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Decreasing the Diagnostic Cerebral Angiogram Requirements for Neuroradiology Fellows Would Be a Mistake

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Quantification of Carotid Stenosis on CT Angiography—Does Gender Matter?

I read with interest the report by Bartlett et al[1] describing a linear relationship between millimeter carotid stenosis, as measured on CT angiography, and derived percent stenosis. According to the "Materials and Methods" section, the authors did not evaluate their patient population on the basis of sex. Tartaglino et al,[2] however, reported that men and women differ in the average size of their internal carotid arteries (ICAs) on CT angiography by a minimum of 10% (larger in men). It is interesting to note that average brain weight is also approximately 10% greater in men than women. Bartlett et al reported an average distal ICA diameter of 4.4 mm. If their study population included equal numbers of men and women, it is plausible that the average distal ICA diameter would have been 4.6 mm in men and 4.2 mm in women. A 1.3-mm residual lumen in a female patient with a distal ICA diameter of 4.2 cm yields a 69% stenosis. Moreover, some women would likely have even smaller distal ICA diameters, resulting in a degree of stenosis <69%. Tartaglino et al[2] found that the 70% stenosis threshold by North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria required a smaller residual diameter for women than for men.

In summary, there is a potential effect of sex on the authors' measurement of 1.3 mm as a threshold value for assigning stenosis ≥70% by NASCET criteria. Although the potential effect is subtle, assigning separate threshold measurements for men and women (even if these differed by only 1 mm) might have further strengthened the authors' conclusions.

References

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Reply:

We thank Dr. Friedman for the interest in our recent article regarding quantification of carotid stenosis on CT angiography.[1] It is correct that the original data were not evaluated according to gender. We realized this oversight after publication of the original article.

We are currently undertaking a rigorous reanalysis of the data to incorporate gender into the model. By using gender-specific receiver operating characteristic curves, our preliminary data have shown that there is indeed a difference in the ideal cutoff values for severe and moderate disease in men and women. The difference, however, is only 0.1 mm, which makes the severe cut-off value for women 1.2 mm and the moderate cutoff value 2.1 mm. We are working to determine the statistical significance of this subtle difference. Because female patients comprised only 31% (42/132) of the original data, analysis of additional female patients may be necessary to have adequate power to examine this relationship.

In summary, our preliminary reanalysis has shown a slight difference in the gender cutoff values for severe and moderate carotid stenosis in CTA quantification. The difference, however, is very subtle, at 0.1 mm, which could be considered within range of acceptable measurement error for any given carotid. We hope to provide a more thorough statistical analysis of the gender differences in the near future.

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Decreasing the Diagnostic Cerebral Angiogram Requirements for Neuroradiology Fellows Would Be a Mistake

There is currently a discussion taking place among academic neuroradiology programs concerning the minimum number of required diagnostic cerebral angiograms for neuroradiology fellows. Currently, fellows in Accreditation Council for Graduate Medical Education–approved programs are required to perform 50 cerebral angiograms to satisfy the requirements. In recent years, some fellowship programs have been lobbying for a reduction in the cerebral angiography requirements for fellows. I think it is important for patient safety and the credibility of our subspecialty to at least maintain the requirements at the current level or, better yet, increase the number to 75.

The pressure to reduce cerebral angiography requirements has developed primarily as a result of increasing noninvasive MR imaging and CT procedure volumes. At institutions that are "fellow driven," fellows are needed to run the MR imaging and CT services. To keep up with growing cross-sectional volumes, opportunities for fellows to perform conventional angiography are compromised. A simple solution is to reduce the number of required angiograms and thus time spent away from cross-sectional services. This solution, however, has 2 serious consequences. First and foremost, patient safety is compromised if fellows finish their training with less than 50 angiograms and begin performing these potentially dangerous procedures unsupervised. The performance of cerebral angiography has not become easier in the last several years and neuroradiology fellows are presumably not smarter than their predecessors. If we considered 50 cerebral angiograms to be a minimum requirement in the past, why are we considering a reduction in the numbers now? The second consequence to decreased training in cerebral angiography is the inevitable erosion of our credibility among other specialties when it comes to the performance of this procedure. Without a doubt, neuroradiologists are currently the experts when it comes to performing and interpreting cerebral angiograms. No other specialty can claim equivalent training in imaging-guided procedures and radiation physics; however, we put our expertise in significant jeopardy if we dilute our training requirements. The competence of trainees who have performed less than 50 cerebral angiograms is suspect at best and places patients and our credibility at risk.

As a subspecialty community, we should carefully weigh the consequences of reducing the fellowship training requirements for cerebral angiography. Diluting the numbers with noninvasive angiography techniques such as MR angiography and CT angiography cannot replace the hands-on training required to competently perform con-

ventional angiography. Although simulator devices can be an important adjunct to training, these too are insufficient to serve as a surrogate for performing angiograms on patients and adequately dealing with the many complications that can occur.

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Agenesis of Bilateral Internal Carotid Arteries in the PHACE Syndrome

We read with interest the paper by Weon et al,[1] in which they described the case of a 14-year-old girl with a complex association of agenesis of the bilateral internal carotid arteries with transcranial collaterals from the external carotid artery, agenesis of the vertebrobasilar system, hypoplasia of the right cerebellar hemisphere, absence of the inferior cerebellar vermis, and facial capillary hemangioma. PHACE syndrome is a constellation of anomalies that includes posterior fossa abnormalities, hemangiomas, arterial abnormalities, aortic coarctation and cardiac abnormalities, and eye abnormalities.[2] The acronym has been subsequently expanded to "PHACES" to include sternal defects, which may be associated in a minority of patients.[2] Among arterial anomalies occurring in patients with PHACE syndrome, Weon et al noted that agenesis of major arteries, such as the internal carotid and vertebral arteries, is usually unilateral and occurs ipsilaterally to the cutaneous lesion.

In a 2001 paper with a coincidentally similar title[3] that was unfortunately not cited by Weon et al, we described 3 patients with PHACE syndrome, one of whom had a complex arterial abnormality bearing some similarities to that described by Weon et al. This patient was a female neonate with bilateral agenesis of the internal carotid arteries, as shown by MR angiography and confirmed by the absence of the carotid canals on the bone window setting of brain CT. Unlike Weon et al's case, in our case the anterior circulation was reconstituted by a huge basilar trunk via enlarged posterior communicating arteries, whereas the external carotid artery branches did not contribute to the anterior circulation except for the right ophthalmic artery originating from a branch of the right middle meningeal artery. Thus, to the best of our knowledge, Weon et al's is the second report on bilateral agenesis of the internal carotid arteries in the setting of PHACE syndrome.

Other features of our case included tricuspid atresia, right hemispheric cerebellar cortical dysplasia (until then a novel feature of PHACE syndrome), and a remarkably minor cutaneous expressivity with a capillary hemangioma of the right pinna in the absence of the disfiguring hemifacial hemangioma that is found in most patients with PHACE syndrome.[2] We did not find ophthalmologic abnormalities or sternal defects, and also the other 2 cases from our series,[3] as well as an additional, unpublished case that we recently observed, did not display the full phenotypic spectrum of the syndrome. Thus, we agree with Weon et al that the PHACE syndrome is heterogeneous and that absence of one or more components is the rule. We also believe that all patients with a facial hemangioma (regardless of size) should undergo neuroradiologic, cardiologic, and ophthalmologic investigations to disclose possible associated abnormalities. It is hoped that future research will establish more precise diagnostic criteria and, one hopes, disclose the genetic background to what we believe is not merely an association of findings—etymologically, a syndrome—but rather a true vascular phakomatosis.

References

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Post-transplant Neurotoxicity: What Role do Calcineurin Inhibitors Actually Play?

It is well known that calcineurin inhibitors (CIs; cyclosporine and tacrolimus) may induce severe neurotoxicity even at therapeutic levels.[1] Major central nervous system (CNS) complications induced by CI include headaches, altered mental status (AMS), seizures, cortical blindness, auditory and visual hallucinations, spasticity, paresis, and ataxia. It is interesting that, in their recent article, Besenski et al[2] found AMS, headaches, and seizures as the most common symptoms not only in the kidney transplant recipients (KTR) whom they studied, but also in a group of pretransplant patients.

The pathogenesis of CI-induced CNS toxicity remains unclear. It has not been determined whether the clinical symptoms in KTR treated with CI are due to the direct drug toxicity, hypomagnesemia or hypocholesterolemia, hypertension, or a combination of these. Is the mechanism due to demyelination, ischemia mediated by vascular spasm, or hypertension?

It has been suggested in the literature that subcortical edema is the result of a hyperperfusion insult promoted by endothelial damage with breakthrough of autoregulation in the posterior circulation, which has paucity of sympathetic innervation. MR imaging perfusion studies have shown areas of signal intensity abnormality, whereas diffusion studies have been negative. Endothelial cell damage could be responsible for direct injury to the capillary bed and alteration of the blood-brain barrier, as well as the release of potent vasoconstrictors resulting in vasospasm. Injury to the blood-brain barrier may occur and recent reports note brain enhancement in several patients.[3]

In the series presented by Besenski et al, none of the patients had hypomagnesemia or hypertension, and only 9% had a cholesterol level $<$120 mg/mL. As the authors stated, this probably contributed to the low incidence of CI CNS toxicity in their study. The authors found posterior reversible encephalopathy syndrome (PRES) in 5% of KTR but also in 4% of the comparison group. It is not clear from this report why the 4% of pretransplant patients had PRES, because they were not hypertensive or taking any neurotoxic medication. PRES can be seen in other conditions such as eclampsia, hypertensive encephalopathy, systemic lupus erythematosus, and thrombotic microagiopathy. Besenski et al's data suggest that, in KTR, PRES is probably not exclusively caused by direct CI CNS toxicity because the incidence was similar in KTR and in the pretransplant comparison group and in their different groups 1, 2, and 3 (the groups were based on the time interval between transplantation and MR imaging examination). They therefore suggested that the etiology of PRES in KTR is multifactorial and needs further investigation. They did note, however, that statistical power was restricted by the number of patients in their study.