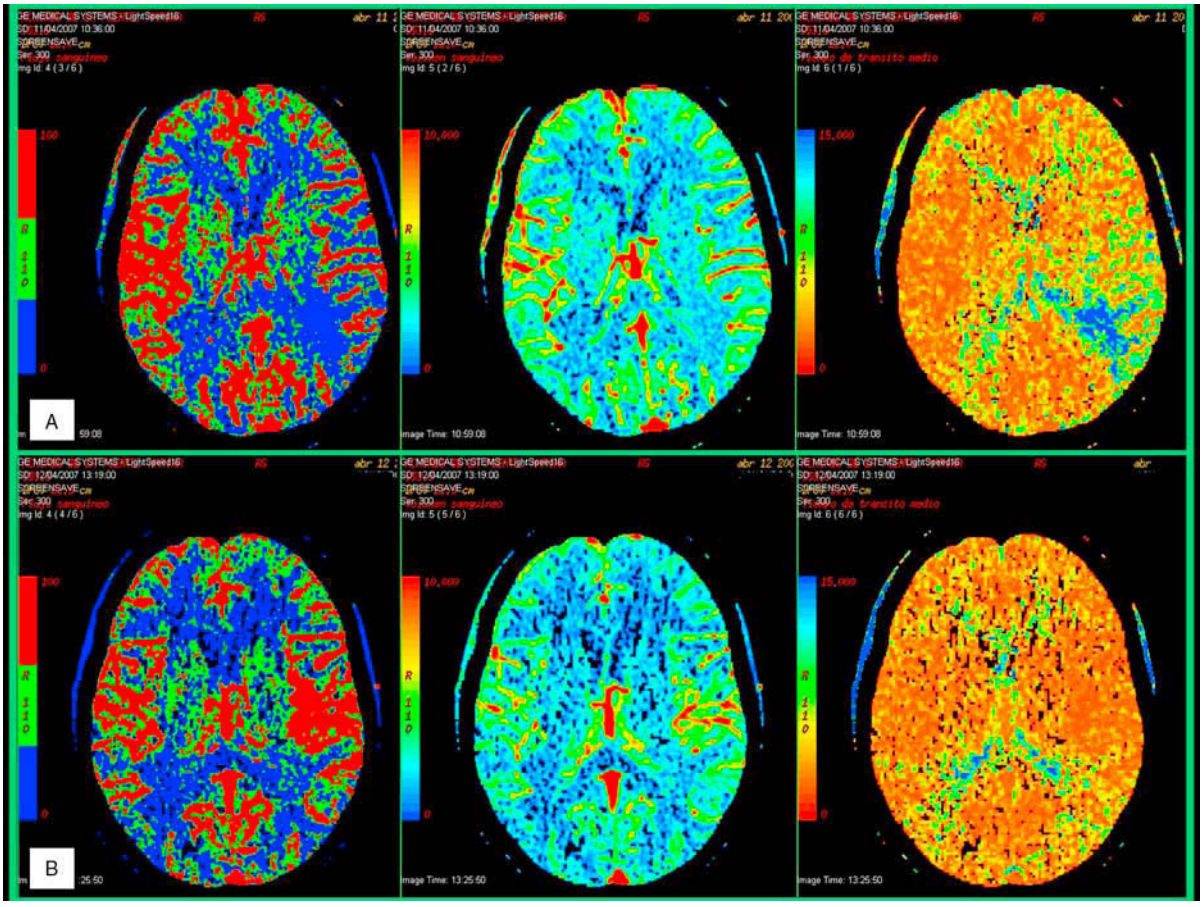


**Figure 1** CT perfusion. Panel A) Left posterior temporal area showing an increase in the mean time for transit of blood flow (6.39 versus 2.71 s), not translated into any significant reduction in blood volume. These findings imply a certain degree of difficulty in the vascularization of this area. Panel B) Perfusion study performed 24 hours after strengthening triple H therapy showing no alteration in the mean time for transit (3.41 versus 3.06 s), cerebral blood flow or cerebral blood volume.

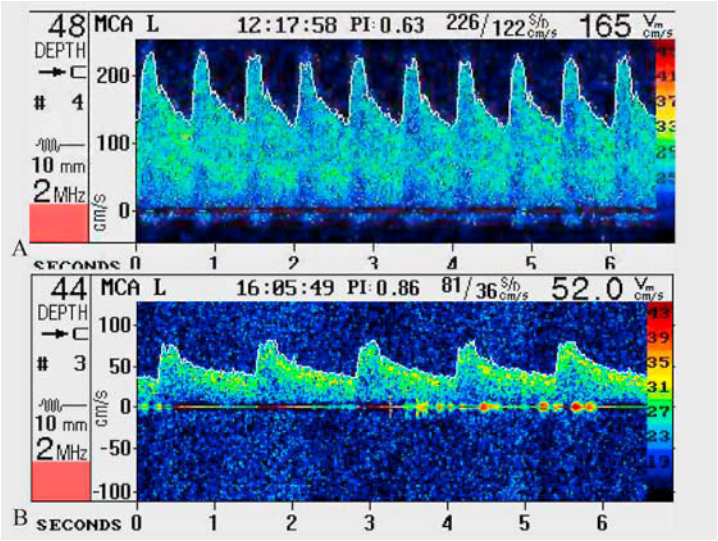


**Figure 2** Transcranial Doppler. **Panel A)** Ultrasound recording from the 16th, when the cortical focality appeared, in which it is possible to observe mean speeds of 165 cm/ s. **Panel B)** Triple H therapy normalized these speeds (52 cm/s).

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