ON-LINE APPENDIX

Noninvasive Screening of Severe Cerebral Vasospasm

The noninvasive screening of severe vasospasm was done using a twice-per-day measurement of Transcranial Doppler (TCD) Velocity of the middle cerebral arteries.[1] A rapidly evolving (>50 cm.s-1) and asymmetric increase of the mean velocity of the first segments of middle cerebral arteries, or an absolute value ≥120 cm.s-1 non-related to global cerebral hyper perfusion was suspected to be related to a severe vasospasm. CT angiography (CTA) was performed when TCD was abnormal, not analyzable and quite systematically between the fifth and twelfth days in nonexaminable patients since TCD may present false negative in anterior or distal segments.[2,3] It was analyzed up the third segment of cerebral arteries. A vasospasm ≥50% or a rapidly evolving vasospasm ≥30–40% on CTA was suspected to be related to a severe vasospasm. CT perfusion was performed in rare cases in our center. It was done in second intention when CTA was not analyzable: for example, downstream a segment with metallic artifacts. In that setting, a severe vasospasm was suspected on color maps when the anomalies (increase of MTT >5.9 s or an MTT difference of greater than 1.1 s between normal and abnormal regions) were systematized to the territory downstream this segment and were not related to peri hematoma edema.[4]

DCIn Analysis

In addition to the vascular imaging used to monitor vasospasm (see above), the patients underwent nonenhanced CT and CTA at admission; nonenhanced CT after aneurysm treatment, after external ventricular drainage, and before hospital discharge; and nonenhanced CT and CTA when neurologic worsening or a severe vasospasm was suspected and within 48–72 hours after angioplasty to check for cerebral infarction and recurrence of vasospasm. The patients also underwent MR imaging at 6–12 weeks after aSAH onset, including at least the following sequences: DWI, FLAIR-weighted imaging, T2*WI, and 3D-TOF-MR angiography. Multiplanar reconstructions of nonenhanced CT, DWI, and/or FLAIR-weighted imaging sequences of any CT and MR imaging available at the acute phase and at follow-up were coregistered on a processing console (PACS).

DCIn was analyzed using the following processes:

1) A cerebral infarction was determined retrospectively with all the available imaging (including follow-up MR imaging) by the presence of any territorial hypodensity/hyperintensity on CT/DWI or FLAIR not related to hematoma or surgical access surrounding density/signal abnormalities or artifacts.
2) DCIn was defined as any cerebral infarction occurring within 2 to 21 days after aSAH, not related to other specific causes such as iatrogenic infarction, low-flow infarction, or infarction due to the mechanical effect of brain shift and herniation.[5] This analysis was blinded to vasospasm quantification and angioplasty territory if any.
3) The time of DCIn onset was estimated on CT according to previously reported methods.[6] We looked retrospectively for the very first examination date in which the following signs were visible: 1) slight gyral effacement and slight loss of gray/white matter interface on CT (<12-hours from stroke onset); 2) obvious cortical sulci effacement and loss of the gray/white matter interface (12- to 24-hours from stroke); 3) mild hypodensity on CT (24- to 48-hours from stroke onset); 4) obvious hypodensity on CT (>48-hours from stroke onset). Clinical data were subject to a second analysis to confirm the DCIn onset, especially if the date of cerebral ischemia was unclear. Patients without any imaging sufficient for DCIn dating were excluded from the analysis.
4) DCIn territory was defined according the usual vascular mapping of the brain.[7] A vascular territory was defined as the whole territory of 1 of the 6 cerebral arteries. Cerebellar artery territories were excluded from the analysis because no DCIns were found in them. A DCIn occurring in only certain segments of a cerebral artery (for example, the posterior M2 segment) was considered to occur in the whole territory of this artery.
5) Postangioplasty DCIn was defined as a DCIn occurring in the territory of a previous angioplasty. Because the effect of chemical angioplasty is transient, post-chemical angioplasty DCIn was considered only if the chemical angioplasty was performed ≤1 day from the DCIn. A balloon angioplasty performed in only certain segments of a cerebral artery (for example, the posterior M2 segment but not in the anterior M2 segment) was considered performed in the whole territory of this artery. In rare cases in which angioplasty was limited to the ICA, it was attributed to both the MCA and anterior cerebral artery territories.
6) A cerebral infarction was attributed to a complication of angioplasty when it occurred clinically and at imaging after angioplasty and in the same territory as an arterial embolism, dissection, or vasospasm worsening on postangioplasty DSA.

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On-line Table: Complications of angioplasty procedure ($n = 145$)[a]

	Technique	Infarction	Hemorrhage	Edema	Others
Intracranial dissection 12/145 (8%)	D-BA ($n = 5$) P-BA ($n = 7$) CA ($n = 0$)	D-BA ($n = 1$) P-BA ($n = 1$)	0	0	Late spontaneous normalization ($n = 9$) Chronic asymptomatic steno-occlusion ($n = 3$)
Intracranial embolism 7/145 (5%)	D-BA ($n = 3$) P-BA ($n = 2$) CA ($n = 2$)	D-BA ($n = 3$) P-BA ($n = 2$) CA ($n = 2$)	0	0	Need of mechanical thrombectomy in 1
Vasospasm worsening 12/145 (8%)	D-BA ($n = 10$) P-BA ($n = 2$) CA ($n = 0$)	D-BA ($n = 1$) P-BA ($n = 0$)	0	0	Spontaneous normalization a few minutes after a new BA or CA in all of them
Reperfusion syndrome 8/145 (5%)	D-BA ($n = 3$) P-BA ($n = 0$) CA ($n = 5$)	0	D-BA ($n = 3$) CA ($n = 1$)	D-BA ($n = 0$) CA ($n = 4$)	Extent infarction with refractory vasospasm before angioplasty in all of them It led to lethal intracranial hypertension in 4 (CA) and cerebral hematoma within the infarcted territory in 3 (BA) and 1 (CA) with favorable outcome

Note:—P-BA indicates proximal balloon angioplasty; D-BA, distal balloon angioplasty; CA, chemical angioplasty.

[a] Infarction, hemorrhage, and edema mean cerebral infarction, symptomatic intracranial hemorrhage, and malignant edema related to the angioplasty procedure.