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Modified hypoglossal-facial nerve anastomosis for peripheral-type facial palsy caused by pontine infarction: A case report and literature review

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ABSTRACT

Background: Peripheral-type facial palsy could be caused by a lesion in the tegmentum of the pons, such as infarction, with a rare occurrence. We herein described a case of unilateral peripheral-type facial palsy induced by dorsolateral pontine infarction and treated this patient using modified hypoglossal-facial nerve anastomosis.

Case presentation: A 60-year-old female presented with dizziness, hearing drop, diplopia, and peripheral-type facial palsy. Brain Magnetic Resonance Imaging showed a dorsolateral pontine infarction on the right side which exactly refers to the location of the ipsilateral facial nucleus or facial nerve fascicles at the pons. Subsequent electrophysiological examinations confirmed poor facial nerve function of this patient and modified hypoglossal-facial nerve anastomosis was then performed.

Conclusions: This case reminded medical practitioners not to ignore the possibility of involvement of a central cause in peripheral-type facial palsy patients. In addition, modified hypoglossal-facial nerve anastomosis served as a useful skill improvement that may help reduce hemiglossal dysfunction while restoring facial muscle function.

1. Introduction

Facial palsy is a common disease in neurological daily practice and can be divided into two types, termed central and peripheral-type facial palsy (P-FP), respectively [1]. In most cases, the absence or presence of forehead movement is crucial in distinguishing central-type facial palsy from peripheral one [2]. Etiologically, P-FP could be induced by the lesion in the tegmentum of the pons [3],

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such as infarction. However, the situation is rare and easily neglected by the clinician.

Different treatment modalities have been used for P-FP in the past decades, including drug, physical therapy, and botulinum toxin injections [4]. When patients remain complete or persistent incomplete facial palsy after receiving the abovementioned conservative therapies, a variety of operative techniques may provide help [5]. Among these surgeries, tensionless hypoglossal-facial nerve end-to-end anastomosis represents the most classic method with optimal outcomes [6,7]. However, total transection of the hypoglossal nerve may result in inevitable hemiglossal atrophy, which severely interferes with speech, mastication, and/or swallowing, significantly decreasing patients' quality of life [8–11]. To preserve tongue function, several variants of classic hypoglossal-facial nerve anastomosis (HFA) have been proposed and subsequently improved in past decades [8,12]. Among these modified surgical procedures for facial nerve reanimation, the ansa cervicalis was less commonly used. Initially, the ansa cervicalis was implemented for recurrent laryngeal nerve reconstruction, showing great superiority in lessening donor nerve morbidity [13]. Moreover, several studies have reported favorable results in the restoration of the mimetic muscles function after the use of the ansa cervicalis [14,15]. At the Department of Neurosurgery in Xinhua Hospital, this surgical procedure was adopted, in which the distal stump of the hypoglossal nerve was sutured with the proximal stump of the ansa cervicalis after end-to-end hypoglossal-facial nerve anastomosis as previously described [16]. According to our experience, facial palsy patients with this technique acquired favorable outcomes and the risk of tongue-related morbidities was significantly decreased.

Here, we report a rare case of P-FP caused by pontine infarction and treated with HFA combined with ansa cervicalis-hypoglossal nerve anastomosis.

2. Case presentation

The patient was a 60-year-old female, with symptoms of inability to raise an eyebrow, close her right eyelid tightly, and movements of the corners of her mouth restricted. She complained of dizziness and a slight slant of corners of her mouth towards the left, accompanied by a hearing drop on the right side and diplopia 7 months ago. Her symptoms of weakness of facial muscles and deterioration of right hearing developed 12 days after the onset. She chose to visit the emergency room of Xinhua Hospital and then was admitted to the Department of Neurology. After admission, this patient was treated with antiplatelet medication, and symptoms including dizziness, diplopia, and hearing drop in the right ear were significantly relieved during hospitalization this time. However, some symptoms gradually aggravated such as being incapable of raising the right eyebrow, unable to close the right eyelid and slanting of the corners of her mouth towards the left. The patient had been diagnosed with hypertension 5 years previously and took nifedipine (40 mg/day) irregularly. In addition, she also had a history of obesity for 20 years with no intervention. After discharge from the Neurology Department, this patient took antiplatelet medication (aspirin 100 mg/day) and received acupuncture and moxibustion therapy.

Disappointedly, her facial palsy was not recovered, and thus, she was admitted into the Neurosurgery Department this time. On admission, the disappearance of the nasolabial fold in her right face and left-slanted mouth corner was observed clearly. Further neurological examination showed that she was unable to achieve eyelid closure completely and the ipsilateral sclera emerged during maximal effort, without detection of impairment of pain and temperature in the face. The muscle strength of the frontalis and orbicularis oculi on the right side was too weak to lift the eyebrow (Fig. 1). In addition, this patient experienced no forms of facial spasm or synkinesis. Normally, telling apart P-FP from C-FP mainly depends on the presence of the upper face involvement. This patient presented unilateral facial palsy that involves her right upper and lower face. Hence, she was diagnosed as P-FP definitely and reached degree V based on the House-Brackmann (HB) score.

After analyzing the brain Magnetic Resonance Imaging (MRI) scan taken during her first hospitalization in the Neurology Department retrospectively, we got a discovery. A lesion was found located in the dorsolateral to the right pons with a hypo-intense

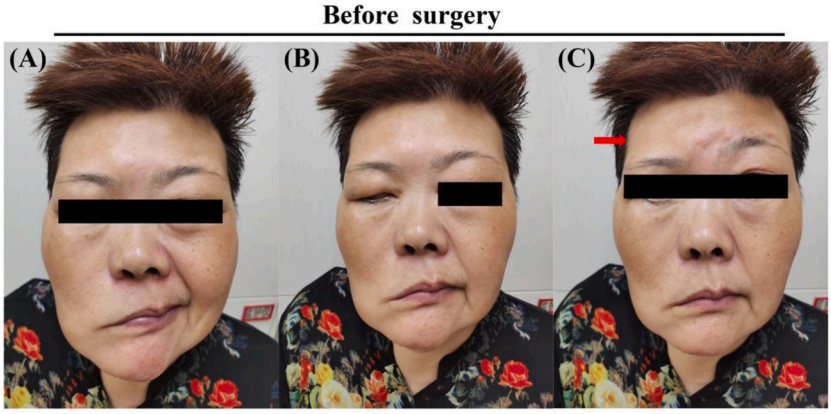

Fig. 1. Representative photographs depicting the patient with right peripheral-type facial palsy. (A, B). Preoperative photographs demonstrating severe facial palsy. (C). The patient showed no movement of the right forehead and eyebrow (red arrowhead) compared to the contralateral healthy side while wrinkling with maximal effort.

signal or moderated-intense signal in T1 or T2-weighted MRI, respectively (Fig. 2A and B). Brain diffusion-weighted image showed no signs of hyper-intense signal (Fig. 2C). Because the duration between the time of onset and brain MRI image acquisition exceeded 12 days or even more and this patient showed a classical clinical presentation of P-FP, we concluded that it was the subacute dorsolateral pontine infarction indicating the true etiology. According to previous literature reports, it was a rare situation in which peripheral facial palsy resulted from a pons lesion. Etiologically, any central lesion which interferes with the ipsilateral facial nucleus or facial nerve at the pons may cause peripheral facial palsy [17,18]. As shown in Fig. 3, we highlighted the approximate area of the facial nerve damage according to the brain MRI image of the patient. In this case, the facial nerve tract was injured due to the pons infarction. Additionally, there may be small perforating artery occlusion in this case due to no vertebrobasilar artery main trunk occlusion or stenosis showed by Magnetic resonance tomographic angiography (MRTA) (Fig. 2D).

We conducted some electrophysiological examinations to test the function of the facial nerve and mimic muscles. The results of electroneuronography (ENoG) indicated that the amplitude of the right facial nerve compound muscular action potential was significantly decreased in comparison to the healthy contralateral side, suggesting the existence of nerve injury on the affected side (Fig. 4A). In addition, electromyography (EMG) further confirmed the presence of insertional activity of the right facial muscles, with tremendously decreased voluntary activity (Fig. 4B). Considering that drug treatment for more than 6 months with no efficacy coupled with the aforementioned electrophysiological data, the patient was recommended for surgical interventions such as HFA. The hypoglossal nerve was transected during the traditional HFA procedure, leading to dysfunction of ipsilateral tongue musculatures such as dysphagia and dysarthria. A variety of improved procedures based on the classic HFA were proposed, which consistently aimed protection of hypoglossal nerve function [19]. To that end, we performed a variant surgical method of classic HFA that the distal stump of the hypoglossal nerve was sutured with the proximal stump of ansa cervicalis after end-to-end hypoglossal-facial nerve anastomosis. Strikingly, patients had favorable outcomes with no or mild hemiglossal dysfunction, and corresponding study results have been

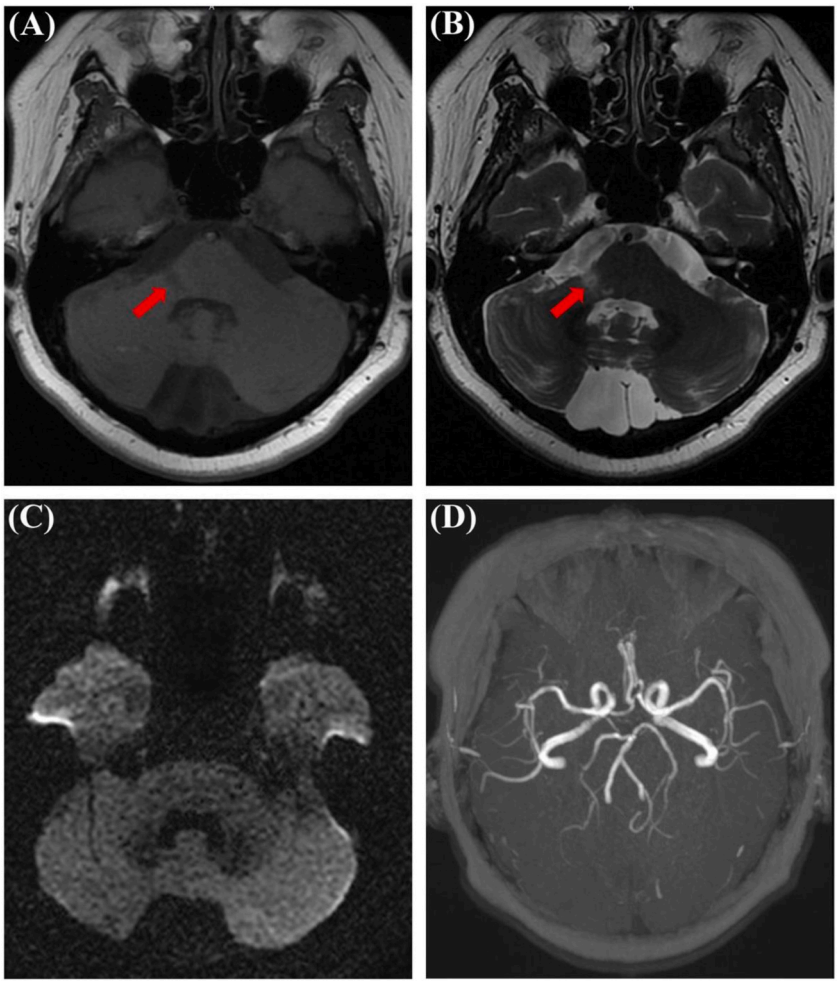

Fig. 2. The brain MRI images showed a subacute infarction located in the dorsolateral to the right pons. (A, B). A lesion with hypo-intense signal or moderated-intense signal (red arrowhead) located in the dorsolateral to the right pons was revealed by T1 (A) or T2-weighted MRI (B), respectively. (C). Brain diffusion-weighted image showing no signs of acute pontine infarction. (D). MRTA shows no vertebrobasilar artery main trunk occlusion or stenosis.

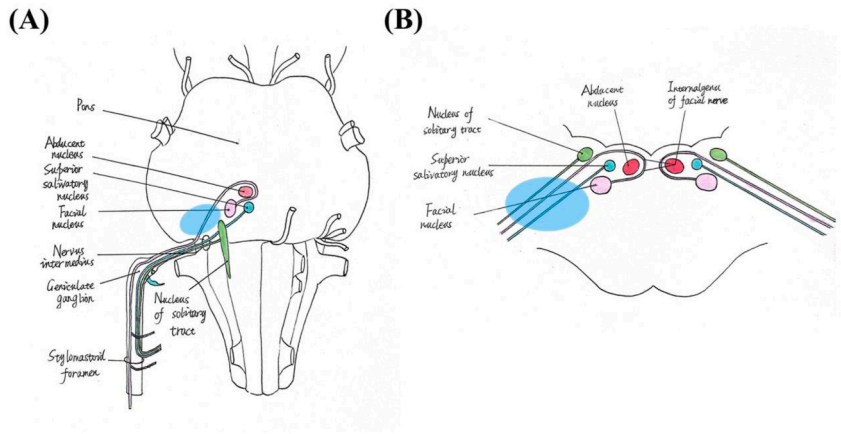

Fig. 3. Schematic drawing illustrated the possible damaged facial nerve tract due to pons infarction in the case. (A). Anterior view of the brainstem, roughly showing the course of the facial nerve. (B). Cross-section through the pons at the level of the internal genu of the facial nerve. The facial nerve tract damage caused by pons infarction was marked by a blue oval.

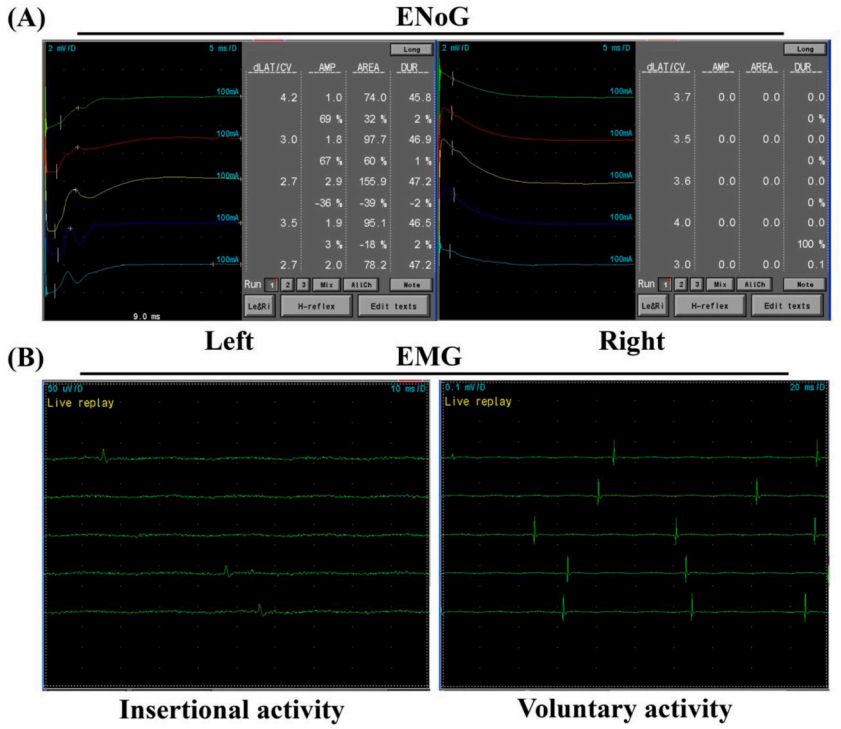

Fig. 4. Preoperative electrophysiological measurement results. (A). Electroneuronography showed that the amplitude of the right facial nerve compound muscular action potential was significantly decreased compared to the healthy contralateral side. (B). Electromyography revealed the presence of insertional activity in the right facial muscles, with greatly reduced voluntary activity.

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The surgical technique detail was the same as previously described [16]. Briefly, we did a preauricular skin incision routinely at first and then completed exposure and dissection of the hypoglossal nerve. The ansa cervicalis found along the course of the hypoglossal nerve was carefully dissected and prepared for subsequent anastomosis with the distal stump of the hypoglossal nerve. The facial nerve was sheared at the stylomastoid foramen and an anastomosis was performed using 9-0 prolene between its distal stump and the proximal stump of the hypoglossal nerve. Two nerve anastomoses areas were protected with nerve sheaths to avoid extra damage. The operation was successful and the patient was discharged under close follow-up. In addition, the patient is now receiving vitamin B1 (20mg tid) and methylcobalamin (0.5mg tid) orally and physical therapy such as massage three times per day until 6 months postoperatively.

3. Discussion

Such case of P-FP due to pontine infarction has been rarely reported. We described the characteristic findings of a patient with dorsolateral subacute pontine infarction who showed ipsilateral P-FP. Moreover, our case also highlighted the applicability of modified HFA in reducing hemiglossal atrophy for severe P-FP.

The facial nucleus in humans has a deep location in the caudal pons [20,21], its axons initially pass dorsally, looping over the abducens nucleus and forming the facial colliculus at the floor of the fourth cerebral ventricle [21]. Then the axons descend ventrolateral to the brainstem and exit at the junction of the medulla oblongata and pons and subsequently enter the internal auditory meatus accompanied by the vestibulocochlear nerve [20]. Cavazos et al. [22] have delineated that some fibers of the corticobulbar tract fibers descend ipsilaterally, and make a loop as caudally as the upper medulla before decussating and ascending to the contralateral facial nucleus. Another study reported that a large proportion of corticofacial fibers run within the ventromedial base of the pons and cross the midline at the level of the facial nucleus [23]. In our case, the P-FP may result from a pontine infarcted region involving the facial nucleus and/or facial nerve fascicles. The pons is supplied by the anterior inferior cerebellar artery (AICA) or basilar artery (BA) [24]. MRTA findings in our patient showed normal vessel morphology and blood flow in the main trunk of AICA and BA. Thus, the stroke mechanism might be a focal occlusion or stenosis of the pontine perforating vessel and/or small AICA branch.

P-FP diagnosis is mainly based on physical examination, complemented by diagnostic imaging and electrophysiological monitoring. Radiological examination is usually utilized to exclude otogenic and neoplastic factors, which cause facial palsy. The electrophysiological monitoring such as ENoG and EMG provide useful information about the degree of facial nerve injury or degeneration and guides subsequent treatment for patients with facial palsy. Some peripheral-type facial palsy patients obtain facial recovery after regular antiviral or other conservative treatments within 6 months after onset. However, there are still plenty of patients leaving with disfiguring faces, such as asymmetric mouth corner and incomplete eye closure. Surgical intervention like HFA may be appropriate for such kinds of patients at this moment. Over the past few decades, the surgically created hypoglossal-facial nerve communication was successfully applied to facial palsy patients. The procedure achieved reanimation of the facial nerve with a major side effect of tongue dysfunction due to the entire resection of the hypoglossal nerve [25]. Thus, researchers put forward a modified technique that an anastomosis was performed between the proximal stump of ansa cervicalis and the distal stump of the hypoglossal nerve after classical HFA to avoid morbidity related to tongue function [16,26]. However, there was an argument about the effectiveness of modified HFA in hemiglossal function preservation. For example, PITTY et al. [26] reported that the atrophy of the tongue was no less severe in patients who underwent ansa cervicalis (also called descendens hypoglossi)-hypoglossal distal stump anastomosis after classic HFA than those who only underwent classic HFA. Hammerschlag et al. [27] also denied the usefulness of ansa cervicalis-hypoglossal distal stump anastomosis in tongue function protection. However, a recent study by Won Young Koo et al. [16] showed that facial reanimation with hypoglossal nerve transfer, combined with hypoglossal nerve neurotization using the ansa cervicalis for complete facial palsy patients, might enable favorable facial restoration and reduce the incidence of tongue dysfunction. The Department of Neurosurgery at Xinhua Hospital believed that the advancement in surgical skills in recent years may improve the effectiveness of this

Table 1
Overview of the reported cases with peripheral-type facial palsy after pontine infarction.

Year	Reference	Gender/ Age	Lesion-localization	Side of facial palsy	Offending vessel	Management
1998	Eggenberger [30]	Male/46	pontine tegmentum	ipsilateral	small vessel occlusion	
1998	Eggenberger	Male/62	pontine tegmentum	ipsilateral	small vessel occlusion	
1999	Roh et al. [31]	Male/66	caudal tegmental pons	ipsilateral		
1999	Roh et al.	Male/61	caudal tegmental pons	ipsilateral		
2001	Utku et al. [28]	Female/ 43	bilateral pontine paramedian tegmentum	bilateral	left vertebral artery	antiaggregant and antihypertensive therapy
2007	Hayashi-Hayata et al. [32]	Male/62	mediolateral tegmental pons	ipsilateral		anti-platelet agent
2007	Hayashi-Hayata et al.	Male/51	mediolateral tegmental pons	ipsilateral		anti-platelet agent
2009	Felicio et al. [33]	Male/73	midline pontine tegmentum	ipsilateral		
2010	Ahn et al. [34]	Male/83	dorsolateral upper medulla and pontomedullary junction	ipsilateral		antiplatelet agents
2012	Han et al. [35]	Female/ 69	dorsolateral ponto-medullary junction	ipsilateral		
2015	Oh et al. [36]	Female/ 72	dorsal pons	ipsilateral		
2019	Cao et al. [37]	Female/ 79	bilateral caudal pontine plus paramedian pontine	right-sided	bilateral vertebral arteries and basilar artery	anticoagulation
2019	Min et al. [38]	Male/51	left pontomedullary junction	ipsilateral	large-artery atherosclerosis	
2019	Min et al.	Male/68	pontine tegmentum and pontomedullary junction	ipsilateral	large-artery atherosclerosis	
2019	Min et al.	Male/74	pontine tegmentum	ipsilateral	large-artery atherosclerosis	
2021	Zhuang et al. [29]	Male/55	bilateral dorsal pontine tegmentum	bilateral		
2022	Seah [39]	Male/36	dorsal pons	ipsilateral		anti-platelet agent

modified HFA in hemiglossal function preservation. Moreover, our preliminary study suggested that 8 of 10 cases undergoing this technique have shown a favorable facial function recovery decreasing from HB grade V to II or IIIs (data not shown). Impressively, only 2 cases showed a mild degree of tongue musculature atrophy with speech, swallowing and chewing unaffected during 7- or 8-month follow-up postoperatively. Hence, we decided to perform the modified HFA for the patient in our case. The operation was smooth and the patient was discharged routinely. It has been almost three months since the surgery. Videophone follow-up showed that the patient's facial palsy improved gradually, reaching HB grade IV currently, and follow-up will continue for this patient.

In addition, we also conducted a comprehensive literature search of the PubMed, Embase, and Cochrane Library databases and extracted all pons infarction-induced P-FPs reported in English (Table 1). 17 cases were retrieved in total. Utku and colleagues [28] described a 43-year-old woman who presented bilateral peripheral seventh nerve palsy. The patient had complaints of other neurological symptoms such as bilateral horizontal lateral gaze palsy, pointing to a pontine lesion. Subsequent cranial MRI demonstrated an ischemic lesion that expanded ventrally in the paramedian tegmental region at the mid-pontine level. Another study by Zhuang et al. [29]. reported a 55-year-old male who presented with bilateral P-FP caused by bilateral dorsal pontine tegmentum infarction. The residual 15 patients only manifested with unilateral P-FP. In all cases of P-FP (Table 1), a brain MRI and/or MRTA were performed to unveil the true etiology. And the reviewed literature attributed pons infarction to vertebral-basilar artery stenosis/occlusion, large-artery atherosclerosis, and small vessel occlusion.

This study had some noteworthy limitations. First, although rare, our case was not the first reported case of P-FP caused by a pons ischemic lesion. Second, only one case was reported in our paper, the sample size is small. In addition, only pons infarction-induced P-FP cases published in English were included in the literature review, which may result in the omission of some cases.

4. Conclusion

The present case suggested that isolated peripheral-type facial palsy may result from a well-placed small pons infarction. This reminded clinicians not to ignore the situation that central nervous system lesions hold a chance to cause peripheral-type facial palsy. In addition, modified HFA served as a useful skill improvement, and might be helpful in reducing hemiglossal dysfunction while restoring facial muscle function.

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Institutional review board statement

The study was reviewed and approved by the Human Ethics Committee of Xinhua Hospital and it conformed to the provisions of the Declaration of Helsinki.

Informed consent statement

Informed consent was obtained from the subject involved in the study.

Author contribution statement

All authors listed have significantly contributed to the investigation, development and writing of this article.

Data availability statement

Data will be made available on request.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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