

Occurrence of Subtle Scoliosis in Accordance with Development of Ipsilateral Cerebellar Diaschisis on Brain SPECT in a Patient with Head Injury

Shin-Tsu Chang¹, Yu-Ming Fan²

¹*Department of Physical Medicine and Rehabilitation, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan, ROC*

²*Department of Nuclear Medicine, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan, ROC*

A 23-year-old male patient with a head injury 20 months ago visited our OPD on account of shortening height from 167 cm to 165 cm within a 2-month period. Scoliosis series of the whole spine X-rays revealed a slight convex in the left side with a 4-degree curve of scoliosis measured by the Cobb's method. Brain SPECT with ^{99m}Tc-HMPAO images showed a reduction of perfusion to the left cerebellar hemisphere, e.g. ipsilateral cerebellar diaschisis (ICD), because the left cerebral cortex had severely wronged including the left frontal, temporal and parietal-occipital regions. We speculated that the occurrence of scoliosis together with impaired cerebellar perfusion following head injury might be associated with damage to the efferent fibers from the cerebellum and the ICD observed in the chronic stage of brain damage probably was caused by a late involvement of the cortico-ponto- cerebellar tract at the level of the frontal cortex.

Key words: head injury, scoliosis, brain SPECT, ipsilateral cerebellar diaschisis

Ann Nucl Med Sci 2005;18:173-179

Diaschisis refers to a functional impairment at a remote site following injury to an anatomically connected area of brain and, presumably due to a loss of afferent input to the remote site [1]. Of the variable type of diaschisis, the crossed cerebellar diaschisis (CCD) defined as a diminution in metabolic rate for oxygen and cerebral blood flow in the cerebellar hemisphere contralateral to a destructive cerebral lesion has been interesting and focusing issue in the past 20 years [2]. Baron and his colleagues [3,4] were the first group to demonstrate CCD in patients with cerebral infarcts by using positron emission tomography (PET) and established the concept of CCD following injury (usually vascular damage) to the motor cortex of one cerebral hemisphere. Since then, adoptions of single-photon emission computed tomography (SPECT) or PET studies have concluded that lesions in a variety of cortical areas could cause CCD [5-16].

Apart from the CCD, Hamano et al. [17] demonstrated the second type of cerebellar diaschisis: cerebellar hypoperfusion ipsilateral to supratentorial lesions, ipsilateral cerebellar diaschisis (ICD), in childhood in 1993. They undertook the important retrospective investigation of cerebellar diaschisis in 55 hemiplegic children to understand the functional maturation of the cortico-ponto-cerebellar tract in developing brain by using brain SPECT with ¹²³I-IMP and found CCD in 6 of 55 patients and ICD in 10 of 55 patients. The CCD occurred in the patients who suffered from brain injuries after 7 years, 5 months of age, while ICD manifested in patients whose brain injuries occurred before 3 years, 1 month of age. They concluded that the production of remote effects, such as CCD and ICD, could be closely related to

Received 10/6/2004; revised 12/2/2004; accepted 12/9/2004.
For correspondence or reprints contact: Shin-Tsu Chang, M.D., Departments of Physical Medicine and Rehabilitation, Tri-Service General Hospital, 325 Section 2, Cheng-Kong Road, Taipei 114, Taiwan, ROC. Tel: (886)2-87923311 ext. 13325, Fax: (886)2-26321954, E-mail: doc31116@yahoo.com.tw

maturation of the cortico-ponto-cerebellar tract in the developing brain during childhood.

However, to our knowledge, none of the previous study showed scoliosis associated with cerebellar diaschisis. The majority of the ignorance about any cerebellar component in the clinical evaluation might be masked by a significant motor deficit, spasticity or absence of evaluation battery [18]. In this report, we present a patient with subtle scoliosis on x-rays and ICD on brain SPECT and speculate that scoliosis and ICD were secondary to injury to the homolateral cerebral hemisphere.

Case Report

A 23 year-old male visited our OPD on account of shortening height from 167 cm to 165 cm within a 2-month period. Traced the history, he had a traffic accident with head injury and received a skull operation to treat left subdural hemorrhage at a local hospital on January 10, 2003. Three days later (January 13, 2003), he was transferred to our hospital for managing fracture in the left ulna and femur bones in the condition of unconsciousness that persisted for more than one month (post-traumatic coma). In May 2003, he developed a progressive weakness of the left limbs and difficulty in walking and first called at our OPD for help. In recent 2 months, he and his friends found that his height became short gradually, he thus visited us again.

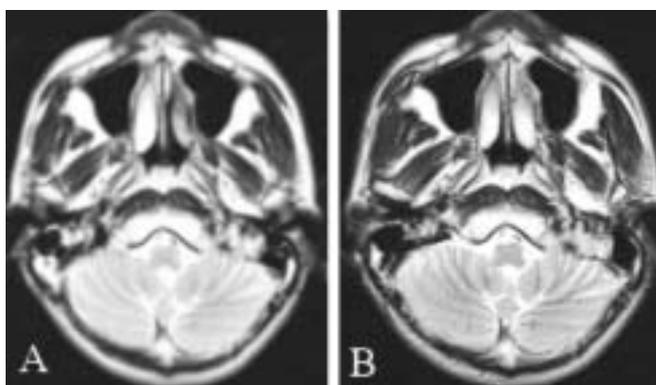


Figure 1. T2-weighted MR images of the brain obtained on May 5, 2004 (A) and on October 26, 2004 (B). In (A), a mild gliosis of the left medial temporal/occipital lobe and that may be due to a prior PCA territory infarction, but no abnormalities in the cerebellum on the both sides. In (B), no abnormalities in the cerebral cortex and cerebellum of both sides are demonstrated.

On physical examinations, he revealed a right-sided spastic hemiparesis. The muscle powers were 5 in the right limbs and 3-4 in the left limbs. Deep tendon reflex showed bilateral hyperreflexia with more prominent in the left side. Obvious palm-mental sign was showing in the left side. He had neither dysphagia nor uninhibited neurogenic bladder, except mild memory impairment and dysarthria. His balance tests showed poor Romberg test and poor dynamic testing. Mild hump was seen over the left side while bending the trunk. There was no length discrepancy in the lower extremities.

The previous magnetic resonance (MR) images obtained on May 5, 2004 showed a mild gliosis in the medial temporal/ occipital lobe of the left cerebral hemisphere but no abnormalities in the right cerebral hemisphere and both cerebellum (Figure 1A). The current MR images of the brain obtained on October 26, 2004 showed no abnormality in both cerebral and both cerebellum hemisphere (Figure 1B). Brain SPECT using ^{99m}Tc -hexamethylpropyleneamine oxime (^{99m}Tc -HMPAO) images obtaining on May 10, 2004 showed inhomogeneous perfusion in the cerebral cortex with decreased uptake in both frontal, left fronto-temporal regions, as well as left occipital lobe. The cerebellum looks like normal (Figure 2A). A second SPECT study performed on October 4, 2004 showed inhomogeneous perfusion in the cerebral cortex with relative decreased uptake in the left hemisphere, including the left frontal, temporal and left parietal-occipital regions. However, the left cerebellum indicated relatively decreased uptake (Figure 2B). The scoliosis series of the whole spine X-rays revealed a slight convex in the left side with a 4-degree curve of scoliosis measured by the Cobb's method (Figure 3).

Discussion

Diaschisis is a pathophysiological phenomenon based on reversible depression of functions anatomically or functionally connected to the damaged area, which has become a more interesting issue over the past twenty years due to advances made in neuroimaging techniques and their possible participation in the neural plasticity.

Contralateral cerebellar blood flow reduction, i.e., crossed cerebellar diaschisis (CCD), has also been studied in

adults with hemispheric brain injury by functional neuroimaging (such as SPECT) in various conditions including ischemic cerebrovascular disease [5-8,14,16,19,20], supratentorial diseases [21], cerebral hematomas [9] and head injury [10,15]. The latest study of association of CCD between dichotomized and continuous analyses published by Komaba et al., who found that hypoperfusion of postcentral and supramarginal regions independently influences CCD and suggested that location of a lesion, not severity, is the main determinant of CCD [22]. In a study to determine whether measurements of CCD in the acute and subacute stages facilitate the prediction of stroke outcome, Takasawa et al. concluded that cerebellar hypoperfusion detected on ^{99m}Tc-HMPAO SPECT in the early subacute stage in patients with supratentorial infarct indicates a more sinister clinical

outcome [23].

Since the second type of cerebellar diaschisis, e.g. the ICD, was first published by Hamano et al. [17] who studied the SPECT images in childhood, some researchers noticed that apart from the CCD, ICD took place in the similar diseases but was rare. In a study of regional cerebral blood perfusion in ^{99m}Tc-HMPAO SPECT in 14 spastic hemiplegic children with hemiplegia of various etiology, including 7 cases with cerebral palsy (five with porencephalic cyst), 2 cases with stroke, 3 cases with hemiconvulsion-hemiplegia-epilepsy syndrome, and 2 cases with traumatic brain injury, 5 children had ICD, who had early cerebral insult due to porencephalic cyst of pre- or perinatal onset [15]. In a recent published paper with functional neuroimaging studies and MR imaging done later in life, three patients with infantile

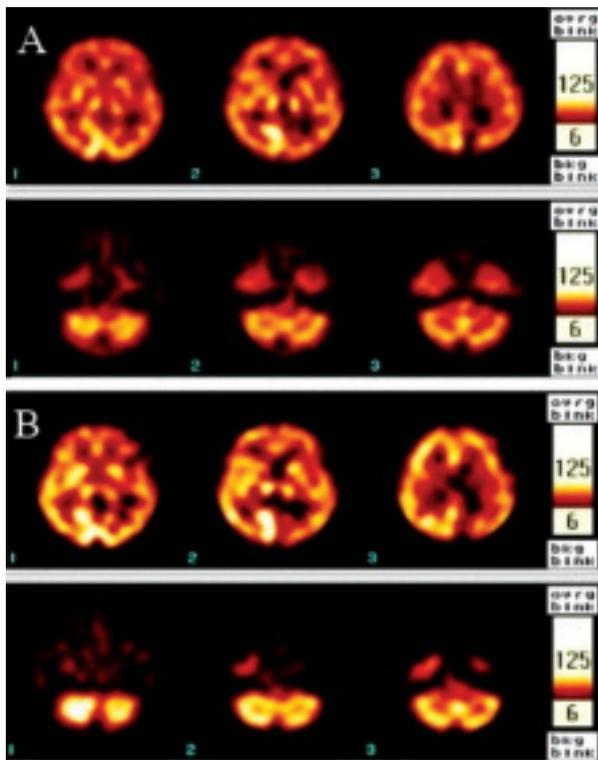


Figure 2. Brain SPECT with ^{99m}Tc-HMPAO images obtained on May 10, 2004 (A) and on October 4, 2004 (B). In (A), inhomogeneous perfusion in the cerebral cortex with decreased uptake of tracer in both frontal, left fronto-temporal region, as well as left occipital lobe (upper). The cerebellum looks like normal (lower). In (B), inhomogeneous perfusion in the cerebral cortex with relative decreased uptake in left hemisphere, including the left frontal, temporal and left parietal-occipital regions (upper). However, the left cerebellum shows relatively decreased uptake (lower).



Figure 3. The scoliosis series of the whole spine of the patient with head injury. There is only a 4-degree curve of scoliosis using Cobb's method with a slight convex in the left side.

hemiplegia syndrome showed significant volume loss in the cerebellar hemisphere, contralateral to the side of the affected cerebrum in two and ipsilateral in one, observations which provide morphological evidence of the phenomenon of CCD and ICD [24].

Tsuda et al. [25] introduced six consecutive aged patients with infratentorial pontine infarction exhibiting mild hemiparetic symptoms or a heavy feeling in the head to evaluate regional cerebral and cerebellar perfusion by SPECT and to calculate asymmetry index. They found that significant asymmetry in cerebellar perfusion was reduced in the contralateral ($n = 4$) or ipsilateral ($n = 1$) cerebellar hemisphere as compared with normal controls. The most important study of the phenomenon of CCD and ICD in head injury was published by Alavi et al. [10] who compared fluorine-18-fluorodeoxyglucose PET images to CT or MR images in 19 patients with head injury. They found that 19% were associated with ICD and 40% were associated with CCD in a total of 68 focal unilateral brain lesions, but of patients with both focal and diffuse brain injuries, ICD and CCD share the same (27%) percentile. More detailed analysis showed ICD of 8% and 13% in the cortical lesion and focal supratentorial lesion compared with CCD of 69% and 50%, respectively. Compared with extraparenchymal and intraparenchymal lesions, ICD of 15% and 21% compared with CCD of 45% and 38%, respectively. Alavi et al. [10] concluded that ICD is seen less often than CCD in patients with focal cortical or extraparenchymal injuries.

Many articles discussing CCD showed that the mechanism is a secondary effect after neural functional deactivation, presumably resulting from a loss of excitatory or inhibitory afferent inputs on the cortico-ponto-cerebellar pathway [26-28] or a retrograde deactivation of the cerebellar hemisphere via the dentato-rubro-thalamo-cortical pathway [29]. Our patient did not have morphological lesions in the right cerebral cortical areas revealed through MR imaging, although some researchers mentioned the cerebellar diaschisis related to the motor and premotor area [30]. Our results are in accordance with the suggestions of Lim and his colleagues who emphasized that cerebellar diaschisis can develop in patients with subcortical hematoma without, not with, cortical structural abnormality [9].

There were few articles discussing the symptoms or signs occurring with CCD, and the ICD was far and away the least. A previous study describing CCD accompanied by hemiataxia in patients with single unilateral supratentorial lesion [29], with findings indicating that the ataxia may depend on the lesions of the cortico-ponto-cerebellar tracts. Another case of hemiataxia after thalamic hemorrhage reported by Engelborghs et al. also demonstrated CCD [31]. Kastenbauer et al. found that the CCD may be an inkling to the mechanism of ataxic hemiparesis in Creutzfeldt-Jakob disease [32]. In contrast, Kim et al. found that the frequency of CCD is significantly higher in patients whose infarctions were in the fronto-parietal lobes or the deep middle cerebral artery territory than in patients whose infarctions were in other regions; nevertheless, they concluded that none of the patients with CCD demonstrated the apparent clinical signs of cerebellar dysfunction [18]. De Reuck et al. also found that CCD appears to be correlated to the lack of significant clinical improvement and suggested that the persistence of CCD has no real clinical significance [33].

To our knowledge, none of the previous studies have mentioned scoliosis changes observed as a result of the ICD. The aforementioned case can be taken as an example of head injury with left spastic hemiparesis, who developed scoliosis as a result of ICD in the second SPECT study, even without abnormal findings in the cerebellum on both sides from MR images as well as the first SPECT study. The present report appears to be significant that evidence of ICD detected on SPECT could be correlated with occurrence of scoliosis. We propose that each cerebellar hemisphere is closely linked to the cerebral cortex on both sides through neural feedback circuits, which is needed for smooth execution of motor function under an intact musculoskeletal system. So if any problem happens in the cerebellum, posture will be altered. That's why our case developed scoliosis in late chronic stage. We speculated the pathophysiological mechanism of the ICD might be related to the perfusion defect in the cerebral cortex resulting from head injury, which inhibited the cerebellar metabolism through the crossed or uncrossed/ipsilateral cortico-ponto-cerebellar pathway. In the meantime, we act in response to the statement of Alavi et al. [10] who concluded that a predominance of CCD is more pronounced

with lesions of the greatest severity in patients with head injury, not the ICD; therefore, our case should have a better prognosis in his future.

References

1. Shamoto H, Chugani HT. Glucose metabolism in the human cerebellum: an analysis of crossed cerebellar diaschisis in children with unilateral cerebral injury. *J Child Neurol* 1997;12:407-414.
2. Reivich M. Crossed cerebellar diaschisis. *AJNR Am J Neuroradiol* 1992;13:62-64.
3. Baron JC, Bousser MG, Comar D, et al. 'Crossed cerebellar diaschisis' in human supratentorial brain infarction. *Trans Am Neurol Assoc* 1980;105:459-461.
4. Baron JC, Bousser MG, Comar D, Dequesnoy N, Saatre J, Castaigne P. "Crossed cerebellar diaschisis": a remote functional suppression secondary to supratentorial infarction in man. *J Cereb Blood Flow Metab* 1981;1(Suppl 1):S500.
5. Pantano P, Baron JC, Samson Y, Bousser MG, Derouesne C, Comar D. Crossed cerebellar diaschisis: further studies. *Brain* 1986;109:677-694.
6. Yamauchi H, Fukuyama H, Nagahama Y, Okazawa H, Konishi J. A decrease in regional cerebral blood volume and hematocrit in crossed cerebellar diaschisis. *Stroke* 1999;30:1429-1431.
7. Gonzalez-Aguado E, Marti-Fabregas J, Marti-Vilalta JL. The phenomenon of diaschisis in cerebral vascular disease. *Rev Neurol* 2000; 30: 941-945. [In Spanish].
8. Kim SE, Lee MC. Cerebellar vasoreactivity in stroke patients with crossed cerebellar diaschisis assessed by acetazolamide and ^{99m}Tc-HMPAO SPECT. *J Nucl Med* 2000;41:416-420.
9. Lim JS, Ryu YH, Kim BM, Lee JD. Crossed cerebellar diaschisis due to intracranial hematoma in basal ganglia or thalamus. *J Nucl Med* 1998;39:2044-2047.
10. Alavi A, Mirot A, Newberg A, et al. Fluorine-18-FDG evaluation of crossed cerebellar diaschisis in head injury. *J Nucl Med* 1997;38:1717-1720.
11. Pantano P, Lenzi GL, Guidetti B, et al. Crossed cerebellar diaschisis in patients with cerebral ischemia assessed by SPECT and ¹²³I-HIPDM. *Eur Neurol* 1987;27:142-148.
12. Brott TG, Gelfand MJ, Williams CC, Spilker JA, Hertzberg VS. Frequency and patterns of abnormality detected by iodine-123 amine emission CT after cerebral infarction. *Radiology* 1986;158:729-734.
13. Baron JC. Crossed cerebellar diaschisis related to recurrent focal seizures. *Epilepsia* 1996;37:417-418.
14. Baird AE, Donnan GA, Austin M, Newton MR, McKay WJ. Preliminary experience with ^{99m}Tc-HMPAO SPECT in cerebral ischaemia. *Clin Exp Neurol* 1991;28:43-49.
15. Sztrihai L, al Suhaili AR, Prais V, Nork M. Regional cerebral blood perfusion in children with hemiplegia: a SPECT study. *Neuropediatrics* 1996;27:178-183.
16. Miyazawa N, Toyama K, Arbab AS, Koizumi K, Arai T, Nukui H. Evaluation of crossed cerebellar diaschisis in 30 patients with major cerebral artery occlusion by means of quantitative I-123 IMP SPECT. *Ann Nucl Med* 2001;15:513-519.
17. Hamano S, Nara T, Nakanishi Y, Horita H, Kumagai K, Maekawa K. Secondary changes in cerebellar perfusion (diaschisis) in hemiplegia during childhood: SPECT study of 55 children. *Pediatr Neurol* 1993;9:435-443.
18. Kim SE, Choi CW, Yoon BW, et al. Crossed-cerebellar diaschisis in cerebral infarction: technetium-99m-HMPAO SPECT and MRI. *J Nucl Med* 1997;38:14-19.
19. Lin WY, Kao CH, Wang PY, Changlai SP, Wang SJ. Serial changes in regional blood flow in the cerebrum and cerebellum of stroke patients imaged by ^{99m}Tc-HMPAO SPET. *Nucl Med Commun* 1996;17:208-211.
20. Muller V, Saur D, Klutmann S, Weiller C, Rother J, Clausen M. Experience with ¹²³I-iodozepam SPECT in acute cerebral infarction. *Nucl Med Commun* 2002;23:1191-1196.
21. Tien RD, Ashdown BC. Crossed cerebellar diaschisis and crossed cerebellar atrophy: correlation of MR findings, clinical symptoms, and supratentorial diseases in 26 patients. *AJR Am J Roentgenol* 1992;158:1155-1159.
22. Komaba Y, Mishina M, Utsumi K, Katayama Y, Kobayashi S, Mori O. Crossed cerebellar diaschisis in patients with cortical infarction: logistic regression analysis to control for confounding effects. *Stroke* 2004;35:472-476.

23. Takasawa M, Watanabe M, Yamamoto S, et al. Prognostic value of subacute crossed cerebellar diaschisis: single-photon emission CT study in patients with middle cerebral artery territory infarct. *AJNR Am J Neuroradiol* 2002;23:189-193.
24. Chakravarty A. MR evaluation of crossed and uncrossed cerebral-cerebellar diaschisis. *Acta Neurol Scand* 2003;108:60-65.
25. Tsuda Y, Ayada Y, Izumi Y, et al. Cerebellar diaschisis in pontine infarctions: a report of five cases. *Eur J Nucl Med* 1995;22:413-418.
26. Miura H, Nagata K, Hirata Y, Satoh Y, Watahiki Y, Hatazawa J. Evolution of crossed cerebellar diaschisis in middle cerebral artery infarction. *J Neuroimaging* 1994;4:91-96.
27. Ito H, Kanno I, Shimosegawa E, Tamura H, Okane K, Hatazawa J. Hemodynamic changes during neural deactivation in human brain: a positron emission tomography study of crossed cerebellar diaschisis. *Ann Nucl Med* 2002;16:249-254.
28. Yamauchi H, Fukuyama H, Kimura J. Hemodynamic and metabolic changes in crossed cerebellar hypoperfusion. *Stroke* 1992;23:855-860.
29. Tanaka M, Kondo S, Hirai S, Ishiguro K, Ishihara T, Morimatsu M. Crossed cerebellar diaschisis accompanied by hemiataxia: a PET study. *J Neurol Neurosurg Psychiatry* 1992;55:121-125.
30. Ishihara M, Kumita S, Mizumura S, Kumazaki T. Crossed cerebellar diaschisis: the role of motor and premotor areas in functional connections. *J Neuroimaging* 1999;9:30-93.
31. Engelborghs S, Pickut BA, Marien P, Opsomer F, De Deyn PP. Crossed cerebellar diaschisis and hemiataxia after thalamic hemorrhage. *J Neurol* 2000;247:476-477.
32. Kastenbauer S, Schulz-Schaeffer WJ, Tatsch K, Yousry TA, Kretschmar HA, Pfister HW. Crossed cerebellar diaschisis: a clue to the mechanism of ataxic hemiparesis in Creutzfeldt-Jakob disease? *J Neurol* 2001;248:1093-1095.
33. De Reuck J, Decoo D, Lemahieu I, Strijckmans K, Goethals P, Van Maele G. Crossed cerebellar diaschisis after middle cerebral artery infarction. *Clin Neurol Neurosurg* 1997;99:11-16.

頭部外傷患者脊椎側彎併發腦造影呈現同側小腦失聯現象

張幸初¹ 樊裕明²

¹國防醫學院 三軍總醫院 復健醫學部

²國防醫學院 三軍總醫院 核子醫學部

一位23歲，二十個月前曾有頭部外傷的男性患者因自覺身高變矮而前來就診，脊椎側彎系列(scoliosis series) X光檢查發現胸-腰椎輕度向左側彎四度 (Cobb's method 測量)，核醫腦單光子射出電腦斷層造影呈現左側小腦放射活性訊號減弱，是為同側小腦失聯絡現象 (ICD)。此現象在以往的文獻中並未被提及，且由於出現脊椎側彎而致身高變化的時間點與ICD發生的時間吻合，故推測此兩者具有很強的關聯性。

關鍵詞：頭部外傷，脊椎側彎，腦單光子斷層造影，小腦失聯絡

核子醫誌2005;18:173-179

93年10月6日受理 93年12月2日修改 93年12月9日接受刊載

聯絡人：張幸初醫師 台北市114內湖區成功路二段325號 三軍總醫院復健醫學部 電話：(02)-87923311轉13325 傳真：(02)-26321954 電子信箱：doc31116@yahoo.com.tw