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Ipsilateral Hemiparesis in Intraparenchymal Hemorrhage: Case Report and Review of Literature

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Abstract: *Stroke presenting as contralateral hemiparesis is predominantly related to contralateral projection of the corticospinal tract. While most corticospinal fibers decussate at the level of the medulla, some tracts continue to descend as ipsilateral anterior corticospinal fibers. The anterior corticospinal tract (CST) has been suggested as one of the ipsilateral motor pathways which contribute to motor recovery following stroke. Few case reports in literature show ipsilateral hemiparesis due to involvement of anterior corticospinalfibers.There is an association in patients with congenital uncrossed corticospinal tracts and posterior fossa malformations. There was also a high correlation of ipsilateral hemiparesis in patients with remote infarction. A previous stroke was seen in 50% of the patients, all except for 1 case were ischemic in nature. Patients with previous infarcts do demonstrate an adaptive compensation for damaged or disconnected regions of an injured area. This emphasizes the need to consider the investigation for previous infarctions or underlying genetic structural defects by using certain imaging modality.We are reporting a case with no prior co-morbitidities,representing in Emergency Department with ipsilateral hemiparesis and seizure.*

I. Introduction

Stroke is commonly recognized as causing neurological impairment of movement on the contralateral side of the body. The reason for this is felt to be the predominance of contralateral corticospinal projections, which arise from the cortical regions of the brain and decussate in the caudal medulla before traveling to the spinal cord [1]. While not all fibers decussate, 70-90% of them do, resulting in this 'crossed' clinical finding [2]. Prior neuroanatomical studies have firmly supported the notion that the primary motor cortex predominantly innervates the contralateral half of the body [3]. This was demonstrated by Dejerine [4] and Foerster [5], when stereotactic procedures performed for relief of Parkinsonism were noted to produce contralateral hemiparesis. However, as Nyberg-Hansen [6] demonstrated in his studies involving primates, cats, and adult rats, projection of pyramidal tract neurons to the ipsilateral spinal cord does exist.Humans are particularly interesting because in some individuals, as many as 30% of the corticospinal axons descend as the ipsilateral ventral corticospinal tract [3]. A careful quantitative motor examination of patients with a contralateral hemiparesis will at times reveal some weakness in the ipsilateral arm and leg. When examiners use the ipsilateral limbs for comparison of motor strength, they notice only that the contralateral limbs are much weaker; they fail to recognize that the ipsilateral limbs are also weak.

II. Case

A 45yr old male patient came to Emergency Department with complaints of weakness of left side of the body since last three days accompanied with stiffness of both left upper and lower limbs for three minutes in the night prior and since then repeated intermittent episodes before presenting to emergency department.He did not seek any medical help for it.On arrival to emergency patient was in post-ictal stage and confused,Vitals were stable.

Past Medical History:-No history of Diabetes Mellitus/Hypertension/Stroke/Coronary artery disease/Seizure Disorder ,he was a chronic alcoholic however had not consumed alcohol in the last 1 week.

Differential Diagnosis:-Withdrawal Seizures,Stroke(Ischemic or Haemorhagic)

Investigations:- MRI Brain(plain) ,ABG (pH-7.38,pCO2-39,pO2-93),Glucose-168mg/dl.

On Examination:-,Neurological examination revealedGCS E3 V5 M6, Powers using MRC grading system **(**Right upper limb-5/5,Right lower limb-5/5 ,Left upper limb-3/5,Left lower limb-4/5) with no deviation of face towards left side,Babinski sign :-Right side flexion,Left was Non -Reactive .

Treatment:- Patient was loaded with Injection Levetiracetam 1gm intravenously.

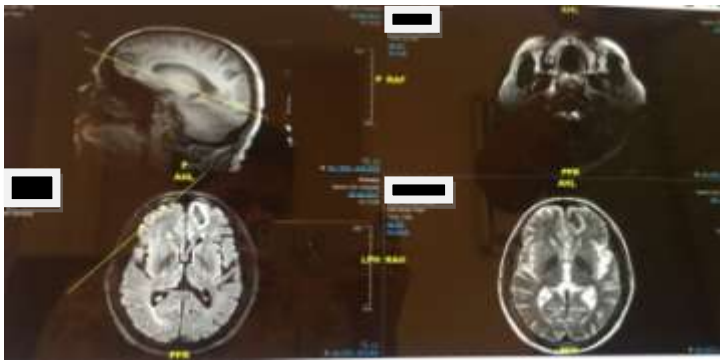

Fig.1-MRI BRAIN picture showing Intraparenchymal-hemorrhage at the gray matter junction in the left anterior frontal lobe and age related cerebral atrophy.After Imaging Neurology consultation was taken, after evaluation Neurologist also documented decrease power on left side andipsilateral hemiparesis.

III. Review Of Literature

Existence of Ipsilateral Hemiparesis in Ischemic and Hemorrhagic Stroke: Two Case Reports and Review of the LiteratureF. Saada N. AntoniosUniversity of Florida College of Medicine, Jacksonville, Fla., USA

After an exhaustive search of ipsilateralhemiparesis, ipsilateral stroke, and ipsilateral TIA yielded 16 publications in PubMed and 10 in BIOSIS.

Summary of case report and series of ipsilateral hemiparesis/plegia with unilateral stroke:-

Authors	Cases	Age,yrs	Gender	Type of stroke	Paresis	Location	Dominant hemisphere	strokes	Location	Structural defect	Imaging
Alurkar et al [1]	1	55	M	IS	R	CR	L	first	-	-	MRI
Terakawa et al [6]	1	62	M	ICH	R	Putamen	L	first	-	PCS	CT
Hosokawa et al [2]	1	-	-	ICH	R	IC & Thalamus	-	first	-	-	CT
Song et al [4]	2	62 41	F M	IS	L R	CR CR	L L	Third second	R CR & R thalamus R CR	- -	MRI MRI
Ago et al[5]	1	59	M	IS	L	CR	-	first	Putamen HS	-	MRI
Ng et al[9]	1	55	M	IS	L	CR& putamen	L	first	-	HGP+PCS	MRI,DTIT
Kang and Choi et al[7]	1	35	M	IS	R	IC,F,Putamen	-	second	-	NYS+CCA	MRI
Patient in literature	2	57 67	M F	IS IS	L R	O,P,F F	L L	second	R CR & R O R IC	-	MRI MRI
Our Patient	1	45	M	IS	L	Frontal lobe	L	first	-	-	MRI

L-Left;R-Right;IS-Ischemicstroke;ICH-intracerebralhemorrhage;IC-internalcapsule;CR-coronaradiata;O-Occipital;P-parietal;F-frontal;HS-hemorrhagicstroke;HGP-horizontal gaze paralysis;;PCS-progressive congenital scoliosis;NYS-nystagmus;CCA-corpuscollasumagenesis;DTIT-diffusion tensor imaging tractography.

After exclusion of non-relevant articles, they left with total of 8 cases from the literature and 2 cases of their own.All cases were hospital based and reported retrospectively.

Table.1.-summary of case reports and series if hemiparesis with ipsilateral hemiplegia/hemiparesis with unilateral stroke (Alurkar et al[1] , Terakawa et al[6] , Hosokawa et al[2] , Song et al[4] , Ago et al[5] , Ng et al[9] , Kang and Choi[7])

Table shows total of 10 patients, 7men, 2 women and 1 who s gender was not recorded.

8 Ischemic supra-tensorial Stroke and 2 Hemorrhagic Strokes (ICH)

IV. Discussion

Ipsilateral Hemiparesis after a Supratentorial Cerebral Ischemic or Hemorrhagic Stroke has rarely been reported.In Literature ten cases have been described in which Stroke was a cause ofIpsilateral Hemiparesis.The most common causes were related to ipsilateral projection from the primary motor cortex. [1]Congenitally uncrossed pyramidal tracts [2], cortical re-organization within the motor areas of the un-affected hemisphere [3].

In our case, Patient developed left hemiparesis associated with Intraparenchymal hemorrhage at the gray matter junction in the left anterior frontal lobe.Although we were not able to obtain MRI Tractographyto confirm our conclusion but our case was at least consistent with uncrossed corticospinaltracts, because it did not

include the primary motor cortex and possibility of cortical reorganization of the affected hemisphere can be ruled out as there was no prior history of stroke.

V. Conclusion

Ipsilateral hemiparesis after ischemic or hemorrhagic stroke is a rare finding, but it can develop as a result of a new ischemic or hemorrhagic stroke in patients with or without a previous history of stroke.Similar cases have been reported with one being confirmed with MRI tractography [8].Patients who have ipsilateral stroke consistent with uncrossed corticospinal tract have been reported to have other associated cerebral anomalies.Not all ipsilateral strokes are due to uncrossed corticospinal fibers. Some may be due to cortical reorganization within the motor areas of the unaffected hemisphere. Patients with previous infarcts demonstrate an adaptive compensation for damaged or disconnected regions of the injured area. All of these considerations emphasize the need to consider investigations for previous infarctions, underlying congenital anomalies, or functional reorganization of the cerebral cortex in such patients. In the future, this consideration may play a large role in patients' status for recovery.

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