Misdiagnosis of Cerebral Palsy - An Error in Paediatric Rehabilitation (A Case Series)
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Abstract: In paediatric rehabilitation, cerebral palsy is considered as one of the likely diagnosis for children presenting with spastic quadriaparesis and developmental delay. The following series emphasizes how in spite of advances in neuro-diagnostic imaging, appropriate history taking and clinical acumen are essential to avoid delay in the accurate diagnosis. In some conditions, the management can be entirely different and a delay in this can affect the functional outcome of the child undergoing rehabilitation.

Keyword: Cerebral Palsy, Cervical Myelopathy, Spastic Quadriaparesis

Introduction: Children with cerebral palsy form a significant patient population under any rehabilitation set-up. These children usually present with delayed developmental milestones along with pyramidal or extrapyramidal symptoms. However, misdiagnosis of cerebral palsy is well known in literature [1]. We present the following case series of three children where cervical myelopathy leading to spastic quadriaparesis was initially misdiagnosed as cerebral palsy and further discuss the relevant etiology for the myelopathy in these children.

Case 1:

Figure 1: MRI spine: Cervical cord thinning with cord hyperintensity at C2-C3
4 year old male child diagnosed earlier as cerebral palsy presented with poor voluntary control of all 4 limbs along with delayed motor milestones. His cognition, speech and bladder-bowel habits were age appropriate. Born to nonconsanguinous parents, birth history was significant for gestational diabetes for mother and caesarean section at 38 weeks for breech presentation. Birth weight was 3 kgs and postnatal period was uneventful. Parents noticed poor neck control and decreased movements of limbs at 6 months of age. On clinical examination, child had spastic quadriaparesis, poor head and trunk control with hyper-reflexia and extensor plantars. MRI brain was normal and MRI spine revealed thinning of cervical cord at cervico-medullary junction and cord hyperintensity opposite C2-C3 suggestive of cervical myelopathy with no CV junction instability (Figure 1). Hence, diagnosis of cervical myelopathy as possible sequelae of breech presentation was made. He underwent neurorehabilitation for ambulation training and hand function.

Figure 2: MRI spine: focal thinning with hyperintensity of upper cervical cord at C1-C2
4 year old male child earlier diagnosed to have cerebral palsy presented to the out-patient department with difficulty in ambulation and delayed motor milestones with age appropriate cognition and speech and occasional night time incontinence. He was second born child of nonconsanguinous parents, delivered at full term through cesarean section for breech presentation. Antenatal period had been uneventful. Birth weight was 3.5 kgs and he cried immediately after birth. Postnatal period was uneventful. On examination, he had spastic quadriaparesis, brisk reflexes, and increased head size in relation to chest circumference. MRI cervical spine done in view of absent perinatal insult and increased head circumference revealed focal thinning of upper cervical cord at C1-C2 with hyperintensity suggestive of myelomalacia (Figure 2). As there was no evidence of compression, possibility of remote insult at birth time was considered as the probable etiology.

CASE 3:

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CASE 3:
11 year old, female child presented with weakness of all 4 limbs since birth and delayed motor milestones associated with bladder-bowel incontinence. She also had restricted neck movements and short neck with decreased sensation on right half of body. Her cognitive abilities and speech were age appropriate. The only child of nonconsanguinous parents, she was delivered at 38th weeks, by caesarean section, section indication being rupture of membranes and decreased fetal heart rate. Birth weight was 3.5 kg and she required tactile stimulation for first cry. Post natal period was uneventful. She had recurrent episodes of urinary tract infection since 4 months of age. She was diagnosed to have cerebral palsy at this time because of decreased head control and poor voluntary control of limbs with history of probable birth asphyxia. She also underwent multiple soft tissue release for spasticity and deformity correction at 4 and half years of age and continued on physiotherapy. Due to worsening of spasticity, a second opinion was sought and considering short neck and limited range of neck movements, CT scan of cervical spine was done. It showed multiple CV junction anomalies including atlanto-occipital assimilation, basilar invagination and consequent cervicomедullary compression (Figure 3). She was surgically decompressed and cervical spine was stabilised. She has been on rehabilitation programme since then to improve her functional abilities and motor control.

Figure 3: MRI cervical spine: cervical cord compression with CV junction anomalies

Discussion:
Cervical Myelopathy in children can result from either congenital and developmental abnormalities or acquired changes [2]. Birth trauma and congenital anomalies of cranio vertebral junction are etiologies unique to pediatric age group. In neonates, the spinal cord is less elastic than vertebral column withstanding less distraction force during prolonged delivery. Hence, injury to cord can happen by atlanto-occipital assimilation, basilar invagination and consequent cervicomедullary compression [3, 4]. Apart from direct compression or distraction force, damage to vertebral artery can result in ischemia of cord leading to myelopathic changes [4]. Also any manipulation or instrumentation for delivery of head causing prolonged traction or rotation of spinal cord has greater likelihood of injury to the upper cervical cord. Contributing factors include weaker cervical musculature, ligamentous laxity, immature vertebral joints and immature growth centers, and horizontally inclined articulating facets which facilitates greater mobility of upper cervical spine and hence, increased susceptibility of injury to upper cervical spinal cord in infants. Birth related spinal cord trauma is usually encountered during forceps delivery or difficult extraction of after-coming head in breech presentation [3]. There has been a decrease in its incidence since the advent of caesarean section for managing the breech presentation and improved obstetric care. Despite this, birth related trauma to spinal cord especially cervical cord is seen on and off [3, 4]. The exact incidence of cervical myelopathy is difficult to determine due to delay in clinical suspicion especially in incomplete injury with no accompanying bony involvement [3]. Hence, previous studies have shown a delay in diagnosis up to 4.4 years of age from birth. Till that age, often these children get misdiagnosed as cerebral palsy Similarly, congenital anomalies of CV junction are

Diagnosed late due to complex regional anatomy, confusing radiological features and low clinical suspicion. The partial or complete fusion of arch of atlas to occipital condyles is called assimilation. This is often accompanied by condylar hypoplasia and congenital fusion of two or more distal cervical vertebra (C2-C3) [4, 5]. Anomalies can predispose odontoid process of axis to migrate upwards onto the assimilated atlas (basilar invagination) and cause compression of upper cervical cord [4, 5]. These children often have pyramidal signs namely, spasticity, hyperreflexia, muscle weakness, gait abnormality [4]. These pyramidal signs combined with low clinical suspicion and difficulty in distinguishing anomalies radiologically in infants due to immature and unfused bones, leads to misdiagnosis of cerebral palsy in them[6]. All the 3 children in this report had been diagnosed earlier as cerebral palsy. Apt clinical examination and appropriate correlation with history led to subsequent evaluation which revealed cervical spinal cord pathology. Hence, it is important to consider other differential diagnosis when a child presents with spastic quadripareisis with delayed motor milestones and to obtain detailed history along with a meticulous clinical examination. Diagnosis can be confirmed by radiological screening like MRI for neural structures or CT for bony pathology. It must be remembered that cerebral palsy is more of a diagnosis of exclusion and often is being over diagnosed.

Reference:

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