Case Report

Persistence of traumatic infantile hemiparesis in an adult - A rare case report

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International Archives of Integrated Medicine, Vol. 10, Issue 4, April, 2023.
Available online at http://iaimjournal.com/
ISSN: 2394-0026 (P) **ISSN: 2394-0034 (O)**
Received on: 25-3-2023 **Accepted on:** 6-4-2023
Source of support: Nil **Conflict of interest:** None declared.

How to cite this article: E.A. Ashok Kumar, Shaik Aneesa Fathima. Persistence of traumatic infantile hemiparesis in an adult - A rare case report. IAIM, 2023; 10(4): 46-53.

Abstract

Hemiplegia is the physical manifestation of an injury to a specific area of the brain that controls motor function. Hemiplegia may develop suddenly, or evolve over days, weeks or months. Hemiparesis/ hemiplegia is rare in children. It causes significant mortality and morbidity. Infections are an important cause of neurological deficit, in the developing countries. Once the injury has occurred, the symptoms should not worsen. However, because of lack of mobility, other complications can occur. Complications may include muscle and joint stiffness, and shortening of limbs. The term acute infantile hemiplegia denotes certain cases of hemiplegia of sudden onset in children, of a few weeks to 6 or more years of age. The outcome may be fatal; or there may be permanent hemiplegia with mental impairment. There may be slow partial recovery; or rapid and complete recovery. We herewith present a 35 old male, who presented with seizures. 15 years ago he sustained head injury as he fell down from a tree. He was diagnosed with subdural hematoma and was evacuated through craniotomy, after which he developed residual right hemiparesis. The right hemiparesisis was still persisting as residual hemiparesis on right side with shortening of limbs. A diagnosis of Post traumatic Infantile hemiparesis on right side was made.

Key words

Infantile Hemiparesis, Traumatic Infantile Hemiparesis.

Introduction

The term infantile hemiplegia is used to describe the clinical condition of the sudden onset of hemiparesis unassociated with intracranial hemorrhage [1]. Infantile or congenital hemiplegia also refers to brain injuries that occur before or at birth and lead to hemiplegia. Juvenile hemiplegia is seen in

patients who sustained injuries above the age of 1 year [2].

Hemiplegia is the physical manifestation of an injury to a specific area of the brain that controls motor function. Hemiplegia may develop suddenly, or evolve over days, weeks or months. Additionally, some infants who appear normal in the newborn period may show symptoms of hemiplegia only after voluntary hand use develops i.e.at the age of 4-5 months. Hemiplegia can also be transient or permanent [2, 3].

Hemiparesis/ hemiplegia is rare in children. It causes significant mortality and morbidity. Infections are an important cause of neurological deficit, in the developing countries [2]. Cases of infantile hemiplegia with probable correlation with coxsackie A9 viral infection is also described [4].

Hemiplegia is not a progressive disorder but it is progressive in conditions for example a growing brain tumor. Once the injury has occurred, the symptoms should not worsen. However, because of lack of mobility, other complications can occur. Complications may include muscle and joint stiffness, and shortening of limbs, loss of aerobic fitness, muscle spasms, bed sores, pressure ulcers and deep vein thrombosis.

Acute hemiplegia in childhood, also known as acute infantile hemiplegia, infantile acquired hemiplegia, Marie-Striimpell encephalitis, polioencephalitis, and hemiconvulsion and hemiplegia (H. H.) syndrome. Hemiconvulsion and hemiplegia (H. H.) syndrome is postnatally acquired hemiplegia in a child apparently neurologically normal at birth. This clearly excludes hemiplegia related to prenatal factors (congenital anomalies, chromosomal aberrations or prenatal infections) and perinatal factors prematurity, birth trauma or perinatal infections). Recognition of acquired hemiplegia in the neonatal and early infancy period is most difficult, as it is confused with congenital disorders. Delineation of the syndrome is further complicated by the frequent failure to recognize congenital hemiplegia before 4 to 5 months of age. Relative acuteness of onset is the determining factor [5].

The term acute infantile hemiplegia denotes certain cases of hemiplegia of sudden onset in children, of a few weeks to 6 or more years of age. The outcome may be fatal; or there may be permanent hemiplegia with mental impairment. There may be slow partial recovery; or rapid and complete recovery [6].

Cases of intermittent nature of symptoms were also noted in patients who had seizures and hemiplegia followed by complete disappearance of hemiplegia/ hemiparesis with residual symptoms like tendency to fall [7]. The prevalence of severe disabilities at 7 years after NAIS (Neonatal Arterial Ischemic Stroke) is low, but most children exhibit some impairment in their growth [8].

Case report

A 35 old male presented with complaints of seizures of one day duration.

Patient was apparently asymptomatic a day earlier, after which he developed Seizures which were generalized tonic clonic seizures, associated with loss of consciousness. It was followed with involuntary muscle contractions lasting for 2 min later on he regained complete consciousness.

15 years ago, patient sustained a head injury as he fell down from a tree. He was admitted to a hospital where he was diagnosed with Subdural hematoma and was evacuated through craniotomy, after which he developed residual right hemiparesis.

No history of trauma during birth, No history of perinatal asphyxia, No history of obstructed labor, No history of prematurity, No history of bleeding diathesis.
Past history - Not a known case of DM, HTN, TB, BA, Thyroid disorders.

General examination: Patient was conscious, coherent, co-operative, normal built and well nourished. No Pallor, Icterus, Cyanosis, Clubbing, Koilonychia, Lymphadenopathy. No pedal edema. Shortening of limbs is noted on right side.
Vitals: Temp – afebrile, BP=140/90 mm of Hg in sitting position in right arm, Pulse=80/min regular in rhythm with no radio-radial or radio-femoral delay.

CNS examination:
Intellectual functions: Patient is conscious/coherent/co-operative, oriented to time, place, person, right-handed. Memory - Intact. He has Normal concentration and intelligence. Speech is spontaneous with good comprehension. No aphasia. No hallucinations/delusions.
Cranial nerves: - Normal.

Motor system

Tone	Right side	Left side
Upper limb	Increased	Normal
Lower limb	Increased	Normal

Power	Right side	Left side
Arm	4/5	5/5
Forearm	4/5	5/5
Wrist	4/5	5/5
Hip	4/5	5/5
Knee	4/5	5/5
Ankle	4/5	5/5

Bulk	Right side	Left side
Arm	28 cm	31 cm
Forearm	18 cm	21 cm
Thigh	48 cm	53 cm
Leg	30 cm	35 cm

Length	Right side	Left side
Upper limb	69 cm	73 cm
Lower limb	90cm	95 cm

No abnormal movements were seen.

Cerebellar functions: - No titubation, no nystagmus, no pendular knee jerk, no dysdiadokinesia, no past pointing, finger nose test was normal on both sides.
Gait-hemiplegic gait

Reflexes

	Right side	Left side
Biceps	3	2
Supinator	3	2
Knee	3	2
Ankle	3	2
Plantar	Extensor	Normal-Flexor

Sensory system – all modalities of sensations are normal
Peripheral nerves – not thickened
Skull and spine: - Normal
CVS examination: S1, S2 - heard, no murmurs
Abdomen examination: Abdomen was soft and not distended, non-tender, no organomegaly
Respiratory system examination: Bilateral normal vesicular bronchial breathing present with no adventitious sounds.

In the beginning from the appearance of patient it looked as if he had poliomyelitis but on examination it was concluded that the shortening of limbs was because of upper motor neuron lesion, not lower motor, hence it was not a case of poliomyelitis. Moreover, he had head injury and right hemiparesis 15years ago which is still persisting as residual hemiparesis on right side with shortening of limbs.

Investigations
Complete blood picture - Hb – 14 g%, RBC count - 4.5 mill/cu mm, WBC count - 6700 cells/cu mm, Platelet count - 2.0 lakh/cu mm. Complete urine examination - Colour – pale yellow, Appearance – clear, Reaction – acidic, Albumin – nil, Sugar – nil, bile salts, bile pigments and ketones absent, Pus cells – nil, Epithelial cells – nil, RBC'S, casts and crystals – absent. Renal function test- RBS – 130, blood urea – 36 mg/dl, serum creatinine - 0.8 mg/dl. Liver function test - S. Bilirubin (total) - 0.4 mg/dl, S. Bilirubin (direct) - 0.1 mg/dl, S. Total Proteins – 6 mg/dl, S. Albumin – 4 mg/dl, S.

Globulin – 3 mg/dl, SGOT – 45 IU/L, SGPT – 35 IU/L, S. Alkaline Phosphatase – 50 IU, X-Ray Chest P A View – Normal, ECG-normal.

EEG - normal; fundus – normal; Plain CT Scan Brain - Plain CT Scan brain- Ill defined area of hypodensity of CSF attenuation noted in fronto-temporo-parietal region of cerebral parenchyma on left side with mild evacuee dilatation of ipsilateral lateral ventricle- possibility of post operative changes, Craniotomy defect present, Chronic lacunar infarcts in left capsuloganglionic region, Minimal periventricular ischemic changes, Mild age related cerebral atrophic changes.

Figure – 1: Showing shortening of upper limb on the right side, Decrease in bulk of arm and forearm on the right side, Arm and Forearm are in adduction, wrist and fingers are in flexion.

Figure – 2: Decrease in bulk of muscle and shortening of lower limb on the right side. The right lower limb is externally rotated.

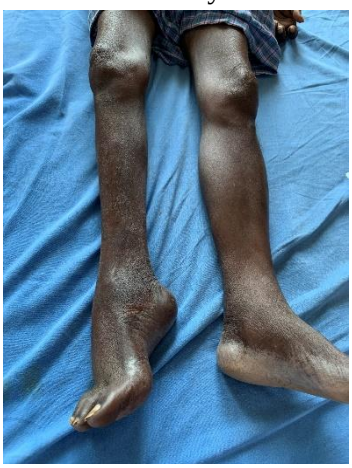
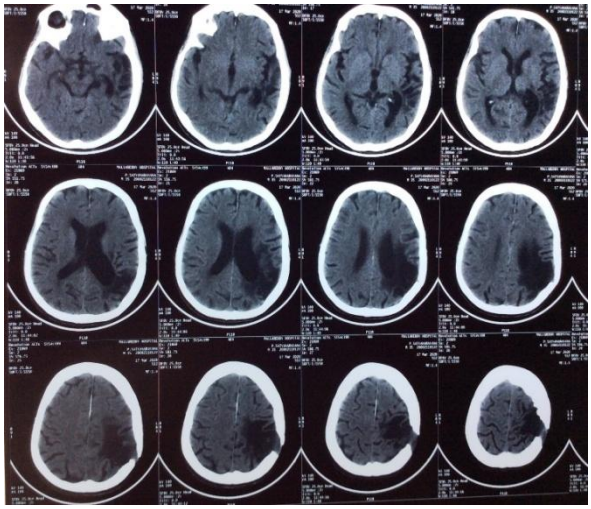


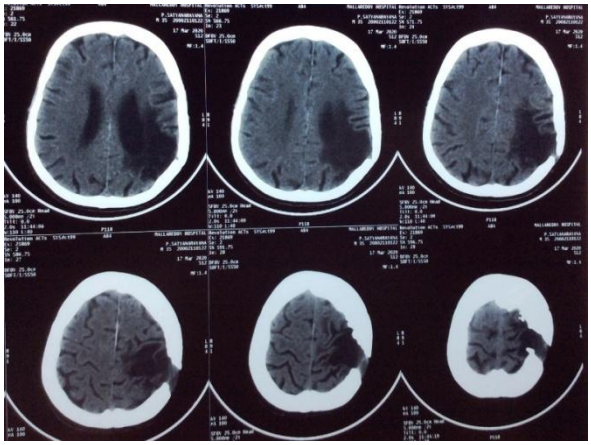
Figure – 3: Showing patient's stance on standing.

Figure – 4: Showing patient's stance in bed.

Plain CT Scan brain (**Figure – 5, 6**) - Ill defined area of hypodensity of CSF attenuation noted in fronto-temporo-parietal region of cerebral parenchyma on left side with mild evacuee dilatation of ipsilateral lateral ventricle- possibility of post operative changes, Craniotomy defect present, Chronic lacunar infarcts in left capsuloganglionic region, Minimal periventricular ischemic changes, Mild age related cerebral atrophic changes.

Figure – 5, 6: Plain CT Scan brain.





Diagnosis

Post traumatic Infantile hemiplegia on right side in an adult.

There is no medical test that confirms the diagnosis of infantile hemiplegia. The diagnosis is made on the basis of various types of information gathered by the physician. There will be a history of hemiplegia during childhood which is not recovered due to various reasons. If this hemiplegia persists into adulthood, it should be diagnosed as infantile hemiplegia.

Plain CT Scan brain(**Figure – 5, 6**) shows - Ill-defined area of hypodensity of CSF attenuation noted in fronto-temporo-parietal region of cerebral parenchyma on left side with mild evacueedilatation of ipsilateral lateral ventricle-possibility of post operative changes, Craniotomy defect present, Chronic lacunar infarcts in left capsuloganglionic region, Minimal periventricular ischemic changes, Mild age related cerebral atrophic changes

Management

Apart from treatment of hemiplegia/ hemiparesis, physiotherapy has a main role in preventing contractures, stiffness. The shortening of limbs is due to growth restriction of both muscles and bones, because of not using the limbs or not giving physiotherapy.

Physiotherapy includes following

(a) **Conventional therapies** (Therapeutic Exercises, Traditional Functional Retraining) which include, Range of motion Exercises,
Muscle Stretching Exercises, Splintingfitness training, Compensatory Techniques.

(b) **Bed positioning**

1. Lying on the affected side; one or two pillows for head, affected shoulder positioned comfortably, Place unaffected leg forward on one or two pillows, Place pillows in front or behind to give support
2. Lying on unaffected side; One or two pillows for the head, Affected arm forward and supported on pillow(s), Affected leg backwards on one or two pillows, Place pillows behind.
3. Positioning of patient,

While sitting in bed: Sitting in bed is desirable for short periods only, Must be upright and well supported with pillows, Giving extra support using pillows under arms or knees.

While lying on back: Keep three pillows in a 'triangle', supporting shoulders and head, Keep affected arm on pillow, Ensure feet in a neutral position.

(c) **Neurofacilitatory techniques such as**

Bobath: it is an approach that focuses to control responses from damaged postural reflex mechanism. Emphasis is placed on affected inputs facilitation and normal movement patterns.

Rood: Emphasize the use of activities in developmental sequences, sensation stimulation and muscle work classification. Cutaneous stimuli such as icing, tapping and brushing are employed to facilitate activities.

Proprioceptive neuromuscular facilitation (PNF):is the use of peripheral inputs as stretch and resisted movement to reinforce existing motor response. Total patterns of movement are used in treatment and are followed in a developmental sequence [9].

(d) **Learning theory approach such as**

Conductive education; motor relearning theory: it emphasizes the practice of functional tasks and importance of relearning real-life activities for patients. Principles of learning and biomechanical analysis of movements and tasks are important [10].

Biofeedback; it facilitates the cognizant of electromyographic activity in selected muscle or awareness of joint position sense via visual or auditory cues.

(e) **Functional Electrical stimulation**

It applies a short burst of electrical current to hemiplegic muscle or nerve. It reduces spasticity in hemiplegic patient.

(f) **Conventional Gait training:** It has focused on part-practice of components of gait in preparation for walking. Includes, Symmetrical weight bearing training; Weight shifting Stepping training (swinging/ clearance). Heel strike, Single leg standing Push off/ Calf rise. Followed by, Circuit training (reaching in sitting and standing, sit-to-stand, step-ups, heel lifts, isokinetic strengthening, walking over obstacles, up and down slopes.

Discussion

Infantile hemiplegia persisting in an adult is a rare case. Infantile hemiplegia is difficult to recognize because of its less incidence. If infant doesn't gain overall control over the paralysed limbs thereby parents ignoring the symptoms may result in infantile hemiplegia. The unborn child can develop hemiplegia through various developmental complications, or infections contracted by the mother during the child's development could also result in infantile hemiplegia. A person suffering from infantile hemiplegia experiences temporary bouts with paralysis on one side of the body. Most often, abnormal labor is an important etiological factor in the congenital group of hemiplegias (23%).Cases of hemiplegia in young infants and children following trauma at birth or later in life following convulsive attacks from any cause are frequent. Some unusual cases also can occur without any known cause and have intermittancy of intial symptoms. Harold, et al., [7] thought that the most probable cause of the lesion causing right hemiplegia was of upper motor neuron type was due to hemorrhage followed by trauma causing destruction to some fibres causing paralysis.

The early symptoms are severe, and consist of convulsions, fever, often vomiting, and always coma". The convulsive element may be lacking, especially in the post-exanthematous cases, and, without it, irritability and stupor pass into coma. Two-thirds of all infantile hemiplegias, had convulsions as presenting symptom. These symptoms persist for twenty-four hours to a week, and, during this period, or as consciousness returns, it is apparent that a hemiplegia is present, affecting the face, the arm, and lastly the leg. The limbs remain flaccid for some days, after which the flaccidity begins to lessen, and the limbs become spastic. In some cases the paralysis completely disappears [7].

Angiography will be abnormal in 65 to 80% of cases, and a number of clinicopathological groups can be defined; 1.occlusive vascular disease at the base of the brain associated with telangiectasia of the basal ganglia (Moyamoya syndrome), 2. occlusive vascular disease at the base of the brain without telangiectasia, 3. narrowing of the origin of the internal carotid artery, 4. distal branch occlusion of intracranial arteries, and 5. corkscrew pattern in small terminal arteries. In the Moyamoya syndrome there is narrowing due to intimal thickening and abnormalities of the elastica. Segmental areas of arteritis account for the changes in many patients with other angiographic patterns. Although over-all mortality is low, residual hemiparesis, mental retardation, and a chronic convulsive disorder are common.4 Negative prognostic indicators

include age under two years, a prolonged seizure at the onset, or the presence of multiple seizures [1].

Arterial ischemic infarction occurring around the time of birth is an increasingly recognized cause of neurological disability in children. Several conditions in the neonatal period predispose to perinatal stroke including prothrombotic disorders, congenital heart disease, meningitis, and systemic infection. Perinatal stroke may present with neonatal seizures during the first weeks of life or may be asymptomatic until months later when the infant is first noted to have pathological handedness. As many as 50% of infants with documented stroke recognized in the newborn period do not develop a hemiparesis. The incidence, clinical presentation, pathogenesis, risk factors, and outcome of this increasingly recognized disorder are reviewed [11].

Based on estimates from population-based studies of infants with seizures, perinatal stroke occurs in approximately 1 in 4000 term births. Most perinatal strokes involve the middle cerebral artery and are caused by thromboembolism from an intracranial or extracranial vessel, the heart, or the placenta [3].
The hemiplegia fully developed has some resemblance to that produced by the apoplectic (symptoms of sudden onset stroke) stroke in the arteriosclerotic adult, where a massive and sudden vascular catastrophe occurs in the territory of the middle cerebral artery. The uselessness of the arm exceeds that of the leg; the facial weakness tends to disappear first. There the similarity ends. The commencement of infantile hemiplegia has no parallel in the adult hemiplegia. In about a third of the infantile hemiplegias the disease appears as a complication of one of the infectious fevers. But, there was no obvious connection between the specific fever and the complicating, though rare, hemiplegia. With the concentration of cases within the first three years, it is probable that many of those which suddenly develop hemiplegia of a permanent character, and are associated with mental deficiency, depend on antenatal and especially natal causes. In many cases a history of prolonged, difficult labour and asphyxia of the infant at birth is obtained.

It is pertinent to note that the etiology and features of infantile hemiplegia are different from that of adult hemiplegia. The following is a list of a few common causes: Cerebrovascular Accident (CVA) or stroke; Intraventricular Hemorrhage of the newborn (IVH), thrombosis: embolism or hemorrhage, Transient ischemic attack (TIA), Head Trauma: brain contusion, subdural hematoma or epidural hematoma , Brain tumor (Primary or metastatic disease), Infection: brain abscess, encephalitis, subdural empyema or meningitis, Vasculitis, Demyelinating disease: multiple sclerosis, acute necrotizing myelitis, Congenital or perinatal injury, Arteriovenous malformations.

Symptoms may include: Stiffness and weakness in muscles on one side of the body, only using one hand during play or favoring one hand before the age of 3 years, Keeping one hand in a fist, Difficulty with walking and balance, Difficulty with fine motor tasks like writing or using scissors, Delay in reaching expected developmental milestones e.g. rolling over, sitting up, crawling, or smiling, Muscle spasms, Emotion-depression, Epilepsy convulsions occur in the major in the majority of cases and is one of the most common and distressing symptoms is the occurrence of convulsive seizures, Mental Changes - Many parents to seek advice because of the mental changes. The mental changes cover a wide range from virtual imbecility to the mildest retardation, but this is usually associated with an asocial outlook, which is related to the convulsive episodes. Problems range from affect of movement ability, Persistent posturing and patterning movements, Stiffness of movements of several types, Floppiness and weakness, Limited useful range of movement, Intellectual difficulties and/ or motivation, Central sensory deficits, Epilepsy.

Conclusion

The term acute infantile hemiplegia denotes certain cases of hemiplegia of sudden onset in children, of a few weeks to 6 or more years of age. The outcome may be fatal; or there may be permanent hemiplegia with mental impairment. There may be slow partial recovery; or rapid and complete recovery. The term infantile hemiplegia is used to describe the clinical condition of the sudden onset of hemiparesis unassociated with intracranial hemorrhage and trauma. It causes significant mortality and morbidity. Infections are an important cause of neurological deficit, in the developing countries.

Once the injury has occurred, the symptoms should not worsen. However, because of lack of mobility, other complications can occur. Complications may include muscle and joint stiffness, and shortening of limbs, loss of aerobic fitness, muscle spasms, bed sores, pressure ulcers and deep vein thrombosis. Infantile hemiplegia persisting in an adult is a rare case.

We herewith present a 35 old male, who presented with seizures. 15 years ago he sustained head injury as he fell down from the tree. He was diagnosed with subdural hematoma and was evacuated through craniotomy, after which he developed residual right hemiparesis. Now the right hemiparesis is still persisting as residual hemiparesis on right side with shortening of limbs. A diagnosis of Post traumatic Infantile hemiparesis on right side was made.

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