Transcranial Doppler Waveforms During Intra-aortic Balloon Pump Counterpulsation for Vasospasm Detection After Subarachnoid Hemorrhage

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BACKGROUND: Transcranial Doppler ultrasound is a standard screening tool for vasospasm after subarachnoid hemorrhage. Prevention of vasospasm-induced delayed cerebral ischemia after subarachnoid hemorrhage depends on optimization of cerebral perfusion pressure, which can be challenged by neurogenic stress cardiomyopathy. Intra-aortic balloon pumps have been utilized to augment cerebral perfusion, but they change the transcranial Doppler waveform, altering its interpretability for vasospasm screening.

OBJECTIVE: To assess the features of the transcranial Doppler waveform that correlate with vasospasm.

METHODS: We retrospectively reviewed cases of subarachnoid hemorrhage that underwent same-day transcranial Doppler ultrasound and angiography. Transcranial Doppler waveforms were assessed for mean velocity, peak systolic velocity, balloon pump-augmented diastolic velocity, and a novel feature, “delta velocity” (balloon pump-augmented velocity – systolic velocity). Relationship of flow velocity features to vasospasm was estimated by generalized estimating equation models using a Gaussian distribution and an exchangeable correlation structure.

RESULTS: There were 31 transcranial Doppler and angiography pairings (12 CT angiography/19 digital subtraction angiography) from 4 patients. Fourteen pairings had proximal vasospasm by angiography. Delta velocity was associated with proximal vasospasm (coefficient –6.8 [95% CI –9.8 to –3.8], $P < .001$). There was no significant correlation with proximal vasospasm for mean velocity (coefficient –13.0 [95% CI –29.3 to 3.4], $P = .12$), systolic velocity (coefficient –8.7 [95% CI –24.8 to 7.3], $P = .29$), or balloon pump-augmented velocity (coefficient –15.3 [95% CI –31.3 to 0.71], $P = .06$).

CONCLUSION: Delta velocity, a novel transcranial Doppler flow velocity feature, may reflect vasospasm in patients with subarachnoid hemorrhage and intra-aortic balloon pumps.

KEY WORDS: Cerebral vasospasm, Intra-aortic balloon counterpulsation, Neurogenic stress cardiomyopathy, Subarachnoid hemorrhage, Takotsubo cardiomyopathy, Transcranial Doppler

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Delayed cerebral ischemia (DCI), defined as neurological deterioration or infarction secondary to vasospasm

when other causes have been excluded,¹ complicates 20% to 30% of cases of aneurysmal subarachnoid hemorrhage (SAH),^{2,3} resulting in permanent disability or death in 10% to 20% of cases.⁴ The cornerstone of DCI prevention in the setting of vasospasm is cerebral perfusion pressure optimization. As 20% to 30% of patients with SAH develop neurogenic stunned myocardium (NSM), thought to occur due to excess catecholamine stimulation of the myocardium,⁵ achieving adequate perfusion can be challenging. In the setting of cardiogenic shock, intra-aortic balloon pumps (IABPs) have

ABBREVIATIONS: CBF, cerebral blood flow; CTA, CT angiography; DCI, delayed cerebral ischemia; DSA, digital subtraction angiography; IABP, intra-aortic balloon pump; ICA, internal carotid artery; MCA, middle cerebral artery; NSM, neurogenic stunned myocardium; PI, pulsatility index; PDAV, peak diastolic IABP-augmented velocity; SAH, subarachnoid hemorrhage; TCD, transcranial Doppler ultrasound; TTE, transthoracic echocardiography

been utilized in order to bridge patients through perilous, but transient, periods of comorbid NSM and vasospasm.⁶⁻¹¹

IABPs change the normal arterial waveform to a characteristic double-peaked waveform (Figure 1). Balloon inflation beginning at the dicrotic notch increases diastolic pressure throughout most of diastole, while deflation prior to the systolic upstroke reduces the systolic ejection pressure and the systolic pressure peak of the following beat. In conjunction, these changes result in improved coronary artery perfusion during diastole and left ventricular afterload reduction during systole.

As a noninvasive and low-risk test, transcranial Doppler ultrasound (TCD) has become a standard screening tool for vasospasm, especially in the middle cerebral arteries, where the

positive and negative predictive values for vasospasm as measured by mean flow velocity compared to digital subtraction angiography (DSA) are 98% and 78%, respectively.¹² TCD mean flow velocities increase with vessel narrowing per the equation $\text{velocity} = (4 \times \text{flow} / \pi) / \text{diameter}$,² such that small changes in diameter lead to large increases in velocity when flow is held constant.¹³ In patients without cerebral vasospasm, IABPs modify the pattern of cerebral blood flow (CBF) velocity without affecting the mean flow velocities, as the augmentation of flow velocity during balloon inflation in diastole is offset by an acute decrease in flow velocity during balloon deflation at pre-ejection.¹⁴

The predictive value of TCD mean flow velocity is likely inaccurate in the setting of an IABP, as the aforementioned alterations in the arterial waveform are reflected in the TCD velocity waveform (Figure 1). Hence, interpretation of this waveform for vasospasm detection requires further study. We sought to correlate features of the TCD waveform with same-day CT angiography (CTA) and, when available, DSA. CTA has previously been shown to have high diagnostic accuracy for vasospasm after SAH compared to DSA, the gold standard.¹⁵

METHODS

Design

We retrospectively reviewed consecutive cases in patients at our institution of SAH with NSM with IABP who underwent same-day TCD and either CTA or DSA. Demographics, baseline clinical status, and imaging results were obtained as part of a prospectively collected outcomes database. Our Institutional Review Board approved the study. Informed consent was obtained from all patients or their legally authorized representatives. All data were adjudicated in a weekly meeting by consensus of the clinical providers.

IABP Indications and Placement

We screened for NSM with admission screening transthoracic echocardiography (TTE) and repeat TTE as indicated by deterioration of hemodynamics. IABPs were placed at the bedside by consulting cardiologists percutaneously through the left femoral artery and advanced into the descending thoracic aorta just distal to the bifurcation of the left subclavian artery. Balloon size was chosen based on the patient's height. Placement was confirmed by chest X-ray. 1:1 counterpulsation, synchronized to the R wave of the electrocardiogram, was maintained throughout the treatment window. Patients were not anticoagulated with heparin while the IABP was in place. In the setting of an IABP and clinical deterioration from DCI, therapy was targeted to mean arterial pressure goals in lieu of systolic blood pressure goals. Other aspects of our clinical protocol for diagnosis and management of SAH have been described elsewhere.¹⁶

TCD Acquisition and Interpretation

TCDs were acquired daily on SAH days 0 to 14 by 1 of 2 board-certified Doppler technicians using a 2-MHz handheld transducer probe (Pioneer TC 4040; Nicolet Biomedical, Inc., Madison, Wisconsin). Examinations were performed between 10 am and 2 pm in order to minimize effects of circadian changes on CBF.¹⁷ Through transtemporal windows, insonation depth varied between 65 and 70 mm for the anterior cerebral artery, between 50 and 60 mm for the middle cerebral

artery (MCA), between 60 and 65 mm for the internal carotid artery (ICA), and between 55 and 70 mm for the posterior cerebral artery. Depth of insonation through the suboccipital window varied between 45 and 75 mm for the vertebral arteries and 70 to 120 mm for the basilar artery.

The software labels the peak velocity as the systolic velocity, although in the presence of an IABP counterpulsation, this peak corresponds to the peak diastolic IABP-augmented velocity (PDAV; see Figure 2). The true systolic velocity was measured from the waveform by one of the authors (NAM) who was blinded to the clinical context. A novel feature, “delta velocity,” was calculated by subtracting true systolic velocity (manually measured) from PDAV (automatically measured). The software calculates mean velocity using the following equation: mean velocity = (peak systolic velocity + [end diastolic velocity $\times$ 2])/3 (M. Witiak, Natus Neurology Incorporated, personal communication June 20, 2016). The pulsatility index (PI) was obtained from the software using the automated systolic velocity ($PI_{\text{auto}} = [PDAV - \text{diastolic velocity}] / \text{mean velocity}$) and also by using the manually measured true systolic velocity ($PI_{\text{meas}} = [\text{systolic velocity} - \text{diastolic velocity}] / \text{mean velocity}$). Cervical ICA velocities were not routinely obtained for measurement of Lindegaard ratios. As the effect of IABPs on TCD velocities was unknown, we made no attempt to grade TCD velocities as mild, moderate, or severe. Instead, TCD velocities were analyzed as a continuous variable.

Angiography and Interpretation

All patients underwent noncontrast CT scanning prior to CTA. Noncontrast CT was performed with a multislice CT scanner (GE Medical Systems, Port Washington, New York) using 120 kV, 170 mA, 2-s scan time, and 5-mm slice thickness covering from skull base to vertex parallel to the orbitomeatal line.

CTA followed immediately. CTA acquisitions were obtained with helical acquisition following a single bolus intravenous contrast injection of 90 to 120 mL of nonionic contrast into an antecubital vein at 3 to 4 mL/s. Imaging was triggered by an region of interest manually placed at the aortic arch and with an appro-

priate delay. Images were acquired from either aortic arch or skull base to vertex (25.0 cm; 140 kV, 170 mA; table speed 3.75 mm/s; 2.5-mm slice thickness with 2.5-mm interval; 1.0 s per rotation). CTA-source images were reconstructed to 1.25-mm thickness at 0.625-mm intervals and 5 mm maximum intensity projection reconstructions in the axial, coronal, and sagittal plane.

DSA was performed on one of either a Philips or Siemens biplane angiography suite. Bilateral extracranial internal carotid arteries and the dominant vertebral artery were selectively catheterized with a 5Fr system. Hand injections of 6 to 8 mL of contrast were used to acquire standard Towne’s frontal and lateral projections simultaneously with image acquisition continued into the late venous phase.

Vasospasm was graded by a neuroradiologist blinded to the clinical context (NM) as described by Jabbarli and colleagues.¹⁸ Briefly, intracranial vessels were segmented into supraclinoid internal cerebral artery, proximal (first segment) and distal (segments 2–4) MCA, anterior cerebral artery, and posterior cerebral artery portions bilaterally, as well as the basilar artery. Each segment was graded independently of other segments. A quaternary grading system was used where 0 = no evidence of vasospasm; 1 $\leq$ 50% vessel narrowing (mild); 2 = 50% to 75% vessel narrowing (moderate); and 3 $\geq$ 75% vessel narrowing (severe). The same system was used for both CTA and DSA.

Statistical Analysis

Relationship of flow velocity features to vessel vasospasm status in each vessel (for instance, R MCA) was estimated by generalized estimating equation models using a Gaussian distribution and an exchangeable correlation structure. $P < .05$ was considered significant. Due to a limited number of TCD and angiography pairings, grading of vasospasm was simplified to a dichotomized variable (present or not present). A subset analysis was performed correlating flow velocity features with only the presence of moderate-to-severe vasospasm (Jabbarli grades 2 or 3). Correlations of various flow velocity features were made separately for proximal vasospasm and distal vasospasm.

TABLE. Patient Characteristics and Clinical Status

Case	Age (y)	Sex	Admission Hunt and Hess	Admission modified Fisher score	Ejection fraction (%)	Duration (d) of IABP placement	IABP complications	Progression to infarction?	Follow-up mRS
1	56	F	3	3	40-45	3	None	No	1
2	60	F	3	4	15-20	7	Iliopsoas hematoma	Yes	0
3	81	F	5	4	<30	1	None	Yes	6
4	76	M	1	1	71	3	None	Yes	6

FIGURE 3. Delta velocity vs proximal vasospasm. An elevation of the delta velocity (PDAV – systolic velocity) was significantly associated with the presence of proximal vasospasm (coefficient –6.8 [95% CI –9.8 to –3.8], $P < .001$).

RESULTS

Four patients ($n = 4$) with SAH, IABP, and same-day TCDs and CTA or DSA were identified (Table). There were 31 TCD and CTA (12) or DSA (19) vessel pairings from those 4 patients, 14 of which had proximal vasospasm by angiography. Delta velocity was associated with proximal vasospasm (coefficient –6.8 [95% CI –9.8 to –3.8], $P < .001$, Figure 3). There was no significant correlation with proximal vasospasm for the mean velocity (coefficient –11.8 [95% CI –27.6 to 3.9], $P = .14$), the systolic velocity (coefficient –7.5 [95% CI –23.0 to 8.0], $P = .34$), the PDAV (coefficient –13.5 [95% CI –28.7 to 1.6], $P = .08$), the diastolic velocity (coefficient 4.4 [95% CI –3.7 to 12.4], $P = .29$), the PIauto (coefficient –0.1 [95% CI –0.2 to 0.0], $P = .17$), or the PI meas (coefficient –3.8 [95% CI –12.0 to 4.4], $P = .36$).

Twenty-nine of 31 TCD and CTA-DSA vessel pairings had distal vasospasm. Delta velocity significantly correlated with distal

vasospasm (coefficient 2.3 [95% CI 0.4–4.1], $P = .02$), as did PDAV (coefficient 5.75 [95% CI 2.0–9.5], $P = .0028$) and mean velocity (coefficient 3.9 [95% CI 0.7–7.1], $P = .02$). There was a trend for correlation with distal vasospasm for systolic velocity (coefficient 4.5 [95% CI 0.7–9.7], $P = .09$) and for diastolic velocity (coefficient 3.5 [95% CI 0–7.0], $P = .05$). There was no significant correlation with distal vasospasm for PIauto nor PI meas.

In a subset analysis assessing the correlation between TCD characteristics and only moderate-to-severe vasospasm (proximal or distal), only delta velocity showed significant correlation (correlation coefficient 2.1 [95% CI 0.1–4.0], $P = .04$).

There were no adverse events from any of the diagnostic tests in this study.

DISCUSSION

In this novel study, assessing the correlation of TCD flow velocity features with vasospasm (as measured by either CTA or DSA), we found that changes in delta velocity, the difference between PDAV and “true” manually measured systolic velocity from the TCD waveform, were strongly associated with radiographic proximal and distal vasospasm. This correlation held true in a subgroup analysis only assessing vessels with moderate-to-severe vasospasm. Mean flow velocity, usually considered the most accurate TCD measure of proximal vasospasm, was not significantly correlated with proximal vasospasm, although it was correlated with distal vasospasm. To the best of our knowledge, this is the first time that the unique aspects of an IABP-influenced TCD waveform have been analyzed for correlation with vasospasm.

Given the small sample, we view our data as hypothesis generating and not definitive. It is curious, for instance, that delta velocity negatively correlated with proximal vasospasm but positively correlated with distal vasospasm. Further work in larger samples is needed to confirm our findings.

Of note, mean flow velocity in our study was computed using the TCD software, from the computer-generated peak systolic velocity (actually the PDAV) using this equation: mean velocity = (peak systolic velocity + [end diastolic velocity $\times$ 2])/3. As IABPs significantly alter the TCD waveform, it is probable that this computation inaccurately describes the mean velocity,

especially as the PDAV was used instead of the true, measured systolic velocity. Furthermore, any values generated using the mean velocity, such as the PI, may be erroneous as well. Future studies should consider using the spectral weighted mean velocity.

There are few reports of TCD waveforms in SAH patients with IABPs, none of which correlate TCD waveform features with vasospasm. Spann et al¹⁹ reported high peak MCA velocities ranging from 160 to 240 cm/s in 4 patients with anterior circulation ruptured aneurysms who were treated electively with IABPs without evidence of NSM. They do not offer a presentation of the waveforms nor a correlation with radiographic vasospasm. Tacconne et al¹⁰ described 2 patients with MCA vasospasm and NSM who underwent TCDs just before and after placement of an IABI.¹⁰ In both patients, systolic flow velocity decreased while diastolic flow velocity increased with an overall increase in antegrade flow. They argue that this significant rise in antegrade flow may have been found in their patients with SAH and vasospasm due to loss of autoregulation, which otherwise attenuated IABP effects on CBF velocities in cardiac patients without acute brain injury.

Indeed, evidence for increased CBF with IABPs in either animals or humans is mixed.¹⁹⁻²⁵ The effect is likely mediated by severity of cardiac dysfunction, with IABP augmentations in CBF found more reliably in patients with more severe reductions in left ventricular ejection fraction.²⁵⁻²⁷ Given the rather severe nature of NSM in most of the SAH-IABP literature, in conjunction with likely impairments in autoregulation seen in severe vasospasm, the patients with concomitant NSM and SAH may be the most likely to experience improvements in CBF with placement of an IABI.⁶⁻¹¹

Limitations

Our study has limitations, primarily the small number of patients and matched TCD-angiogram pairs. Furthermore, we cannot comment on the Lindegaard ratio, another TCD feature that has been correlated with vasospasm,¹³ as we did not routinely assess velocities in the cervical ICA. We are also limited in our ability to infer effects on CBF, as these patients did not undergo direct CBF monitoring. Finally, our cohort was enriched for vasospasm as IABPs were placed specifically for the purpose of preventing DCI in patients with known vasospasm.

The development of a reliable marker of vasospasm is critical in patients with IABPs as IABP placement complicates performance of DSA, the gold standard test for vasospasm. Furthermore, there are concerns regarding the safety and burden of labor on nursing for transporting patients with IABPs to CTA. Thus, following the delta velocity on TCD as a measure of vasospasm holds promise as a noninvasive bedside tool to monitor for the presence of vasospasm.

CONCLUSION

Delta velocity, the difference between PDAV and systolic flow velocity, may be associated with radiographic vasospasm. Future study is required to corroborate this novel finding.

Disclosures

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