Transcranial Doppler for vasospasm diagnosis

Transcranial Doppler, which is still recommended for arterial vasospasm by the American Heart Association Management Guidelines for SAH, has a number of limitations like operator dependence and interobserver variability, inadequate acoustic windows in 10% of patients, but, much more important, high variability of TCD vasospasm criteria in the literature as well as highly controversial results [1) 2)].

However, in a number of cases TCD vasospasm could be identified and proven by DSA; therefore TCD should not be neglected from clinical routine [3)]

Criteria

Mild, 120-140 cm/s

Moderate, 141-200 cm/s

Severe,>200 cm/s.

The following TCD values were considered pathological: mean blood flow velocity of the middle cerebral artery (MCA) >120 cm/s and mean blood flow velocity of the anterior cerebral artery (ACA) >50 cm/s Lindegaard index >3.

Although current US guidelines support the use of TCD for the diagnosis of vasospasm in aSAH, its routine application nationally is far from widespread [4) 5) 6)].

Transcranial Doppler provides a noninvasive method for recording blood flow velocity (and indirectly, diameter) in the basal cerebral arteries and therefore is especially useful in detecting vasospasm following subarachnoid hemorrhage. Vasospasm most commonly involves the basal arteries, where the changes in vessel diameter will be inversely proportional to the mean velocity measurements. Examination of patients requires that the examiner be experienced and familiar with the vascular anatomy and the various TCD indicators of vasospasm. Normal mean velocity for the MCA is 62 +/- 12 cm/sec. Significant spasm on angiogram of the MCA corresponds to a mean velocity of 120 cm/sec. Mean velocities of the MCA of 200 cm/sec or greater indicate severe spasm and correlate with 50% or greater narrowing on angiogram. Cerebral blood flow changes that can occur after subarachnoid hemorrhage and as a result of vasospasm may affect velocity values. A simultaneous index of CBF with either direct flow measurement techniques or by recording extracranial carotid artery velocity measurements may be helpful in reflecting these changes. Knowledge of the time course of the development and resolution of vasospasm using TCD can help the clinician predict which patients are at higher and lower risk of developing ischemic deficits, thereby guiding treatment. Several features of TCD assessment of vasospasm are similar to angiography. High TCD velocities, like severe angiographic vasospasm, are associated with delayed ischemic deficits and infarction, although some patients can remain asymptomatic despite these changes. Delayed ischemic deficits or infarctions in patients following subarachnoid hemorrhage usually will be preceded by markedly elevated velocity

or other indicators of severe vasospasm [7].

Transcranial Doppler (TCD) is widely used as a daily routine method to detect vasospasm in patients after aneurysmal subarachnoid hemorrhage (aSAH).

The reliability of transcranial Doppler ultrasound was better at detecting high mean cerebral blood flow velocities in patients with symptomatic vasospasm related to middle cerebral and internal carotid artery distributions than for anterior cerebral artery distribution. Transcranial Doppler ultrasound was as sensitive as cerebral angiography at detecting symptomatic vasospasm. High mean cerebral blood flow velocities can be apparent before the presence of symptomatic vasospasm. Daily transcranial Doppler ultrasound monitoring could provide early identification of patients with aneurysmal subarachnoid hemorrhage who are at high risk for symptomatic vasospasm and may be helpful at following success of endovascular treatment [8].

All patients included in a study received a standardized diagnostic and treatment protocol. 50 patients admitted with aSAH from 01/2013 to 12/2013 received daily TCD measurements. 39 patients admitted from 01/2014 to 09/2014 received no TCD measurements. Data on clinical grade (HUNT + HESS grade), severity of bleeding (BNI grade), localisation of aneurysm and angiographic or clinically relevant vasospasm were prospectively collected. The GOS, mRS and the NIHSS were used as clinical outcome parameters.

Patient baseline characteristics and clinical data were comparable; treatment modality of the aneurysm was not different between the groups (p = 0,7756). No significant difference between the Hunt and Hess grade (p = 0,818) and the BNI grade (p = 0,1551) was observed. There was also no significance concerning the incidence of angiographic or clinically relevant vasospasm between both groups (p = 0,5842 and p = 0,7933). GOS, mRS and NIHSS as the primary outcome parameters showed no significant difference in morbidity and mortality between both groups (mortality p = 0,8544).

With the limitation of an explorative cohort study the results indicate that routine TCD studies do not improve the overall outcome of patients after aSAH [9].

The ipsilateral to contralateral middle cerebral arteries (I/C mBFV) demonstrated a more significant correlation to vasospasm than the absolute mean flow velocity [10].

Case series

1999

The purpose of this study was to determine the correlation between transcranial Doppler (TCD) velocities and angiographic vasospasm after aneurysmal subarachnoid hemorrhage.

METHODS: In the first part of this study, patients were retrospectively reviewed to correlate middle cerebral artery absolute blood flow velocities with angiographic vasospasm. In the second part of the

study, the middle cerebral artery/ipsilateral extracranial internal carotid artery velocity ratio (Lindegaard ratio) was prospectively correlated with angiographic vasospasm. Angiographic vasospasm was independently graded, by observers blinded to the TCD results, as either none, mild (less than one-third artery luminal narrowing), moderate (one-third to one-half narrowing), or severe (more than one-half narrowing). The sensitivity, specificity, likelihood ratios for positive and negative TCD results, positive and negative predictive values, and kappa and P values were calculated.

RESULTS: One hundred one patients were analyzed in the first part of the study, and 44 patients were analyzed in the second part. Interobserver agreement regarding angiographic vasospasm was good (kappa = 0.86). Despite significant correlation between mean velocities and the degree of vasospasm, the clinical dependability of TCD velocities (evaluated using predictive values and likelihood ratios) was limited. The positive predictive value of velocities of > or =200 cm/s for moderate/severe angiographic vasospasm was 87% but that of lower velocities was approximately 50%. The negative predictive value of velocities of <120 cm/s was 94% but that of higher velocities was approximately 75%. Only the likelihood ratios for velocities of <120 or > or =200 cm/s were useful (likelihood ratio for negative result = 0.17, likelihood ratio for positive result = 16.39). Overall, 57% of patients exhibited maximum velocities in the indeterminate range between 120 and 199 cm/s. Lindegaard ratios did not improve the predictive value of TCD monitoring.

CONCLUSION: For individual patients, only low or very high middle cerebral artery flow velocities (i.e., <120 or > or =200 cm/s) reliably predicted the absence or presence of clinically significant angiographic vasospasm. Intermediate velocities, which were observed for approximately one-half of the patients, were not dependable and should be interpreted with caution [11)].

1995

In 40 patients middle cerebral artery trunk (M1) flow velocity was recorded just before 54 carotid angiography in 54 cases exhibiting vasospasm after aneurysm rupture. Angiographic vasospasm distribution was studied; cases of symptomatic vasospasm were noted and were compared with transcranial Doppler data. Angiographic vasospasm was present in M1 in 41/54 carotid angiograms. Postulating that all the cases of M1 angiographic vasospasm should be identified by transcranial Doppler, the theoretical sensitivity of TCD was 76%. In this series however the real sensitivity of TCD in vasospasm diagnosis was only 70%: besides 13 cases where vasospasm was not present in M1 (mainly after ACoA Aneurysm rupture), TCD failed to identify 3 cases of M1 angiographic vasospasm. Vasospasm may not be located in M1 even when severe and symptomatic (4 cases in this series). Transcranial Doppler remains a mediocre tool for identifying vasospasm after anterior communicating artery aneurysm rupture (sensitivity: 55%). Its reliability is better after internal carotid aneurysm rupture (sensitivity: 72%) and excellent after middle cerebral artery aneurysm rupture (sensitivity: 93%). In order to test the drugs or methods used to prevent or combat vasospasm, angiography has to be considered when during the vasospasm risk period TCD does not demonstrate vasospasm in M1, either in patients in whom clinical deterioration is occurring without other obvious explanation, or in all patients [12)].

1994

A retrospective analysis was undertaken to determine whether cerebral vasospasm following subarachnoid haemorrhage (SAH) correlates with the age of patients. For at least 3 weeks after bleeding 80 subjects underwent very close follow-up with clinical examination and transcranial Doppler records of the blood velocities within the basal cerebral arteries. Firstly a correlation between

measured maximal mean blood flow velocities and age was made. Secondly, according to their age and the maximum of recorded mean velocities (v), the patients were divided into groups as follows: age 55 years or less, age more than 55 years; and maximum velocity v1 < 90 cm/s, 90 cm/s < v2 < 120 cm/s, 120 cm/s < v3 < 160 cm/s, v4 > 160 cm/s. There was a significant correlation of the measured maximum mean velocities and the age of the patients ($r = -0.525$, $p < 0.01$). With regard to the velocity groups there was a significant (chi-squared statistic for contingency tables, $p < 0.01$) difference between both age-groups: 32% (n = 18) of the younger fell into group v4 with maximum mean velocities of more than 160 cm/s, but none of the older had such. Vice versa, 63% (n = 15) of the older compared with only 14% (n = 8) of the younger fell into group v1 with maximum mean velocities of less than 90 cm/s. Clinical follow-up also depicted differences between both age groups. 13 of 18 younger patients with maximum mean velocities > 160 cm/s exhibited symptomatic vasospasm with a delayed neurological deficit. This typical course did not occur in the older age group [13].

1)
Connolly ES Jr, Rabinstein AA, Carhuapoma JR, Derdeyn CP, Dion J, Higashida RT, et al. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2012;43:1711–37.
2)
Han SJ, Rutledge WC, Englot DJ, Winkler EA, Browne JL, Pflugrath L, Cronsier D, Abla AA, Kliot M, Lawton M. The Presto 1000: a novel automated transcranial Doppler ultrasound system. J Clin Neurosci. 2015;22:1771–5.
3)
Deb S, Gogos AJ, Drummond KJ, Teddy PJ. The role of transcra- nial Doppler ultrasound monitoring in patients with aneurysmal subarachnoid haemorrhage. J Clin Neurosci. 2012;19:950–5.
4)
Kumar G, Alexandrov AV. Vasospasm Surveillance With Transcranial Doppler Sonography in Subarachnoid Hemorrhage. J Ultrasound Med. 2015 Aug;34(8):1345-50. doi: 10.7863/ultra.34.8.1345. PubMed PMID: 26206820.
5)
Alexandrov AV, Sloan MA, Tegeler CH, Newell DN, Lumsden A, Garami Z, Levy CR, Wong LK, Douville C, Kaps M, Tsivgoulis G; American Society of Neuroimaging Practice Guidelines Committee. Practice standards for transcranial Doppler (TCD) ultrasound. Part II. Clinical indications and expected outcomes. J Neuroimaging. 2012 Jul;22(3):215-24. doi: 10.1111/j.1552-6569.2010.00523.x. Epub 2010 Oct 26. PubMed PMID: 20977531.
6)
Le Roux P, Menon DK, Citerio G, Vespa P, Bader MK, Brophy G, Diringer MN, Stocchetti N, Videtta W, Armonda R, Badjatia N, Bösel J, Chesnut R, Chou S, Claassen J, Czosnyka M, De Georgia M, Figaji A, Fugate J, Helbok R, Horowitz D, Hutchinson P, Kumar M, McNett M, Miller C, Naidech A, Oddo M, Olson D, O'Phelan K, Provencio JJ, Puppo C, Riker R, Roberson C, Schmidt M, Taccone F. The International Multidisciplinary Consensus Conference on Multimodality Monitoring in Neurocritical Care: evidentiary tables: a statement for healthcare professionals from the Neurocritical Care Society and the European Society of Intensive Care Medicine. Neurocrit Care. 2014 Dec;21 Suppl 2:S297-361. doi: 10.1007/s12028-014-0081-x. Review. PubMed PMID: 25608916.
7)
Newell DW, Winn HR. Transcranial Doppler in cerebral vasospasm. Neurosurg Clin N Am. 1990 Apr;1(2):319-28. Review. PubMed PMID: 2136144.
8)
Suarez JI, Qureshi AI, Yahia AB, Parekh PD, Tamargo RJ, Williams MA, Ulatowski JA, Hanley DF, Razumovsky AY. Symptomatic vasospasm diagnosis after subarachnoid hemorrhage: evaluation of transcranial Doppler ultrasound and cerebral angiography as related to compromised vascular

distribution. Crit Care Med. 2002 Jun;30(6):1348-55. PubMed PMID: 12072693.
9)

Ehrlich G, Kirschning T, Wenz H, Hegewald AA, Groden C, Schmiedek P, Scharf J, Seiz-Rosenhagen M. Is there an influence of routine daily transcranial doppler examination on clinical outcome in patients after aneurysmal subarachnoid hemorrhage? World Neurosurg. 2016 Jan 5. pii: S1878-8750(15)01714-3. doi: 10.1016/j.wneu.2015.11.091. [Epub ahead of print] PubMed PMID: 26768855.
10)

Nakae R, Yokota H, Yoshida D, Teramoto A. Transcranial Doppler ultrasonography for diagnosis of cerebral vasospasm after aneurysmal subarachnoid hemorrhage: mean blood flow velocity ratio of the ipsilateral and contralateral middle cerebral arteries. Neurosurgery. 2011 Oct;69(4):876-83; discussion 883. doi: 10.1227/NEU.0b013e318222dc4c. PubMed PMID: 21558976.
11)

Vora YY, Suarez-Almazor M, Steinke DE, Martin ML, Findlay JM. Role of transcranial Doppler monitoring in the diagnosis of cerebral vasospasm after subarachnoid hemorrhage. Neurosurgery. 1999 Jun;44(6):1237-47; discussion 1247-8. PubMed PMID: 10371622.
12)

Creissard P, Proust F, Langlois O. Vasospasm diagnosis: theoretical and real transcranial Doppler sensitivity. Acta Neurochir (Wien). 1995;136(3-4):181-5. PubMed PMID: 8748851.
13)

Boecher-Schwarz HG, Ungersboeck K, Ulrich P, Fries G, Wild A, Perneczky A. Transcranial Doppler diagnosis of cerebral vasospasm following subarachnoid haemorrhage: correlation and analysis of results in relation to the age of patients. Acta Neurochir (Wien). 1994;127(1-2):32-6. PubMed PMID: 7942178.