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**Cerebral Aneurysms in a Patient with
Osteogenesis Imperfecta and Exon 28
Polymorphism of *COL1A2***

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Cerebral Aneurysms in a Patient with Osteogenesis Imperfecta and Exon 28 Polymorphism of *COL1A2*

Petrucellis et al¹ present an interesting case of a patient with osteogenesis imperfecta and a ruptured aneurysm at the fenestrated basilar artery. However, they misidentify the fenestration as a vertebral fenestration and, as such, do not seem to relate the fenestration to the basilar aneurysm. Figures 1A and 2A beautifully show well-known features of the basilar fenestration just above the vertebral junction^{2,3}: the joining of both vertebral arteries, subsequent division of the basilar artery into 2 arms, effective widening of the distance between the lateral walls of both arms compared with the basilar diameter beyond, and rejoining of the fenestrated arms into 1 artery.

The relationship of aneurysms of the proximal basilar trunk and basilar fenestrations is well known.^{2,3} A substantial series by Campos et al² of 59 aneurysms of the basilar trunk found 35.5% in association with definite fenestrations, all but 1 at the proximal end of the fenestration. It is possible that other fenestrations were there but were not discerned because the aneurysms were superimposed over the basilar fenestrations, with the result of a higher incidence. With easy-to-do maximum intensity projections or multiplanar reformations with high resolution on CTA or MRA,⁴ viewed with a high index of suspicion, we can now readily show fenestrations. With sectioning of image datasets, aneurysms will less likely superimpose fenestrations.

In the case report by Petrucellis et al,¹ the discussion of osteogenesis imperfecta is interesting and educational for that entity. However, by not paying attention to the details of their own images, they missed the real point of this case. The important entity of aneurysm at the basilar fenestration is considered to develop as a result of hemodynamic forces on the "crotch" of the fenestration, leading to aneurysms in patients without osteogenesis imperfecta. In this patient with osteogenesis imperfecta and a fenestration aneurysm, the question raised is whether osteogenesis imperfecta is an innocent coincidental bystander.

The authors claim that the aneurysm seen 4 months after coiling is new, with angiograms showing a difference between the right posterior oblique views in Fig 1 and left posterior oblique, lateral, and Towne views in Fig 2. Again, the lack of attention to detail of these images leads the authors to claim that a new aneurysm developed in 4

months. This conveniently shows the neck of the so-called "new aneurysm" in the same spot as the treated aneurysm, just at the left side of the proximal split of the basilar fenestration. We can compare Fig 1A with Fig 2C for the closest possible orientation, and this comparison gives strong suggestion of the same aneurysm with a refilled neck after coiling, a common enough finding. It seems, then, that this aneurysm is not a rare, newly developed one but another occurrence of lack of attention to the details of the case.

Many reports describing coiling of aneurysms at the basilar fenestration are in the literature.⁵ Perhaps this is the first reported case in a patient with osteogenesis imperfecta, but the discussion in this case avoids this main theme through oversight of important findings and claims others that are dubious. The *American Journal of Neuroradiology* has an educational responsibility to show readers exemplary neuroimaging cases and interpretations, in addition to rigorous scientific reports and interesting musings of authors in discussion.

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