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Fluorine-18-FDG Evaluation of Crossed Cerebellar Diaschisis in Head Injury

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This study investigates the phenomenon of crossed cerebellar diaschisis in head injury patients. **Methods:** We visually compared fluorine-18-fluorodeoxyglucose (FDG)-PET images to radiograph computed tomography or magnetic resonance images in 19 patients with head injury. **Results:** We found that of 68 focal unilateral lesions, 40% were associated with contralateral cerebellar hypometabolism and 19% were associated with ipsilateral cerebellar hypometabolism. Of supratentorial, extraparenchymal lesions (n = 20), 45% were associated with contralateral cerebellar hypometabolism, whereas 15% had ipsilateral cerebellar hypometabolism. Intraparenchymal lesions were associated with contralateral cerebellar hypometabolism in 38% of the patients and with ipsilateral cerebellar hypometabolism in 21% of the patients. Of the cortical lesions that were the patients' most severe injury, 69% were associated with contralateral cerebellar hypometabolism, whereas only 8% were associated with ipsilateral cerebellar hypometabolism. In patients with focal supratentorial lesions alone, 50% of all focal lesions were associated with contralateral cerebellar hypometabolism and 13% had ipsilateral hypometabolism. Of patients with both focal and diffuse brain injuries, 27% of the focal lesions had contralateral cerebellar hypometabolism and 27% had ipsilateral cerebellar hypometabolism to the most severe focal injury. **Conclusion:** Crossed cerebellar diaschisis is seen more often in patients with focal cortical or extraparenchymal injuries and is not seen in patients with multiple or diffuse brain injuries. Furthermore, this predominance is more pronounced with lesions of the greatest severity.

Key Words: PET; head injury; cerebellar diaschisis

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Since the advent of functional brain imaging, several studies using PET and SPECT have shown that lesions such as infarction and tumor in the cerebral cortex are associated with contralateral cerebellar hypometabolism or hypoperfusion (*1–9*). The evidence to support a similar pattern secondary to head trauma has been lacking in the literature. Studies have reported changes in regional blood flow and metabolism in areas remote from the original site of injury. We have reported both ipsilateral and contralateral cerebellar hypometabolism as the result of head trauma (*10,11*). This study reports a comprehensive analysis of fluorine-18-fluorodeoxyglucose (FDG)-PET studies performed on head trauma patients. Specifically, we have determined the presence and laterality of cerebellar hypometabolism in relation to primary lesions above the tentorium.

MATERIALS AND METHODS

All patients in this retrospective analysis were participants in an ongoing study of central nervous system abnormalities that accompany severe head injury. Twenty-nine patients (with no history of previous neurological disorder or head trauma) who sustained head injury severe enough to require hospitalization were enrolled in this study. Therefore, there were no inclusion or exclusion criteria for the analysis reported in this article. All patients underwent radiograph computed tomography (CT) and/or magnetic resonance imaging (MRI) and [^{18}F]FDG-PET imaging to determine the structural and functional consequences of head injury. Of the 29 patients, 22 were men and 7 were women. The subjects ranged in age from 18 yr to 59 yr with a mean age of 27 yr. Glasgow coma scores were assigned to each patient at the time of initial presentation to the emergency room. These scores ranged from 3 to 14 with a mean of 9 (15 represents complete consciousness and 3 denotes deep coma). Glasgow coma scores were also determined at the time of the PET scans; these scores ranged from 4 to 15 with a mean of 11.

CT images were obtained using a GE 8800 or GE 9800 CT scanner. Slices were obtained with a thickness of 5 mm in the majority of patients and no intravenous contrast was used. Magnetic resonance images were obtained using a GE 1.5 Tesla superconducting magnet and a multislice spin–echo technique. Axial images were generated with a pulse repetition time of 1500–2500 msec and two echoes (TE 25 and 120 msec) yielding mixed intensity and T-2 weighted images.

MRI and CT images of the brain were evaluated blindly by a neuroradiologist. However, no interior intrarater reliability was determined for either the anatomic or functional images. Anatomic lesions were identified and recorded. Focal lesions were categorized as one of the following: gunshot wound, cortical contusion, intracerebral hematoma, tissue tear hemorrhage, focal axonal damage, basal ganglia hemorrhage, secondary infarction, epidural hematoma, acute subdural hematoma, subacute subdural hema-

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toma, chronic subdural hematoma, subarachnoid hemorrhage or intraventricular hemorrhage. Diffuse lesions were categorized as either secondary brain swelling, diffuse cerebral concussion or diffuse axonal injury (DAI). The diagnosis of DAI was made by the attending neurosurgeon on the basis of clinical and radiological findings, was rated as mild, moderate or severe and was then integrated into the rankings. Up to five anatomic lesions for each patient were placed in rank order by the attending neurosurgeon, according to the apparent clinical severity of those lesions. These lesions were then classified using the Abbreviated Injury Scale for 1990 (AIS-90) code, with reference to lesion type, location, laterality and size. These categories of lesions were then compared to the results obtained by the FDG-PET scans.

A total of 46 FDG-PET studies were performed on the 29 patients, with the initial study performed 3 days to 5½ months after the trauma, with a mean interval of 29 days. Of the 29 patients, 13 had more than 1 study, 9 had 2 studies and 4 had 3 studies. The second and third studies were done from 2 wk to 3 yr and 5 mo after the initial study. These repeated scans were performed as part of the established protocol, which required follow-up studies. However, because of the variety of circumstances, only a limited number of subjects underwent such scans.

PET images were obtained with a modified positron emission transverse tomograph (PETT-V) system (*12*). The regional cerebral metabolic rate of glucose was determined after the intravenous administration of FDG using a method described previously (*13*). The modified PETT-V system used seven simultaneously generated cross-sectional images of the brain along the rostrocaudal axis. Two sets of scans obtained in every patient yielded a total of 14 overlapping image slices per session at 8.8 mm intervals. The PETT-V system has an axial resolution of 16.5 mm full width at half maximum. Scanning time was adjusted so that a minimum of one million counts were obtained per slice. The head was secured by a head holder with straps over the forehead and chin, and the imaging plane was parallel to the canthomeatal line.

PET studies were read blindly by an experienced nuclear medicine physician. Qualitative PET readings were made, which noted the presence and location of supratentorial hypometabolism and the presence and laterality of cerebellar hypometabolism. The observer determined whether the abnormality noted had clear-cut boundaries and could be considered focal versus an abnormality that was poorly defined and rated as representing a diffuse area of dysfunction. Cerebellar metabolism and/or blood flow was determined by comparing one cerebellar hemisphere to the other, as well as to other structures throughout the brain. This was validated previously by reviewing a large database of normal PET studies accumulated in our laboratory. It has been our experience that cerebellar metabolism is comparable to that of temporal lobes and, therefore, PET scans in the study that revealed decreased cerebellar metabolism on both sides relative to temporal lobes were considered to show bilateral effect.

Although the initial PET scan for each patient was used for generating the data for this study, subsequent PET scan results were also analyzed to determine changes over time for abnormal findings. One patient was found to have a technically inadequate study with respect to cerebellar imaging. This patient was eliminated from the data analysis, leaving a total of 18 patients with both adequate initial PET scans and anatomic studies.

Data were first examined for associations between categories of lesions and patterns of cerebellar hypometabolism. A total of 127 anatomic lesions were recorded for the 29 patients. These included diffuse and focal lesions, both above and below the tentorium. Diffuse lesions (n = 16) diagnosed in this patient population included primary cerebral concussions, DAI, nonfocal subarachnoid hemorrhages and diffuse secondary brain swelling. Because such diffuse lesions were considered bilateral, cerebellar hypometabolism could not be characterized as ipsilateral or contralateral to these diffuse lesions. Accordingly, cerebellar hypometabolism was characterized in absolute terms—right, left, bilateral or none.

TABLE 1

Diffuse lesion type	Total	Cerebellar hypometabolism			
		Right	Left	Bilateral	None
Secondary brain swelling, diffuse	3	2	0	0	1
Cerebral concussion	2	2	0	0	0
DAI, mild	4	2	1	0	1
DAI, moderate	3	0	1	0	2
DAI, severe	3	0	1	0	2
Sum of all diffuse lesions	15	6	3	0	6
Percent of all diffuse lesions	100	40	20	0	40

Focal lesions (n = 112) seen in this patient population included skull fractures, cortical contusions, intracerebral hematomas, extraparenchymal hematomas, basal ganglia hemorrhages, localized intraventricular hemorrhages, hemispheric penetrating injuries, subdural hygromas, localized cerebrospinal fluid leaks, localized hydrocephalus, focal secondary brain swellings, tentorial herniations, cranial nerve injuries and primary brain stem lesions. For this analysis, skull fractures, tentorial herniations, primary brain stem and cranial nerve injuries, cerebrospinal fluid leaks and localized hydrocephalus were excluded from the data analysis. Also, bilateral and midline lesions were not included in the analysis since cerebellar hypometabolism could not be characterized as ipsilateral or contralateral. The remaining 68 lesions were included in the final portion of the data analysis, all of which were unilateral and supratentorial.

In addition, data were examined for associations between categories of patients and patterns of cerebellar hypometabolism. The patient population was divided into those patients with diffuse lesions only, with focal lesions only and with both focal and diffuse lesions. For patients with only focal lesions or with focal and diffuse lesions, the relationship between the most severe focal injury, as determined by the clinical and imaging findings and the cerebellar hypometabolism, was determined for the final analysis. From this relationship, the percentage of patients with ipsilateral and contralateral cerebellar hypometabolism was calculated. One patient with diffuse injury alone was noted in the population and was excluded from the study.

Statistical analysis of each category of head injury compared with cerebellar hypometabolism used chi-square tests to determine if the frequency of contralateral cerebellar hypometabolism was significant compared with a normal distribution. Two-tailed tests were used because they are more conservative and therefore protect against results in the opposite direction to that hypothesized.

RESULTS

The association of supratentorial lesions with cerebellar hypometabolism was determined for both diffuse and focal lesions. As shown in Table 1, 40% of all diffuse lesions observed in the patient population were associated with right-sided cerebellar hypometabolism, whereas only 20% were associated with left-sided cerebellar hypometabolism.

Table 2 shows that 40% of focal unilateral lesions (n = 68) were associated with contralateral cerebellar hypometabolism. This is significantly greater than the 19% of focal unilateral lesions that were associated with ipsilateral cerebellar hypometabolism [chi-square (3,n = 68) = 19.64, p = 0.002)]. When intraparenchymal lesions such as cortical contusions, hemispheric penetration injuries, intracerebral hematomas and basal ganglia hemorrhages were considered as a separate group (n =

TABLE 2

Focal or unilateral lesion type	Total	Cerebellar hypometabolism			
		Ipsilateral	Contralateral	Bilateral	None
Gunshot wound*	2	0	2	0	0
Cortical contusion*	26	7	9	1	9
Intracerebral hematoma*	9	0	4	1	4
Tissue tear hemorrhage*	1	1	0	0	0
Focal axonal damage*	3	1	2	0	0
Basal ganglia hemorrhage*	3	1	0	0	2
Infarction, secondary*	4	0	1	0	3
Epidural hematoma†	4	0	2	0	2
Acute subdural hematoma†	11	2	4	2	3
Subacute subdural hematoma†	1	0	0	0	1
Chronic subdural hematoma†	2	1	1	0	0
Subarachnoid hemorrhage†	1	0	1	0	0
Intraventricular hemorrhage†	1	0	1	0	0
No. (percentage) of focal intraparenchymal lesions (*)	48 (100)	10 (21)	18 (38)	2 (4)	18 (38)
No. (percentage) of focal extraparenchymal lesions (†)	20 (100)	3 (15)	9 (45)	2 (10)	6 (30)
All focal lesions (percentage)	68 (100)	13 (19)	27 (40)	4 (6)	24 (35)

48), there was a significant association with contralateral cerebellar hypometabolism observed in 38% of the patients [chi-square (3,n = 48) = 14.66, p = 0.002)] compared with ipsilateral cerebellar hypometabolism in 21% of the patients (see Table 2). Focal extraparenchymal lesions (n = 20) were associated with a predominance of contralateral cerebellar hypometabolism in 45% compared with ipsilateral cerebellar hypometabolism in 15% (see Table 2). There was also a significant association between focal lesions (for all subcategories) ranked as the patient's most severe injury (n = 13) and contralateral cerebellar hypometabolism [chi-square (3,n = 13) = 13.77, p = 0.003)]. Nine of these lesions (70%) were associated with contralateral cerebellar hypometabolism and only one (8%) was associated with ipsilateral cerebellar hypometabolism (Table 3).

Patients were also compared according to the type of injuries sustained (Table 4). Of the patients with focal injuries alone (comprising 38 lesions), 50% of the lesions were associated with contralateral cerebellar hypometabolism and 13% were associated with ipsilateral cerebellar hypometabolism. This association was significant compared to that of patients with both focal and diffuse injuries [chi-square (3,n = 68) = 9.02, p = 0.03)]. In the patients with mixed focal and diffuse injuries, there were a total of 30 focal lesions. Of these focal lesions there was an equal distribution of cerebellar hypometabolism, with 27% having ipsilateral hypometabolism and 27% having contralateral hypometabolism.

The results obtained from patients with more than one PET scan show that the cerebellar hypometabolism may change over time. As shown in Table 5, of the 13 patients receiving more than one PET study, 7 exhibited a change in the presence and/or laterality of cerebellar hypometabolism from one study to another. Four patients did not show any change in cerebellar hypometabolism. The change in cerebellar hypometabolism was not particularly associated with the bilaterality of focal lesions (three of seven patients exhibited shifts). All four patients with unilateral supratentorial injuries exhibited a change in the direction of the cerebellar hypometabolism on repeat scans. A change in cerebellar hypometabolism was not associated with the presence or absence of diffuse injury because only five of eight patients with diffuse injuries exhibited a change between studies and two of three patients without diffuse injuries manifested a change. In spite of the limited sample, the repeat scans provided some interesting results, which clearly demonstrate temporal evolution of cerebral function as the patient recovers from the effects of head injury.

TABLE 3

Most severe focal injury type	Total	Cerebellar hypometabolism			
		Ipsi-lateral	Contra-lateral	Bi-lateral	None
Gunshot wound	2	0	2	0	0
Cortical contusion	6	1	2	1	2
Intracerebral hematoma	1	0	1	0	0
Epidural hematoma	1	0	1	0	0
Acute subdural hematoma	3	0	3	0	0
All lesions	13	1	9	1	2
Percentage of all lesions	100	8	70	8	15

DISCUSSION

In von Monakow's original development of the theory of diaschisis (*14,15*), and the subsequent work by Kempinsky (*16–18*), four important characteristics of the phenomenon were specified: an impairment of brain areas distant from the site of the primary lesion; a mechanism involving a loss of excitation to the areas secondarily affected; the occurrence of the phenomenon along neuroanatomic pathways connecting the sites of primary and secondary damage; and reversibility of the phenomenon with time (*9,14–18*). The observed phenomenon of crossed cerebellar diaschisis by functional neuroimaging techniques is largely consistent with these theoretical criteria (*1–9*). A well-defined, unilateral cerebral injury may be associated with contralateral cerebellar hypometabolism with or without clinical dysfunction (*1–9*). It has been suggested that

TABLE 4

Patient group type injury type	Total focal lesions	Cerebellar hypometabolism			
		Ipsi-lateral	Contra-lateral	Bi-lateral	None
Focal lesions alone	38	5	19	4	10
Percentage	100	13	50	11	26
Focal and diffuse lesions	30	8	8	0	14
Percentage	100	27	27	0	46

TABLE 5

Patient no.	Total studies	Cerebellar hypometabolism (R/L/N)	Diffuse lesions	Bilateral lesions
1	2	R → R	Yes	Yes
2	2	R → R	No	Yes
3	2	N → N	Yes	Yes
4	3	N → N → N	Yes	Yes
5	2	R → N	Yes	No
6	2	L → R	Yes	Yes
7	2	R → L	No	No
8	2	N → N → R	Yes	No
9	2	L → N	No	No
10	3	L → Inad → N	Yes	Yes
11	3	N → N → R	Yes	Yes
12	2	N → Inad	—	—
13	3	L → Inad → L	—	—

R = right; L = left; N = none; Inad = inadequate.

the pathophysiology involves an interruption of excitatory input from the corticopontine tract fibers originating in or traveling through the area of primary injury to their synapses in the pontine nuclei (*7*). Explaining the reversibility of the phenomenon is more problematic because crossed cerebellar diaschisis may have a protracted course. In fact, the early pathological descriptions of cerebellar atrophy in such chronic cases would likely result in a persistent nonreversible decrease in cerebral blood flow as is often seen in neurodegenerative diseases (*7*), and in this sense are inconsistent with the original criteria described by von Monakow and Kempinsky (*14–18*). Feeney and Baron suggested that chronic, irreversible cerebellar hypometabolism may be secondary to anterograde transneuronal degeneration (*9*). Furthermore, permanent changes may occur after an initial period of purely metabolic and possibly reversible depression.

The results from this study are generally consistent with those observed for nontraumatic mechanisms of cortical injury and with the theoretical phenomenon of diaschisis. Traumatic lesions involving the cerebral cortex or directly adjacent to it (i.e., localized lesions in the extraparenchymal space) are frequently associated with crossed cerebellar hypometabolism. Exceptions may well be related to the complicating factor of multiple co-existent injuries in the same patient. The fact that the crossed cerebellar hypometabolism was most consistently seen in relation to the most severe lesions indicates that these lesions may dominate the effect. Furthermore, patients with focal cerebral injury alone showed a remarkable consistency of crossed cerebellar hypometabolism. However, in patients with several lesions, other significant injuries may play a role in the development of cerebellar hypometabolism. This study also showed that over time there are changes in the presence and laterality of cerebellar hypometabolism, which may be related to the natural course of the anatomic and metabolic abnormalities and due to successive competing effects of different lesions.

Diffuse injuries did not themselves appear to be associated with a particular laterality of cerebellar hypometabolism. Furthermore, patients with diffuse injuries had cerebellar hypometabolism patterns that differed from that observed in the patient group as a whole. In fact, diffuse injury tended to "dilute" the association of focal lesions with crossed cerebellar diaschisis. In the presence of diffuse injury, no significant pattern of crossed cerebellar hypometabolism was seen. This is understandable given the dependence of the phenomenon on discrete and specific interruption of white matter communication between the area of primary injury and pontine nuclei. If white matter fibers are diffusely damaged, no such discrete and specific interference can be expected. This "dilution" of crossed cerebellar hypometabolism with diffuse axonal injury may itself provide indirect evidence for the occurrence of diaschisis more frequently than noted in this sample in those head trauma patients without diffuse axonal injury.

The difficulties in determining the significance of the findings in this study lie in the limited number of patients and lesions in the population described. Furthermore, this is a qualitative study that was adequate for the purposes of this initial investigation. However, quantitative analysis may add additional information to these preliminary findings.

CONCLUSION

The results of this study indicate that the phenomenon of crossed cerebellar diaschisis, as described in supratentorial neoplasm and infarction, is also seen in head trauma patients. This is particularly the case with patients suffering from focal, unilateral lesions and can be easily demonstrated using PET imaging. The phenomenon may be obscured by the effects of multiple lesions in the same patient and is not seen in patients with diffuse axonal injury. The crossed cerebellar diaschisis seen in acute head injury patients often changes in presence and laterality over time and may be secondary to the competing effects of multiple lesions in the same patient. The clinical significance of crossed cerebellar diaschisis in acute head trauma is unclear, as is its effect on eventual outcome in such patients.

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