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Case Study

Pure dysarthria in a young adult with deficit of the cerebral perfusion of the left frontal-parietal circuit

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Abstract
Dysarthria is one of neuromotor disorders that caused by deficits in varying factors required for speech expression with intact spoken language. There are five major types of dysarthria: flaccid, spastic, ataxic, hyperkinetic and hypokinetic types, and all causes of dysarthria can be resulted from trauma, stroke, degenerative neurologic disorders, or developmental disorders, but seldom in immaturation. We report a case of a 19-year-old man with dysarthria, whose scintigraphic findings showed moderately decreased uptake of the left frontal and left parietal lobes. We assumed that the cerebral perfusional deficit in the left frontal-parietal circuit might contribute to pure dysarthria. A detailed description of the circuit was discussed in the text.

Keywords: Pure dysarthria, Frontal-parietal circuit, Frontal lobe, Hypoperfusion, Development, Immaturity, Scintigraghic rehabilitation, Single-photon emission computed tomography

Introduction
Speech is a multifaceted neuromuscular complex achieved through the dexterit synchronization of five subsystems, e.g. breathing, phonation, tone, pronunciation, and prosody, which can be affected by muscular dysfunction to impaire audibility, spontaneity, precision, and communicative competence [1]. Dysarthria, one of neuromotor disorders, results from deficits in speed, but the matter of the verbal language stays undamaged; therefore, patients can still have the ability of writing and comprehending spoken language [1]. Pure dysarthria has been known as isolated dysarthria, which occurs owing to some congenital neurological deficits without stroke [2].

There are varying causes of pure dysarthria, including infection, trauma, toxicity, neglect and abuse, head injury, central auditory processing disorder, vascular disorder, neoplasm, degenerative neurologic disorders in children, degenerative neurologic disorders in adults, for instance, Parkinson's disease, supra-nuclear palsy, corticobasal degeneration, multiple system atrophy, ataxia telangiectasia, and Huntington disease [3]. However, it is rare to find in young adults without the above disorders.

We report a case of a young man with dysarthria, who did not have any history of stroke, or any disorders as mentioned above. According to the cerebral scintigraphic findings, he had moderately decreased uptake of the left frontal and left parietal lobes, which might be related to his dysarthria.

Case Present
A 19-year-old young adult came to our hospital on February 24th, 2024 for disability card at his first OPD. He was going to serve in the army. He has a history of hypertension, tinnitus of the right ear, bilateral flat feet, and also motion pain of bilateral fingers for many years. There was local tenderness of hand joints and knee joints on the right side. We noticed the way he talked unclearly and imprecisely since his childhood; his language disorder was confirmed as dysarthria under our expertise by using relevant veluation tool [4]. He also mentioned being sexually harassed when he was a child.

At OPD, X-ray films of bilateral hands and feet showed no visible lesion. The brain technetium-99m ethyl cysteine dimer (Tc99m-ECD) single-photon emission computed tomography (SPECT) was arranged to explore the cause of dysarthria. Because SPECT displays larger disparities in image quality than PET, conversion of SPECT images can be attributed by a standard database. One method for computerized diagnosis of cerebral perfusional SPECT was adopted, according to the Z-score imaging system (eZIS), in which easy eZIS software can incorporate a computerized program to share a standard database in SPECT studies [5]. SPECT and SPECT-CT images showed inhomogeneous uptake of the cerebral cortex with moderately decreased uptake over the left frontal and parietal lobes (scale: 0-1) (Figure, 1b) and minimally decreased uptake to the left basal ganglia (Figure 3). When the patient visited us again on March 9th, 2024, we told him the report and he got his disability card finally.

Figure 1a. Axial view of SPECT-CT Scan.

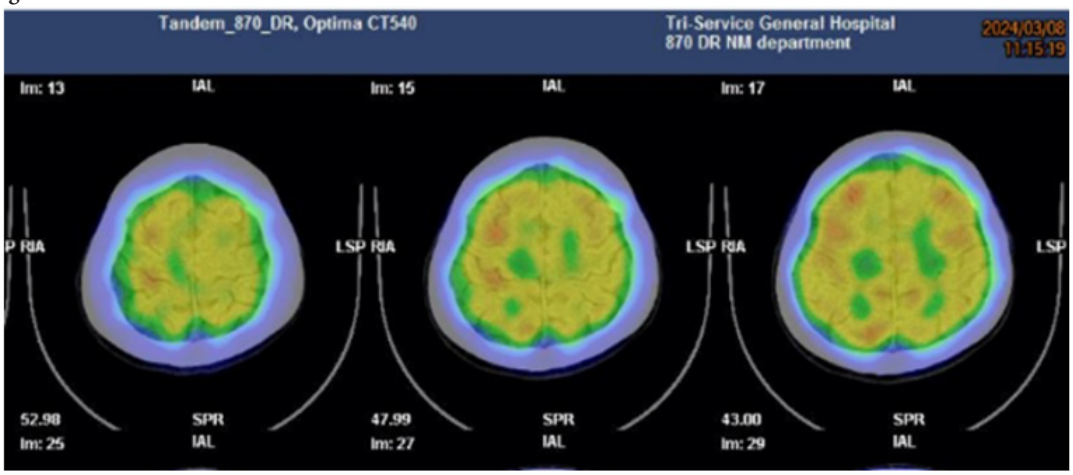

Figure 1b. The SPECT-CT shows the continuous transverse view from head to feet, finding inhomogeneous uptake of the cerebral cortex with mildly decreased uptake over the left frontal and parietal lobes.

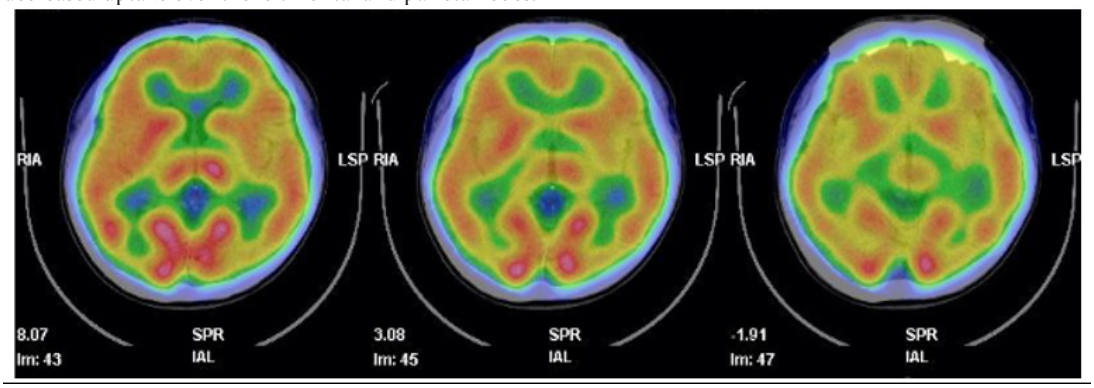

Figure 1. Axial view of SPECT-CT Scan.(1a, 1b) The SPECT-CT shows the continuous transverse view from head to feet, finding inhomogeneous uptake of the cerebral cortex with mildly decreased uptake over the left frontal and parietal lobes.

Figure 2. axial view- 26,27,28 upper panel
Figure 2. coronal view – 18, 19, 20, lower panel

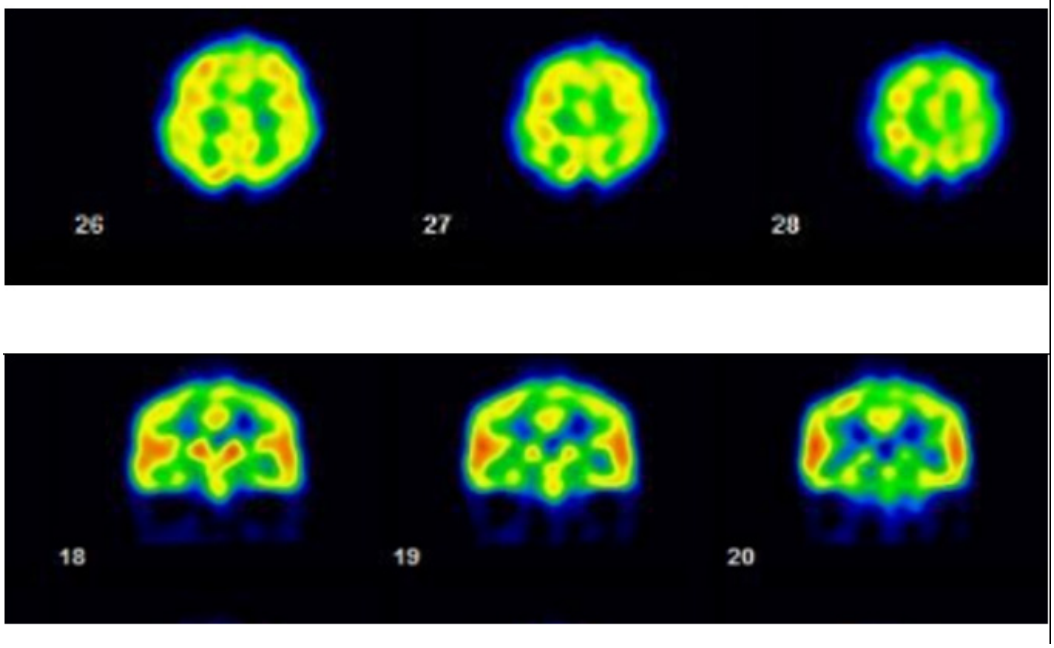

Figure 2. Brain technetium-99m ethyl cysteine dimer single photon emission computed tomography (SPECT).Inhomogeneous uptake of the cerebral cortex with mildly decreased uptake over the frontal lobe cortices. The left figure shows an axial view (No. 26,27,28); the right one shows a coronal view (No. 18, 19, 20)

Figure 3. Axial view of SPECT-CT Scan .Inhomogeneous uptake of the basal ganglion with decreased uptake over the left globus pallidus.

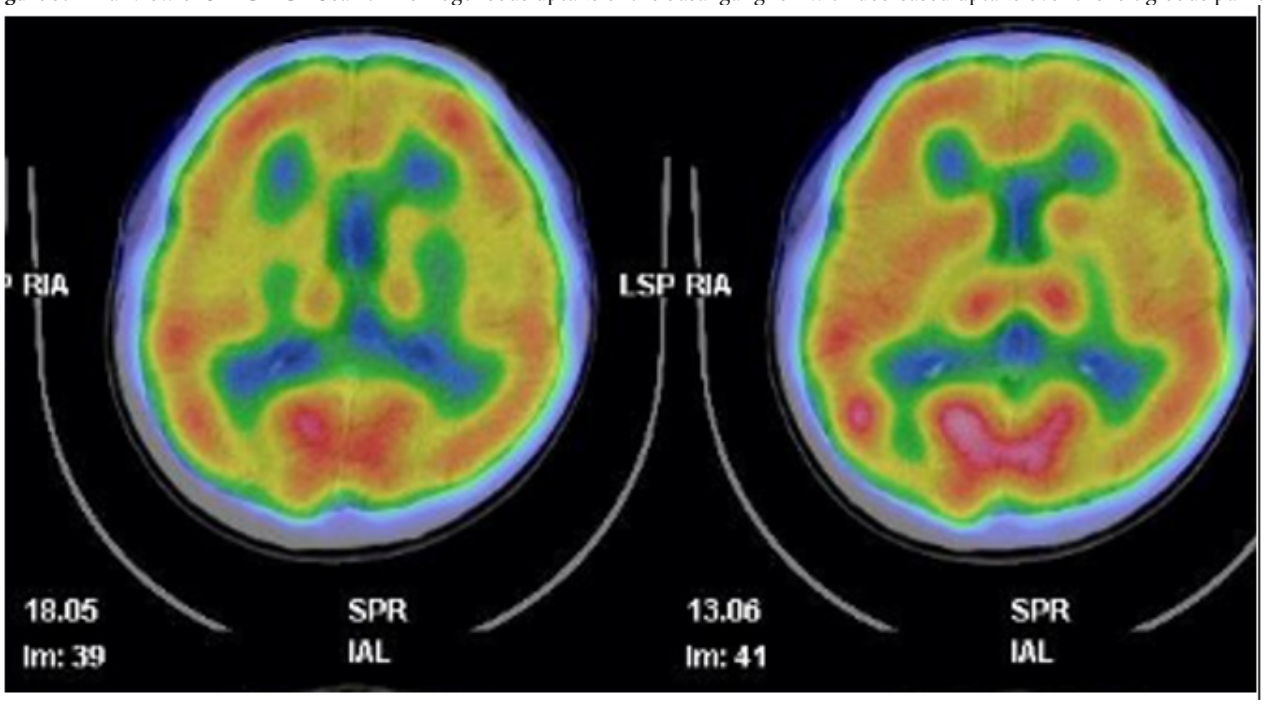

Figure 4. The easy Z-score imaging system (eZIS). It shows a low Z-score in the posterior cingulate gyrus with unclear reason.

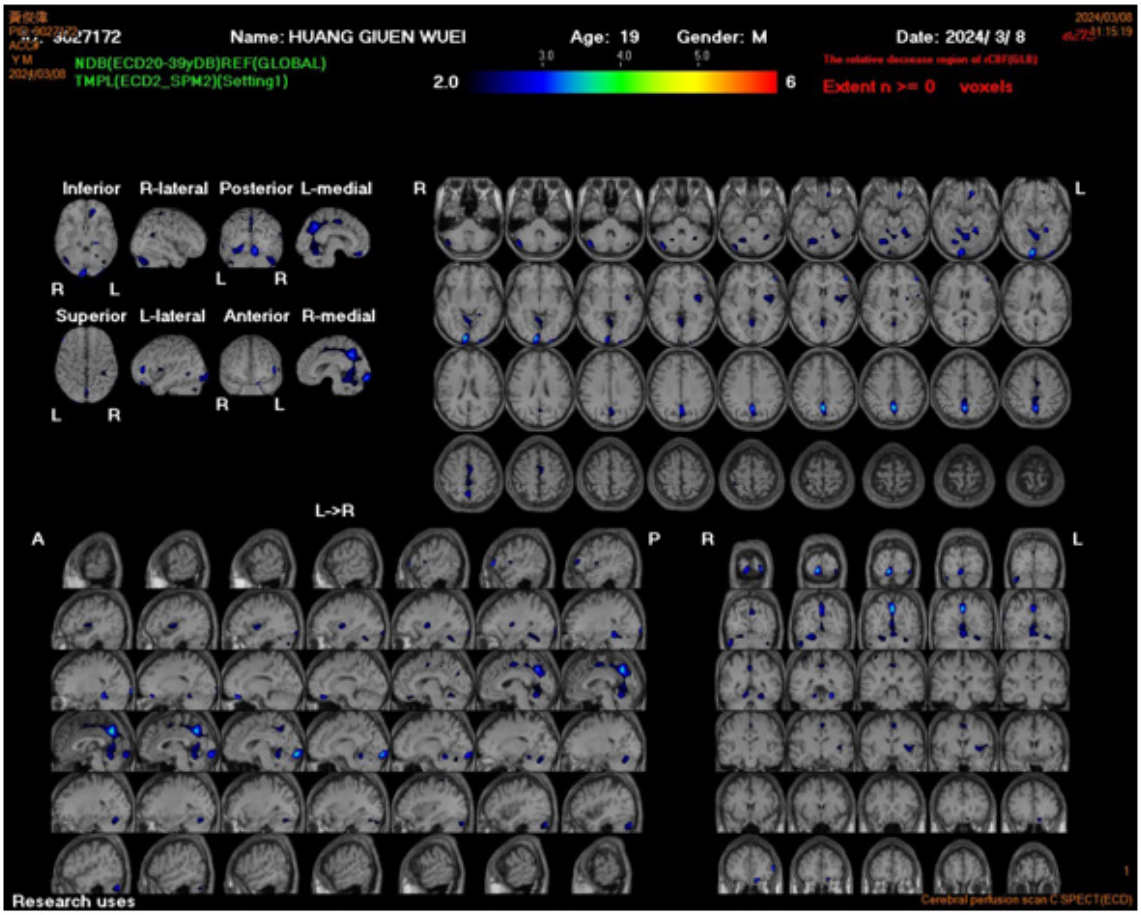

Figure 4. The easy Z-score imaging system (eZIS). A method for automated diagnosis of brain perfusion SPECT, an easy Z-score imaging system (eZIS). Since SPECT exhibits greater variations in image quality among different centers than PET, conversion of SPECT images may be necessary for sharing a normal database. In our case, it shows a low Z-score in the posterior cingulate gyrus with unclear reason.

Discussion

Pure dysarthria, also called isolated dysarthria, develops rarely after a stroke, and even without a stroke [2]. Our patient experienced pure dysarthria since childhood without a history of stroke or any other neurologic disorders. Brain perfusion scintigraphy showed moderately decreased uptake of the left frontal and left parietal lobes. To our knowledge, this is the first case to present pure dysarthria in a young adult since childhood occurred without a definite known etiology, which disorder is highly related to hypoperfusion of the left frontal lobe and left parietal lobes.

There are five types of dysarthria defined by Mayo Clinic Classification (commonly used - groups dysarthria based on the location), for instance, hyperkinetic type, hypokinetic type, ataxic type (dysfunction in cerebellum), flaccid type (lower motor neuron) , and spastic type (upper motor neurons linked to the cerebral motor cortex) [6]. Our case does not belong to any one of the five types above.

There are two major categories for describing childhood dysarthria, developmental language impairment and specific language impairment. The former means various developmental disorders together with impairment in cognition, speech and language, and the latter is a developmental disorder that occurs with dysarthria only and without disable intellecture, hearing failure, neurologic/motor disorders, or socio-emotional dysfunction [7]. Our case was similar to the latter, and the literature has demonstrated meager language development in kids without other developmental or sensory disorders being mostly attributed to genetic factors [7].

The pathophysiology of pure dysarthria remains unclear. Hypoperfusion of the cerebral cortex might play a significant role. A previous article mentioned that congenital dysphasia might be caused by developmental language disorders of unknown origin, and children suffering from congenital dysphasia can have regional cerebral blood flow hypoperfusion involving language areas (such as the inferior frontal lobe and temporoparietal area) [8]. The results of images of our case were similar, even though our case is pure dysarthria. A study by Okuda et al [2] investigated the relationship between lesion locations and cerebral blood flow in those with pure dysarthria and found that pure dysarthria is highly associated with cortical hypoperfusion in the frontal lobe, probably due to disruption of cortico-subcortical networks after subclinical infarctions, and they concluded that hypoperfusion of frontal cortexl, principally in the anterior opercular and medial frontal areas, plays an significant role in the progress of pure dysarthria [2]. Most of the cases with bilateral infarctions of the internal capsule (IC) or corona radiate (CR) raised the fact that IC-CR lesions persuade pure dysarthria more frequently in bilateral than unilateral. The scintigraphic findings of our case fit their conclusion that pure dysarthria might be caused by lesions involving the IC or CR, but we only found lesions on the unilateral side in brain SPECT images, e.g. left frontal and parietal lobes.

The vascular mechanism has been proposed as a likely reason accounting for such a cortical hypoperfusion in cerebral regions [2]. In addition, an alternative reason terms “diaschisis,” in which reduction of neural activities in a specific part of the brain can be caused by disruption of neural networks from a remote lesion (white matter lesions may interrupt cortico-subcortical links unnessasory for speech output) [2]. We believe the fact is attributed to white matter lesions, but not enough information to apply in our case.

Talking about the evolutionarily ancient "frontal-parietal circuit", there are some articles to discuss its importance. During religious narration, self-identified religious individuals activated a frontal-parietal circuit, consisting of the dorsolateral prefrontal, dorsomedial frontal, and medial parietal gyri [9]. A frontal-parietal circuit largely tuned to various magnitude aspects affords the phylogenetic substrate for spatial-numerical correlations, while enculturation and sensorimotor experience shape their unambiguous silhouettes [10]. The body ownership provoked by the rubber hand illusion has been associated with a neural complex involving a frontal-parietal circuit, and the parietal cortex plays a essential role in the rapidity of visual and perceptible multi-sensory integration in the illusion [11]. In the study of Intentional forgetting (IF), there are two networks highly related to the IF, one is the "core regions" that are composed of a mainly lateralization of frontal-parietal circuit in the right side and the other is "supportive network" involving frontal-hippocampal connections [12]. People with mild cognitive impairment always have inferior intertemporal decision-making competence, which has confirmed as impairment of executive function due to degeneration of frontal-parietal circuit [13].

Dysarthria has been demonstrated highly associated with developmental disorders caused by brain damage during perinatal stage or acquired later in life associated with stroke, traumatic brain injury, or progressive neurologic disorder(s) [14], but never describing the factor of maturation. Immature hypoperfusion of the frontal and parietal lobes might be the underlying reason account for pure dysarthria. The factor of maturation plays a crucial role in the development of brain, and hypoperfusion caused by immaturation may direct to spastic dysarthria commonly involving vulnerable corticospinal/corticobulbar tracts, for instance, the cortical motor areas (chiefly the precentral gyrus and premotor cortex), the internal capsule, or the cerebral peduncles of the midbrain [14]. Besides, innervation to most cranial nerves nuclei with bilateral effects has clinical significance in speech disorder, that is, bilateral corticobulbar pathologies will create a more severe and permanent dysarthria than a unilateral lesion presenting with mild and transient symptom [14]. Our case shows unilateral hypoperfusion in the left frontal lobe, compatible with this article.
Concerning the mechanism of pure dysarthria, we consider it might be highly related to the maturation which is not enough in the frontal and parietal lobe and basal ganglion. A recent article confirmed that genes might cause immaturation, e.g. the FOXP1 (Forehead Box P1) gene, which contributes to developmental speech disorders with having common characteristics of apraxia and dysarthria, such as motor programming dysfunction, and speech influency, and whose people with missense variants always have communicative deficits [15]. The etiology of dysarthria is still unclear, we may need more advanced tools to investigate the causes of developmental lesions, like internal capsule and corona radiate. Without a clear pathophysiological explanation, there is much space for scholars to realize.

Brain ECD-SPECT acts an astonishing clinical tool in varying disorders for assessment of neural phenomenon corresponding with brain lesion(s) [16-19] and observation of outcome/progression after relevant therapy [20-28].

Conclusion

In conclusion, hypoperfusion of the frontal and parietal lobes is highly related to pure dysarthria, like our case. The left frontal-parietal circuit might engage in recreation regarding language function. Immaturation of the cerebral cortex (frontal and parietal lobes) might contribute to the occurrence of pure dysarthria. Brain SPECT study is very useful in the field of scintigraghic rehabilitation.

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