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Pediatric Crier
Department of Pediatrics Newsletter

http://www.upstate.edu/gch/academics/newsletter.php

New Faculty: New Special Needs Doc

We are pleased to welcome and introduce one of our new providers in the Golisano Center for Special Needs, Dr. Christina Alaimo.

Dr. Alaimo is a Board-Certified Behavior Analyst (BCBA-D) and NYS Licensed Behavior Analyst with a NYS limited permit to practice psychology. She received her doctorate in behavior analysis from The Graduate Center, City University of New York in 2021. She has extensive experience assessing and treating a range of pediatric feeding disorders. Her research has primarily focused on staff and caregiver training in interventions to treat children's food selectivity. Dr. Alaimo tells the Crier she is excited to join the dedicated team at the Golisano Center for Special Needs Pediatric Feeding Program and continue to support children and their caregivers.

A Match Made in Heaven

As most of you probably heard, March 18th was Match Day and we had a very successful match! We are pleased to introduce you to the 15 members of the Pediatric Class of 2025.

David Cea-Flores, St. George's
Mohammed Doklaijah, Beirut Arab University, and peds residency at Hamad
Tara Dowd, Royal College, Ireland
Ghina Fakhri, American Univ of Beirut (including herPeds Residency)
Maxence Gilles, Idaho Coll of Osteopathic Med
Michael Kennedy-Yoon, University of Utah
Lamia Jouhar, University of Damascus and Peds Residency at Hamad
Amna Khan, Shifa College, and West Virginia Univ
Michael Markel, Technion Israel Institute of Technology
Areeb Mustansar, Aga Khan University
Precious Sanni Adeniyi, University of Lagos
Sara Taha, University of Khartoum/Cork Univ, Ireland
Khalil Tamr Agha, Damascus/Amer Univ Beirut (Peds Res), Child's Mercy KS (pulm)
Deepa Thomas, A.J. Institute of Medical Sciences
Lauren Waichenberg, Ben-Gurion University of the Negev

Don't miss Dr. Nelsen's welcome on Twitter and Instragram
https://www.instagram.com/upstatepedsresidency/

https://twitter.com/upstatepedsres2

More New Residents!

In addition to our new interns, we are excited to be able to introduce two additional residents who will be joining the team in the next month.

First, we want to welcome Dr. **Mohamed Khallaf** who will be starting on April 11th and is filling the open spot in the current PL1 year. Mohamed completed his medical school degree and his pediatric residency at Cairo University. Most recently he has been a clinical research assistant at Stead Family Children's Hospital in Iowa.

Second, we want to welcome **Dr. Mayank Sharma** who will be joining us on May 1st and will be joining the Class of 2023. Mayank is a graduate of Calcutta National Medical College and then completed his PL1 and PL2 year at Jackson Memorial Hospital in Miami before transferring to our program.

We are excited about all 17 of our new residents but be sure to keep an eye out for Mohamed and Mayank and welcome them to the peds family!

PRESSing On

We previously reported that PL2 Laaibah Ejaz was selected to participate in the PRESS (Pediatric Residents Experience in the Subspecialties) at Stanford. Even as we go to print she is there on campus in the PICU. Before leaving, she let us know, "I found out today that only 5 residents all across the country were selected to be part of the Stanford PRESS program (picture attached). I wanted to share this happy piece of info with you and I'm so honored to be representing Upstate… I hope to make our department and our institute proud!"

We just heard again from Laaibah and received the following update:
"I'm halfway done with my Stanford away rotation and wanted to share a few pictures. I'm having a great time working hard in the PICU and have been receiving excellent feedback from my attendings which makes me appreciate my Upstate training even more! The group picture is of the 4 residents who got selected for the PRESS program from all over the country.

We are very proud of Laaibah and hope she has a wonderful experience at Stanford.

How the West was Won

Last month we shared all of the exciting fellowship matches. In the meantime, PL2 Billy Hall has signed a contract for an exciting practice opportunity in 2023. He was offered a pediatric outpatient provider position with Valley Children's

Health in Bakersfield, California. Billy tells the Crier that most of his family is on the west coast so this works out perfectly! Congratulations, Billy!

In the News:

The UPAC Buzz

The Crier is pleased to introduce a new publication from our outpatient clinic! You might call it our sister newsletter. Dr. O'Malley shared the following: "I wanted to share with you that we have started a UPAC Newsletter, largely inspired by the Peds Crier. I lobbied for the companion name of the UPAC Screamer, but we landed on the UPAC Buzz. Enjoy!

Click here: **March 2022 UPAC Buzz**
https://sway.office.com/U1JAgv7Veh0kOj2x?ref=Link

Radiothon raises $170,000 for the GCH

Thanks to all who participated or donated to this year's Radiothon for Kids which raised $170,000 in two days of fundraising with more than 950 community donations. The radiothon was held Feb. 24 and 25. Funds raised this year will be used primarily for unmet needs like meals and gas cards for families with hospitalized children, and items for the Child Life Program.
https://www.upstate.edu/news/articles/2022/2022-03-04-radiothon.php

The CURE Award

Our very own Kimberlee Garver, a pediatric social worker with Upstate's Cystic Fibrosis Clinic, has been named winner of the CURE (Caring, Understanding, Relationship, Education) Award from the Central New York Cystic Fibrosis Foundation. She received the honor at the foundation's Feb. 10 annual meeting.

The Autism Intervention Study

Our very own Dr. Hank Roane was featured in Upstate Online after receiving a grant for a study that is intended to help in discovering a defining standard of care for people with autism. You can read all about it at:
https://www.upstate.edu/news/articles/2022/2022-03-07-roane.php

Education Corner

Dr. Jennifer Nead

We would like to thank the following residents and faculty who were instrumental in making the clerkship's formative standardized patient exam (SPE) a huge success this academic year. Student feedback has been overwhelmingly positive!!! Students genuinely appreciate all of the time, feedback, and words of wisdom that these pediatric educators have provided in prepping them for their summative SPE. We could not hold this invaluable educational session without all of your help!

Faculty: Anjali Sura, Andrew Osten, Gloria Kennedy, Joan Pellegrino, Kate Okhman, Melissa Schafer, Elizabeth Lodge, Tyler Greenfield, Mitchell McKinnon, Rhonda Philopena, Moshe Roberts

Residents: Vaishali Adlakha, Colleen Feeney, Simi George, Andrew Brooks, Holly Stacey, Evan Ho, Jason Silkey, Sophie Duron, Ruby Sangha, Laura Mejia-Connolly

Andrew Brooks, despite his very busy PGY2 schedule, consistently signed up for TWO SPE sessions EACH clerkship rotation block! Andrew, please reach out to Chris Kuehnle for a small token of our appreciation.

We are currently looking for resident and faculty volunteers to help out with our formative SPE for this upcoming academic year! The formative SPE occurs each clerkship rotation block (i.e. every 5 weeks). Volunteers role play the caregiver of a newborn with jaundice. Students elicit history and exam findings from the caregiver. After this 20-minute timed session, the resident/faculty volunteer provides the student with feedback using provided history/exam and communication skills checklists. Training is provided and the formative SPE session usually lasts about 40 minutes. Volunteers reach out to their assigned student(s) and schedule an in person or Webex session that fits their individual schedules. Volunteers can sign up for as many sessions as they want during the academic year. This is a great educational activity to list on CVs. If you are interested, please email Christine Kuehnle.

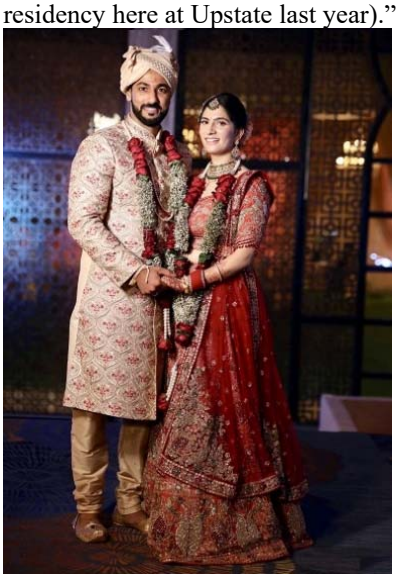
Thank you all for everything that you do! - Jen Nead, Leah Bennett, and Chris Kuehnle

Fellow Publication!

Congratulations to our Peds ID fellow, Dr. Danielle Daniels (and her lovely assistant, Dr. Greg Conners) on their recent publication in Pediatric Emergency Care: A Review of the Diagnosis and Management of Acute Flaccid Myelitis in the Emergency Department (attached).

Tying the Knot in India

Congratulations to Vaishali Adlakha Harne who went home to India last month to get married! The beautiful bride tells the Crier, "Even though Prateek and I got legally married some time ago, we were glad we got the opportunity to celebrate ceremonially with our families in New Delhi, India on March 12th and 13th. Some of our residency friends were able to join us virtually! Prateek is a first year adult GI fellow in Texas. I'm excited to start my fellowship in Texas in July as well. (Prateek's full name is – Prateek Harne, he graduated from IM residency here at Upstate last year)."



APRIL BIRTHDAYS

4/2 Jina Patel
4/10 Shehzad Ahmed
4/18 Elena Wolner
4/19 Angela Wratney
4/20 Mide Ajagbe
4/24 Jenica O'Malley

The Crier has been crying for over 20 years now. We thought we would end this issue with the opposite of a crier with a big smile from Luca. Apparently, life in the Meneses-Duron home is pretty good!

A Review of the Diagnosis and Management of Acute Flaccid Myelitis in the Emergency Department

Danielle K. Daniels, MD and Gregory P. Conners, MD, MPH, MBA†*

Abstract: Since 2014, biennial rises in acute flaccid myelitis (AFM) have brought attention to this rare but debilitating condition. Children with AFM typically present with acute onset, flaccid weakness accompanied by longitudinally extensive gray matter injury demonstrated on magnetic resonance imaging. A clearer understanding of the epidemiology and suspected pathogenesis of AFM may result in increased recognition. The purpose of this review article is to guide emergency physicians in recognizing key clinical features, initiating diagnostic evaluation and providing appropriate interventions for children with suspected AFM.

Key Words: acute flaccid myelitis, enterovirus D-68, paralysis

(*Pediatr Emer Care* 2022;38: 126–132)

TARGET AUDIENCE

This CME activity is intended for all practitioners who care for pediatric patients presenting with symptoms consistent with acute flaccid myelitis, which may include pediatricians, general practitioners, pediatric emergency physicians, general emergency physicians, and pediatric intensive care physicians.

LEARNING OBJECTIVES

At the completion of this article, the reader should be better able to:

1. Explain the epidemiology and suspected pathogenesis of patients with acute flaccid myelitis (AFM).
2. Identify the key clinical features that will aid in prompt diagnosis.
3. Propose appropriate diagnostic evaluation and management of patients presenting with AFM.

In the late summer of 2014, an alarming number of children were presenting to emergency departments with severe respiratory illness. Further investigation revealed circulation of a specific strain of enterovirus, enterovirus D-68 (EV-D68).[1,2] First identified in 1962, EV-D68 cases were infrequently reported before 2014.[3,4] Amidst this outbreak, an unusual cluster of children presenting with acute onset paralysis was identified.[5] The Centers for Disease Control and Prevention (CDC) developed a case definition for what is now termed acute flaccid myelitis (AFM): "acute onset of flaccid limb weakness plus imaging demonstrating involvement of the gray matter of the spinal cord in one or more vertebral segments."[5] Although increased recognition has brought attention to other possible infectious etiologies, EV-D68 remains the likeliest cause of the recent rise.

Given the rapid progression of paralysis and associated respiratory failure of AFM, prompt recognition facilitates specimen collection that may aid in the diagnosis and early provision of appropriate support. Although the mainstay of treatment is supportive care, development of future therapies is under investigation.

CASE PRESENTATION

A 7-year-old previously healthy boy presents to the emergency department with several hours of left arm weakness, right facial droop, stridor, and drooling. Mother reports 6 days of preceding rhinorrhea, nasal congestion, and cough, all of which were gradually improving. Yesterday evening; however, he developed new-onset fever and was complaining of "buzzing" in his left arm. This morning, he developed right-sided facial droop and difficulty swallowing his secretions, with trouble breathing. He was unable to raise his left arm above his head. Mother brought to the emergency department at the suggestion of their pediatrician.

Upon arrival to the emergency department, the patient is unable to speak in full sentences and has stridor. Comprehensive neurologic examination reveals right facial nerve weakness, left arm weakness, and to a lesser extent, right arm weakness with diminished reflexes. With concern for bulbar weakness and inability to protect his airway, he is electively intubated, and a comprehensive evaluation is initiated.

EPIDEMIOLOGY AND PATHOGENESIS

As the name suggests, AFM involves rapid progression, often over hours to days, of limb weakness. This is the result of injury to the anterior horn cells of the spinal cord, and occasionally the motor nuclei of the brainstem, as demonstrated on imaging.[6] Although the pathogenesis remains uncertain, epidemiologic trends suggest the role of a viral trigger. The majority of AFM cases have been reported in late summer and early fall, mostly between August and November (Fig. 1).[8] Since the CDC began monitoring in 2014, peaks have been biennial with 654 confirmed cases to date.[7] Although a 2020 peak was anticipated, AFM rates remained relatively flat. This unanticipated decline coincides with the decrease in endemic respiratory viruses during the COVID-19 pandemic, further supporting a viral etiology.[9,10]

Children have suffered from acute flaccid paralysis of various causes, including infection and neuroinflammatory conditions, for centuries, with occasional outbreaks occurring in waves associated with infectious triggers.[11] The polio virus outbreak in the 1950s brought public attention to paralysis as a complication of viral infection.[11] After the near eradication of polio, geographic specific outbreaks of acute flaccid paralysis have been associated with West Nile virus and enterovirus A71 (EV-A71).[10,12] In 2012, a cluster of 5 children in California, all previously vaccinated against poliovirus, developed a polio-like illness. Two eventually tested positive for EV-D68, suggesting a role for this virus.[13]

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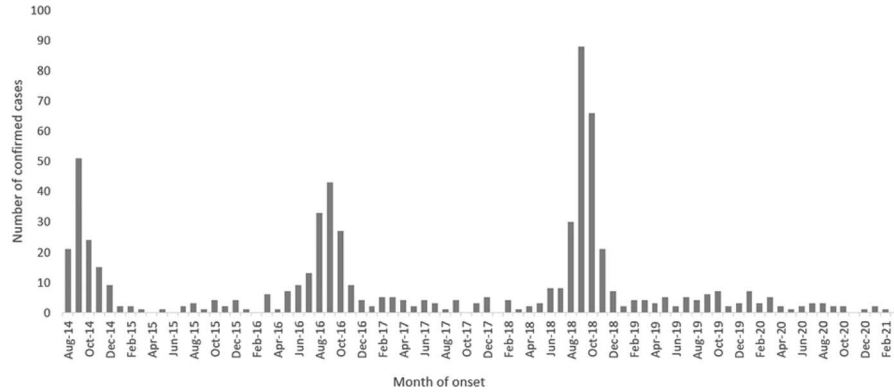


FIGURE 1. Confirmed AFM cases reported to the CDC from August 2014 through April 2021.[7]

The AFM cases associated with EV-D68 have since been identified around the world.[13–15] During peak years, AFM cases are temporally and geographically associated with circulation of enteroviruses, specifically EV-D68 and, to a lesser extent, EV-A71.[3,14–16] Although EV-D68 itself is not found within the cerebrospinal fluid (CSF), higher levels of EV-D68–specific antibodies are found in the CSF of AFM patients than in controls.[17] Animal models have demonstrated the neurotropic nature of EV-D68, with viral invasion resulting in neuronal injury, suggesting a role for its pathogenicity.[18] Furthermore, mice directly infected with EV-D68 can exhibit AFM-like paralysis.[19]

PRESENTATION

Recognition of AFM is critical for the emergency department provider. Two-thirds of patients seek initial care in the emergency department, with over 50% requiring ICU admission.[20] The majority of reported AFM cases occur among the pediatric population. The median age for AFM is 6.3 years with a slight predilection for male sex (61%).[21] Patients typically experience a preceding viral prodrome, with fever, respiratory, or gastrointestinal symptoms. In cases associated with EV-A71, children may have lesions involving the hands, feet, or mouth before or at the time of neurological weakness.[22] The median time from preceding febrile illness to limb weakness is 6 days.[23] Children gradually recover from their initial febrile illness as weakness suddenly emerges.[24] In the days before onset of weakness, patients may experience return of fever plus meningeal signs such as headache or neck pain. Older children may articulate neuropathic-type pain in affected limbs.[19] Less commonly, patients will report preceding rash.[23]

Children with AFM typically present with weakness or complete paralysis of one of more limbs that develops rapidly over hours to days.[21] The severity of deficits can vary greatly, from subtle weakness of a single limb to quadriplegia associated with autonomic dysfunction and respiratory failure.[25] Although upper extremities are more commonly affected, just over one-third have involvement of all 4 limbs.[8] Weakness tends to be asymmetric, and more commonly affects the proximal muscle groups than the distal.[26] At the onset of weakness, some patients present with cranial neuropathy (33%), altered mental status (28%), or respiratory distress requiring mechanical ventilation (33%).[8,20]

HISTORY AND PHYSICAL EXAMINATION

When obtaining a history, young children may be unable to express a complaint of "weakness," therefore, it is important to ask age-specific questions that assess function. For example, is the child not feeding himself, dropping items, or using one limb less often?[23] Are they having difficulty holding up their head or swallowing their secretions? Have parents noticed a hoarse or weak cry? Anuria or constipation should raise concern for urinary/bowel retention, which is more commonly associated with EV-A71 versus EV-D68.[20,21]

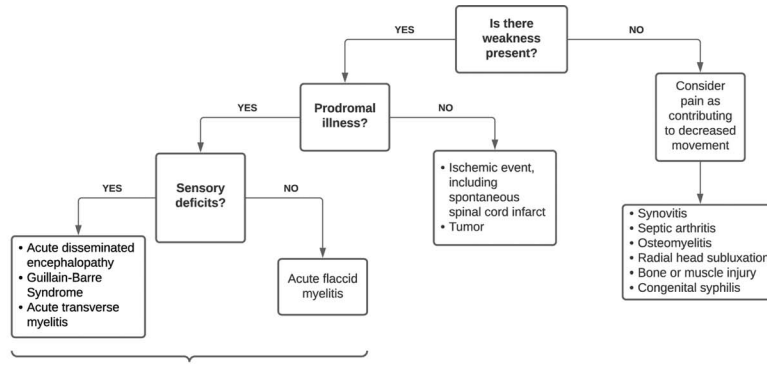
It is important to perform a complete physical examination with an age-appropriate neurologic examination. Frequent reevaluation is necessary since neurological decline can progress rapidly, often over the span of hours.[24] Children may be unable to raise their hand above their head, or to raise their knee. Neurologic examination may reveal flaccid, hypotonic muscles with hyporeflexia. As mentioned previously, approximately one-third of patients present with cranial nerve dysfunction, requiring careful evaluation for associated bulbar weakness.[8] Associated autonomic instability resulting in temperature or blood pressure instability has been described.[27] Sensory deficits, associated seizures, and encephalopathy are unusual features and should prompt evaluation for alternative diagnoses.[28]

DIFFERENTIAL DIAGNOSIS

There is a broad differential diagnosis for the child presenting with acute onset weakness. Acute flaccid myelitis should especially be suspected when a preceding illness is reported, especially in the late summer to early fall months. In addition to clinical history, the physical examination, imaging and CSF studies will help differentiate AFM from mimicking diagnoses (Fig. 2). In nonverbal children, pseudoparalysis secondary to limb pain may result in an unwillingness to move the arms or legs. Consider diagnoses such as synovitis, septic arthritis, osteomyelitis, radial head subluxation, or limb injury. If true weakness or paralysis is detected, a history or prodromal viral illness and the temporal progression of disease can help guide your diagnosis. Conditions, such as stroke or tumor, are unlikely to present with preceding illness. Similar to AFM, spinal cord infarction may present with rapid neurological decline over hours, whereas malignancy may present with a slower, more indolent decline.

Children with Guillain-Barre syndrome may report a preceding illness. The associated weakness tends to be symmetric, progressing from the lower extremities, whereas AFM typically presents with asymmetric weakness, more commonly involving the upper extremities. Unlike AFM, sensory deficits, such as paresthesia, are common in Guillain-Barre syndrome.[28]

Both acute disseminated encephalomyelitis and acute transverse myelitis can follow a prodromal illness. Sensory deficits are common in both, whereas seizures or associated encephalopathy is seen with acute disseminated encephalomyelitis.[28] Acute transverse myelitis may result from demyelinating conditions,



	CSF Findings	MRI Findings
Acute flaccid myelitis	Mild to moderate pleocytosis, +/- elevated protein	Gray matter predominant lesions, diffuse central gray matter involvement early, localized to anterior horn cell later
Acute disseminated encephalopathy	Mild to moderate pleocytosis, +/- elevated protein	Discrete gray and white matter involvement
Guillain-Barre syndrome	Normal to mild pleocytosis, elevated protein	Normal cord, may involve nerve roots
Acute transverse myelitis	Mild to moderate pleocytosis, +/- elevated protein	Discrete lesion involving the white matter with or without gray matter

FIGURE 2. Differential diagnosis for AFM.

such as myelin oligodendrocyte glycoprotein (MOG)-associated myelitis or neuromyelitis optica (NMO). Although these initially present with flaccid paralysis, spasticity with hyperreflexia will eventually develop.[24] Cerebrospinal fluid findings and imaging can further help differentiate AFM from these conditions.[14]

DIAGNOSTIC TESTING

Although the CDC has created a case definition for the purposes of surveillance, there are no standard diagnostic criteria for AFM.[28] The diagnosis is supported by the presentation, neuroimaging, and laboratory investigations. A plan for performing the necessary imaging and specimen collection should be individualized for each patient. Some, such as magnetic resonance imaging (MRI) and lumbar puncture, are best performed early in the disease course. These may require sedation or general anesthesia with endotracheal intubation, depending on the patient's ability to cooperate and risk of respiratory failure because of muscle weakness. Other tests are less invasive but may be of more value when performed later. Pediatric neurologist and infectious diseases specialists can help guide diagnostic decision making.[14]

Magnetic resonance imaging of the spinal cord and brain is essential in confirming the diagnosis. Limited scanner access, or the need for sedation or general anesthesia, may limit the ability to obtain imaging in the emergency department setting. It is important that the patient be evaluated in a facility with the capacity to safely obtain imaging in a timely fashion to confirm the diagnosis.[24] The characteristic finding is T2 hyperintensity predominantly involving the gray matter of the spinal cord (Fig. 3). These lesions are longitudinally extensive, often involving more than 3 segments.[29] In the acute stage, the hyperintensity may be ill-defined with diffuse central gray matter involvement. As time progresses, this may become more clearly demarcated, localized to the anterior horn cells.[6] The cervical and thoracic spines are the most commonly involved regions of the cord.[28] There may be involvement of the brainstem, although supratentorial lesions are rare and should prompt evaluation for alternative diagnoses.[29] Since early changes may be subtle or even read as normal, neuroimaging should be repeated if there is sufficient ongoing clinical suspicion for AFM.[28]

Laboratory investigations should include collection of CSF, respiratory specimens (nasopharyngeal and oropharyngeal swabs), serum, and stool. Earlier collection of specimens may increase their diagnostic yield.[23] Cerebrospinal fluid pleocytosis is common, often with a lymphocytic predominance.[16,29] Cerebrospinal fluid protein levels may be mildly elevated. When available, CSF should be sent for meningitis and encephalitis PCR panels to evaluate for alternative infectious etiologies. Respiratory samples should be sent for enterovirus RT-PCR and tested for other common respiratory viruses. Although a negative result does not exclude AFM, there is emerging evidence that virus detection is associated with more severe illness and poor recovery.[30] Serum

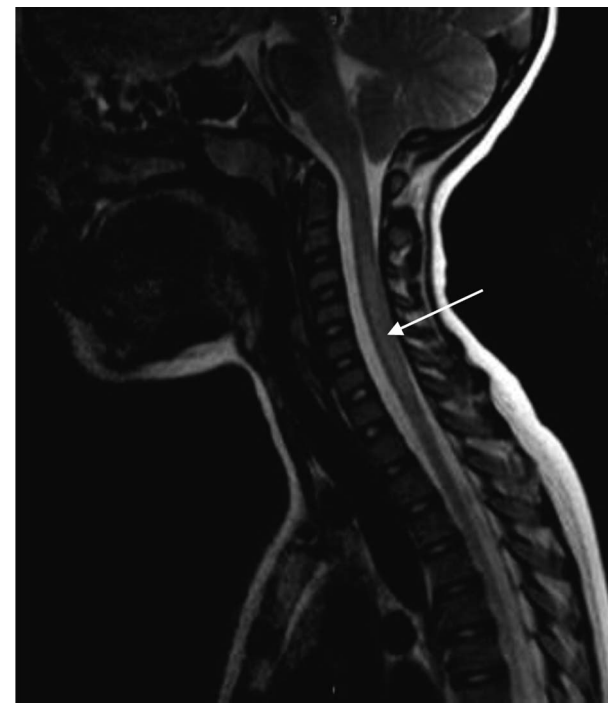


FIGURE 3. MRI cervical spine in a child diagnosed with AFM, demonstrating an ill-defined T2 hyperintensity spanning the cervical spine with involvement of the gray matter and to a lesser extent, white matter.

testing to evaluate for alternative diagnoses should include anti-MOG IgG and NMO IgG. Depending on the geographic location, serum and CSF antibody testing for West Nile Virus should be considered. Stool samples may be sent for enterovirus PCR, although the yield remains low for EV-D68 (higher among patients with EV-A71).[28] To assist in the diagnosis, as well as aid in continued research, it is recommended that specimen collection be coordinated with the local health department, who can, in turn, coordinate with the CDC.[31]

ACUTE MANAGEMENT

Currently, the mainstay of treatment of AFM is supportive care. As discussed previously, neurologic decline can progress rapidly; careful evaluations for cardiorespiratory compromise are critical. Management of respiratory failure, hemodynamic instability and urinary retention in the setting of autonomic dysfunction is essential. Once the diagnosis is suspected, transfer to a facility capable of both appropriately testing and caring for critically ill pediatric patients is necessary. Persons under investigation for AFM should be reported to the local health department as soon as possible. Consultation with neurology and infectious diseases specialists can guide decision making in testing and initiating therapies. The AFM Physician Consult and Support Portal allows medical professionals to seek consultation with neurologists specializing in AFM.[32] No FDA-approved pharmacologic interventions have been shown to be effective in preventing the progression of paralysis. The most commonly used therapies include intravenous immunoglobulin (IVIG), corticosteroids, and therapeutic plasma exchange.[25,28] Of these, IVIG is the most widely accepted. The potential benefit of IVIG is suspected to arise from pooled neutralizing enteroviral antibodies found amongst the general population.[33] In mouse models, early use of IVIG has resulted in less severe neurologic outcomes; however, this effect not been observed in humans.[34] There is not sufficient data to support or refute the potential benefits of corticosteroids or therapeutic plasma exchange.[35] There is no indication for use of fluoxetine, antiviral medications, interferon, or other immunomodulatory therapies.[35]

Rehabilitation should begin early and is considered an essential component to the patient's recovery. Early rehabilitation may improve long-term neurologic sequalae.[36] Select patients may benefit from nerve or tendon transplants, therefore early referral to an experienced center may optimize the potential for positive results.[37]

PROGNOSIS

Although mortality associated with AFM remains low, morbidity due to neuromuscular impairment is common. Approximately three-quarters of children will experience some degree of residual physical limitation resulting in impairment of daily function.[38] Long-term data, however, is lacking given the relative novelty of the diagnosis. The extent of recovery appears to correlate with the underlying etiology, with EV-A71–associated cases demonstrating more rapid and complete recovery then those with EV-D68.[22] Although most recovery of function occurs within the first several months, continued recovery may occur months to years beyond the initial diagnosis.[38] For those with severe residual deficits, nerve or tendon transfers have been beneficial in regaining function.[37] Intense rehabilitation may aid in the process of continued recovery.[36] Future development of enteroviral vaccines, targeted antiviral therapies or EV-D68 monoclonal antibodies, may eventually lead to AFM prevention or improved recovery.[39]

DISPOSITION

If AFM is suspected, stabilization, investigation and admission are necessary. Patients suspected of having AFM should be transferred to a facility capable of providing high-level pediatric care. Progression to respiratory failure should be considered in deciding whether the patient requires admission to the intensive care unit. Close monitoring for hypoxia and hypercarbia, as well as testing negative inspiratory force, can aid in medical decision making.[28]

CASE CONTINUED

After intubation, the patient is admitted to the pediatric intensive care unit for further evaluation and management. Pediatric neurology and pediatric infectious diseases services are consulted. After discussion with consultants, IVIG and methylprednisolone are administered intravenously. Magnetic resonance imaging of the brain and spine reveals T2-weighted hyperintensity within the pons, medulla, and cord at the C4 level through T4, involving the gray matter and, to a lesser extent, white matter. Lumbar puncture is performed and notable for pleocytosis with 52 WBC (L 50 N 31 M19), protein 66 mg/dL, and normal glucose. Cerebrospinal fluid PCR pathogen panel is negative. Cerebrospinal fluid bacterial culture is eventually negative. Respiratory viral panel (PCR) is positive for rhino/enterovirus. The respiratory specimen is sent to the state laboratory for further serotyping, confirming EV-D68 infection.

The patient has a prolonged, 1-month hospital stay with only mild improvement in strength of the forearm. He is extubated on the seventh day of admission, however re-intubated shortly thereafter for bulbar weakness with inability to protect his airway. Two weeks into the patient's admission, tracheostomy and G-tube are placed.

Additional evaluations include EMG, demonstrating severe acute motor axonal neuropathy with early signs of reinnervation affecting the upper extremities. Additional serum studies reveal negative GM1-antibody, anti-MOG antibodies, NMO antibodies and West Nile Virus antibodies.

Physical therapy is initiated early in the patient's course and the patient is eventually discharged to a rehabilitation center. From the rehabilitation center, a referral is made to a center specializing in nerve transposition surgery. Over the course of 1 year, the patient demonstrates slow return of function of his right arm, and to a lesser extent his left arm. He is decannulated and continues to work with speech therapy with ongoing evaluation to assess his risk for aspiration.

CONCLUSIONS

Acute flaccid myelitis should be suspected in patients presenting with acute onset, flaccid limb weakness. Even when findings are subtle, AFM should be treated as a medical emergency, as neurologic dysfunction can progress rapidly, resulting in respiratory failure. Awareness of the epidemiology and key clinical features of AFM can increase recognition of this rare, debilitating condition. As soon as AFM is suspected, MRI imaging and collection of appropriate laboratory specimens can help solidify the diagnosis, as well as rule out other conditions on the differential. Pediatric neurology and infectious diseases consultations can help guide diagnostic and therapeutic decision making; however, the cornerstone of treatment remains supportive care. Although long-term neurologic sequalae are common, rehabilitation and nerve transfers may facilitate recovery.

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