Letter to the Editor Regarding "Bioccipital Lobe Hypoperfusion and Anton's Syndrome Resolution with Intravenous Thrombolysis"

Dear Editor

We have read with great interest the article by Rotkopf et al.[1] The authors presented a rare case of Anton's syndrome confirmed by multiparametric computed tomography (CT) and completely resolved after the administration of recombinant tissue plasminogen activator (rtPA). We absolutely agree with the authors that multiparametric CT imaging may aid in quickly proving the underlying stroke in Anton's syndrome, especially helpful considering the discrepancy between the patient's perception and clinical examination results. However, few potential limitations, including incorrect legends, some misleading information, and inadequate arguments need to be addressed regarding this article.

First, the Figure 1(A) showed noncontrast CT in the article, but wrongly annotated Figure 1(D) in the legend. Correspondingly, CT angiography image was labeled Figure 1(A) by error. In addition, the cerebral blood flow map and cerebral blood volume were described in reverse in the case presentation.

Second, the authors pointed out that the only remaining symptom was bilateral loss of the menace reflex, on admission to the stroke unit half an hour later. That is to say, the patient's vision had completely returned to normal at that time. Nevertheless, the negative menace reflex

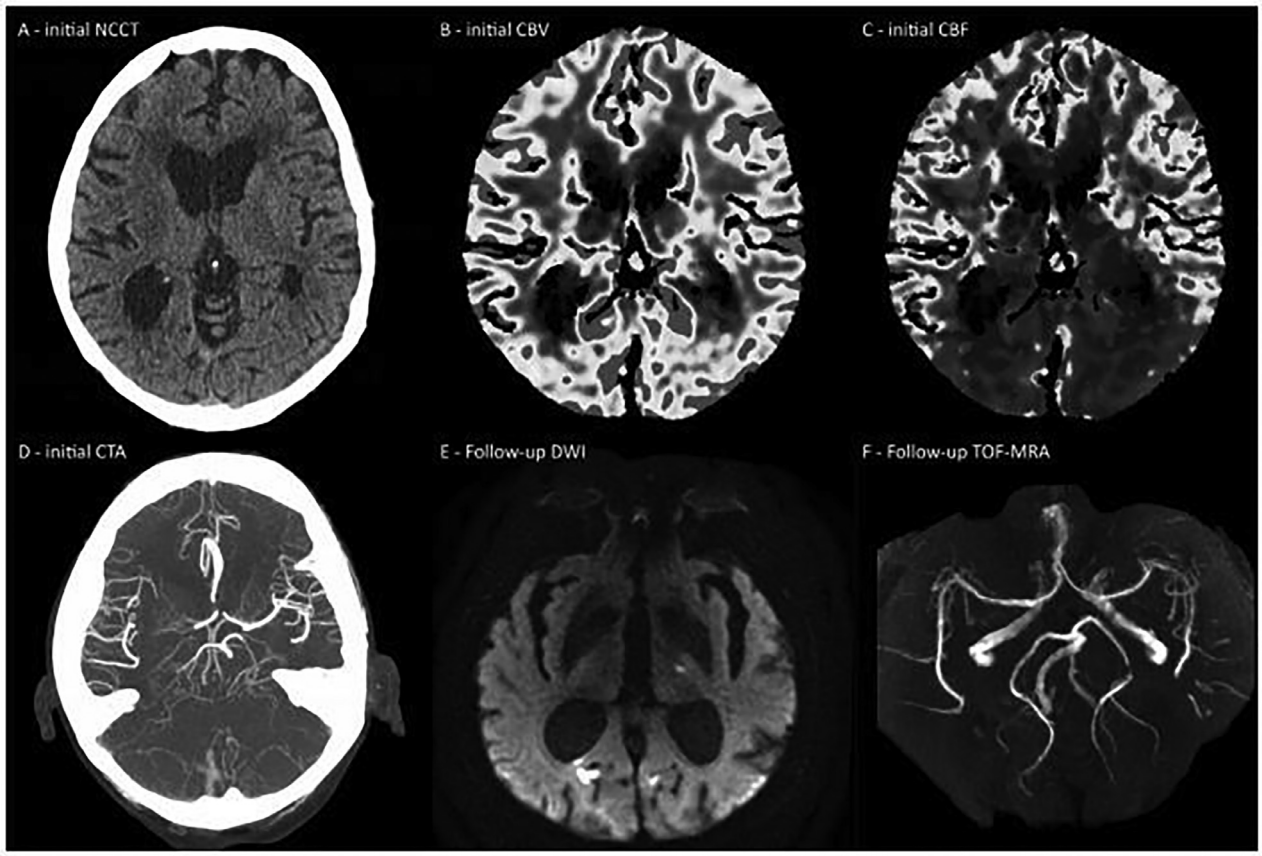

Figure 1. *Bilateral P2 segment occlusion with corresponding perfusion deficit in the posterior occipital cortex. (**A**) Depth-weighted maximum intensity projection of CT angiography demonstrating bilateral P2 segment occlusion (red arrows), (**B**) Cerebral blood volume perfusion map, (**C**) Cerebral blood flow perfusion map, (**D**) Initial noncontrast CT, (E) Diffusion-weighted magnetic resonance imaging at day 5 (F) Time-of-flight MR angiography at day 5. Abbreviation: CT, computed tomography.*

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on both sides with normal vision was not in line with common sense.

What's more, we hold the same opinion with the authors that Anton's syndrome is characterized by a combination of visual anosognosia (denial of loss of vision) and confabulation of visual experience in the setting of obvious visual loss and cortical blindness.[2] However, we disagree with the following view that Anton's syndrome represented a form of visual neglect. As illustrated by previous studies, visual neglect is a common syndrome in which patients neglect one side (typically the left) of space without visual field defect and often caused by the right middle cerebral artery strokes as well as other causes, especially right parietal lobe lesions.[3-5] However, Anton's syndrome refers to cortical blindness with confabulation in spite of obvious visual loss, mostly due to the damage to the bilateral occipital cortices.[6-7] The lesions location and clinical manifestation of the 2 syndromes are distinctly different. Therefore, such a statement might not be appropriate.

Finally, according to the authors, cardioembolic stroke caused by atrial fibrillation was considered the most likely cause in this case. However, the patient's past medical history and condition of extracranial vasculopathy were not mentioned here, and the bilateral posterior cerebral artery focal occlusion was observed on CT angiography. Accordingly, on the basis of TOAST etiological classification,[8] the etiology of this patient was undetermined. It might be more accurate to say that this patient was attributable to mixed-type (artherosclerotic plus cardioembolic) infarction due to atrial fibrillation and bilateral posterior cerebral artery occlusion.

While, we again congratulate the authors for their impressive work, namely, an attempt to quickly reveal the underlying etiology of Anton's syndrome by multiparametric CT imaging, despite some flaws. It would be interesting to further analyze the neuropsychological characteristics in patients with Anton's syndrome.

Shugang Cao, MM,*
Xiaoxing Ni, MM,
Wen'an Xu, MM
Department of Neurology, Hefei Affiliated Hospital of Anhui Medical University, Hefei, Anhui Province, China
E-mail address: caoshugg@126.com

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