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Acute Flaccid Myelitis: Are We Vigilant Enough?

With the eradication of poliomyelitis from most countries, acute flaccid myelitis (AFM) due to non-polio enteroviruses and other viruses is an emerging disease. Besides the vaccine-associated paralytic polio, AFM outbreaks due to other viruses are also a hazard. AFM comprises of patients with acute flaccid paralysis (AFP), characteristically asymmetric limb weakness, with MRI suggestive of a spinal cord lesion in grey matter and spanning one or more vertebral segments [1]. Over the last decade, multiple outbreaks have been reported from countries such as USA, European countries, and Japan [1]. Two outbreaks have already been reported from India [2,3]. However, the pathogen testing was limited and inconclusive in both the cohorts. Even with ongoing AFP surveillance, AFM has not been frequently reported from India. Similar to Australia, we believe that there is misdiagnosis and under-recognition of AFM. During the initial disease course, AFM is frequently misdiagnosed as transverse myelitis due to an often extensive involvement of the spinal cord, not classically limited to the grey matter of the spinal cord [5]. Hence, there is a need for creating awareness regarding this evolving entity.

With many viruses involved such as EVD68, EVA71, etc. and poor yield of pathogen testing, it is often difficult to establish causality for AFM [1-3]. Therefore, it is time that patients with AFP should also be tested for other viruses beyond the poliovirus. This can later help in strengthening the AFP surveillance system. Survey studies for non-polio AFM throughout the country may be an initial step in this aspect, in the absence of active ongoing surveillance. However, the surveys need to be more robust to capture the epidemiological aspects of both AFM and associated respiratory/gastrointestinal illnesses. The key epidemiological parameters should include the whereabouts of patients (for source identification), age group, details of neuroimaging, and virological studies, contact tracing, etc. for patients in both the groups. Besides, AFM clusters and outbreaks need to be investigated meticulously to avoid an epidemic staring at us.

PRIYANKA MADAAN AND LOKESH SAINI*
Pediatric Neurology Division, Department of Pediatrics, Advanced Pediatric Centre, PGIMER, Chandigarh, India.
**drlokeshsaini@gmail.com*

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Acute Flaccid Paralysis in a Child: It Is Not Guillain-Barré Syndrome Always!

A 6-year-old-girl presented with complaints of difficulty in walking for 5 days. Initially, the child started limping on the left side, followed by unable to bear weight; within two days, the right lower limb also got involved and she became non-ambulatory. She also complained of dull aching pain in lower limbs, especially in the upper thigh, more on the left side. There was no history of proceeding febrile illness, trauma or intra-muscular injection. She was completely immunized as per the national immunization schedule. She had tenderness in the left flank, lower back and bilateral thigh, keeping the hips in a semi-flexed position, not allowing any passive movement or formal tone examination. Even knee jerks could not be elicited bilaterally. In the left hip joint power was 2/5 and 3/5 power in the right knee, left hip and knee joint. A clinical possibility of acute flaccid paralysis (AFP) was kept, with a differential diagnosis of Guillain-Barré syndrome (GBS), viral myositis, polymyositis, transverse myelitis, paralytic polio myelitis, Perthes disease, septic arthritis of the hip joint and pseudoparalysis due to unnoticed trauma, or with pelvis/femur fracture. On investigations, *X*-ray of the hips, nerve conduction study and serum creatine phosphokinase were normal. Ultra-sonogram revealed a heterogeneous collection in left iliopsoas muscle, extending to the pelvis and inguinal region. Pus was drained by percutaneous pigtail catheter and she responded favorably to intravenous vancomycin and she was able to walk after three days.

Although predominant causes of painful, hyporeflexic weakness of bilateral lower limbs are GBS and viral myositis, often pseudoparalysis due to trauma, scurvy or referred pain from the loin, lower back or hip joint may mimic GBS, thereby causing diagnostic confusion [1]. The classic triad of psoas abscess (fever, flank pain, and limitation of hip joint movement) can be found only in 30% of patients [2].

The atypical presentation with bilateral painful gait instability in absence of fever, trauma or intramuscular injection in our case clinically resembled GBS or pathology localized to lumbosacral plexus or spinal cord. However, instead of performing costly and tedious investigations like MRI and nerve conduction study, simple ultrasonography may clinch the correct diagnosis easily. Pseudoparalysis in children under 15 may be caused due to various etiologies like skeletal trauma, lymphadenitis or muscle aches from a viral illness, transient synovitis, septic arthritis, osteomyelitis, pyomyositis, fasciitis, cellulitis, rheumatological diseases such as juvenile idiopathic arthritis, acute rheumatic fever and malignancies like sarcoma and leukemia [3,4]. Hence, atypical presentation of iliopsoas abscess requires a high index of suspicion on part of pediatricians, to establish a timely diagnosis.

PRATEEK KUMAR PANDA AND INDAR KUMAR SHARAWAT*
Pediatric Neurology Division, Department of Pediatrics, All India Institute of Medical Sciences, Rishikesh, India.
**sherawatdrindar@gmail.com.*

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What is the Determinant of 2019 Novel Coronavirus Prognosis in Children?

We read the article by Li, et al. [1] with interest and would like to offer some observations about the study based on the current literature.

In the pathogenesis of a standard viral infection, the pathogen's contact with the mucosa is initially followed by an innate immunity response (macrophage, antigen presenting and natural killer cell). Subsequently, adaptive immunity comes into play and is responsible for the elimination of infected cells, activation of the antibody response, and production of memory T-cells. T-cells are the primary decisive element in adaptive immunity capability. For this reason, the adaptive immune response mediated by the thymus is a process that regulates the immune response responsible for preventing invasive damage from a virus. Therefore, the thymus is the most influential organ in the transmission of viral disease [2].

The thymus generally decreases in function and anatomically shrinks with age. This function and size loss becomes specially prominent after the age of 50 [3]. Thymic involution and the gradual decrease in T-cell count and ability with age are together termed as immunosenescence [4]. The primary reason for morbidity and mortality in COVID-19 cases is due to lung manifestation. The primary reason for a frequently severe clinical presentation in patients of ages 50 and up is thought to be due to a deficient, irregular and uncontrollable antiviral response as a result of thymus involution and immunosenescence. Important factors in achieving an adequate immune response are an increase in thymus activity and T-cell action along with immune system coordination.

When examining the critical COVID-19 cases in the literature, the male gender seems to be more common; this is speculated to be due to greater tobacco use and ACE-2 receptor expression. The literature also shows that thymic involution is more apparent in males compared to females. This difference in thymic involution indicates that males face a greater extent of immunosenescence. We believe this mechanism might be responsible for clinical worsening in males [5].

We believe that thymus regression and lung immunosenescence are the main deciding factors of lung involvement depth in adult COVID-19 patients. But, we do not know thymus activity in children cases with severe COVID-19. We feel that there is a need to examine the patients for thymus size, and look for association between thymus size and the severity of lung involvement.

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SADIK YURTTUTAN
Neonatal Intensive Care Unit, KSU University SUA Hospital, Kahramanmara, Turkey.
sdkyurttutan@gmail.com

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