Pediatric Invasive Group A *Streptococcus* Infections with Central Nervous System Involvement: A Single Institution Case Series in Southern New England

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ABSTRACT

Pediatric invasive Group A *Streptococcus* (iGAS) infections are rare, affecting sterile sites including the central nervous system (CNS), and cause significant morbidity and mortality. An increased incidence of pediatric iGAS infections, including cases with CNS involvement, has been noted following the COVID-19 pandemic both domestically and internationally. Regional New England iGAS data specific to pediatric populations have been limited. This case series describes three pediatric cases of iGAS with CNS involvement which presented within the same two-month period in early 2025, all from within 20 miles in Massachusetts. All were initially admitted to the pediatric intensive care unit, patients had meningitis and subdural collections on brain imaging, and subsequently required at least four weeks of intravenous antibiotics. One underwent surgical intervention. All three had seizures requiring long-term anti-epileptic therapy, and each had residual symptoms, including seizures, focal weakness, and developmental delay. GAS infections with intracranial involvement are often severe and life-threatening. This series of three pediatric intracranial iGAS cases is particularly unique due to their similar presentations, timing, and geographic proximity. With recent literature indicating rising rates of iGAS infections globally and our regional experience, GAS should be considered a potential culprit for patients presenting with invasive bacterial infections.

KEYWORDS: Invasive Group A *Streptococcus*; intracranial infection; bacterial meningitis; subdural empyema

INTRODUCTION

Infections caused by *Streptococcus pyogenes*, or Group A *Streptococcus* (GAS), are typically benign and commonly present as throat or skin infections. Invasive disease is defined as an infection of sterile sites, often requiring prolonged hospitalizations and antibiotic therapy.[1] These may more commonly include bacteremia and cellulitis, and less commonly meningitis and/or central nervous system (CNS) disease.[2-4] Historically, invasive GAS (iGAS) disease has been rare, especially in pediatric patients. In the United States (U.S.) from 1997 to 2014, the annual incidence of pediatric iGAS ranged from 1.4 to 2.3 cases per 100,000.[5] It is estimated that meningitis and/or CNS disease accounted for approximately 1.9 to 5.2% of annual pediatric iGAS cases.[5]

The COVID-19 pandemic saw a 28% reduction in iGAS cases in the U.S.,[6] followed by a surge in cases both domestically[7,8] and internationally.[9-11] This increase is attributed to a historic low in GAS infections during the pandemic, most likely due to social distancing and masking requirements, leading to reduced population immunity, followed by resurgence of viral respiratory infections pre-disposing iGAS after the lifting of mandates.[4,7,8] Limited data on the regional burden of iGAS in several New England states (Massachusetts, Vermont, and Rhode Island) are consistent with the aforementioned trends in iGAS incidence.[12-14] However, little is known about the regional burden of iGAS specific to the pediatric population.

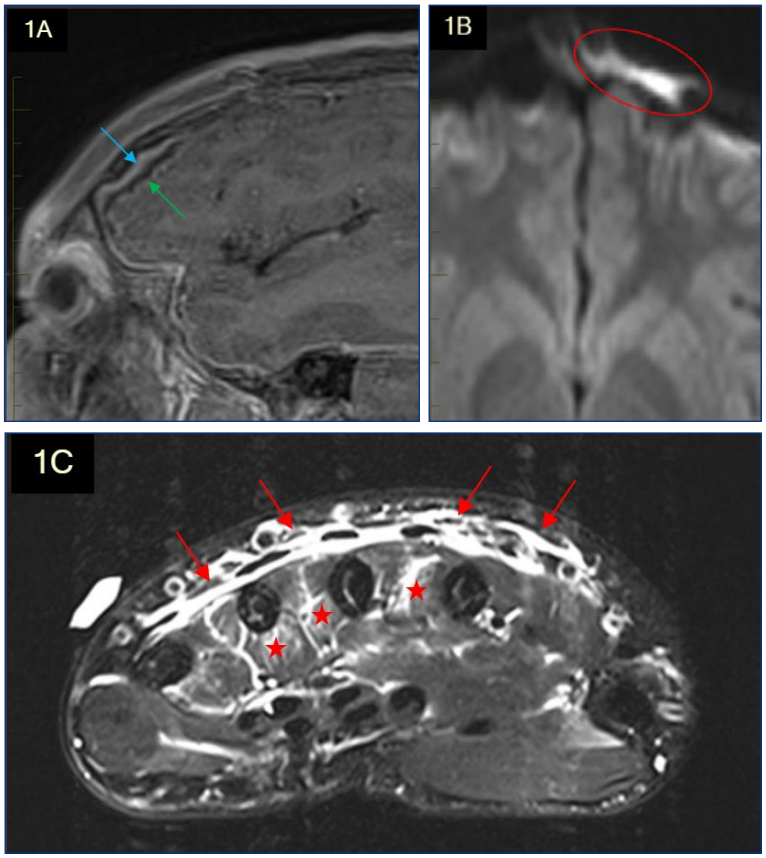
We describe three pediatric cases of iGAS, all with CNS involvement, occurring in patients from nearby municipalities in Massachusetts, within the same two-month period in early 2025: (1) a 12-year-old male with GAS bacteremia, meningitis, subdural empyema, and tenosynovitis; (2) a 9-year-old female with GAS sinusitis complicated by meningitis, subdural empyema and abscess; and (3) a 3-month-old female with GAS meningitis and bacteremia.

Case 1

A 12-year-old male with a past medical history of migraines presented to the Emergency Department (ED) with a four-day history of progressive headache and one day of vomiting, fever, neck pain, and sore throat. Household members had mild respiratory infections the week prior. The patient had no recent travel, animal bites or scratches, or consumption of raw or undercooked foods. He had all age-appropriate immunizations.

On physical exam, the temperature was 38.2˚C, the heart rate was 98 beats per minute, the respiratory rate was 18 breaths per minute, and the blood pressure was 110/57 mm Hg. He was ill-appearing and had nuchal rigidity, though had normal mental status and a non-focal neurologic exam. During his ED course, he developed a diffuse erythematous maculopapular rash on the left upper extremity and left torso, as well as pain with palpation and active movement of the left hand and wrist. His laboratory testing revealed a WBC count of 14.4 x 10^9/L with 9% band forms and a C-

Figure 1. [**A**] Fat-saturated, parasagittal T1-weighted MR image after contrast administration shows non-enhancing subdural fluid between enhancing left frontal dura (blue arrow) and leptomeninges (green arrow). [**B**] Trace diffusion-weighted image in transverse axial plane shows hyperintense signal consistent with purulent fluid in left frontal subdural collection, consistent with subdural empyema. [**C**] Fat-saturated, T2-weighted image shows hyperintense signal of multiple extensor tendon sheaths (red arrows) and interosseous muscles (red stars) of multiple fingers consistent with extensor tendinitis and myositis.



reactive protein (CRP) level of 175.03 mg/L (reference range 0–10 mg/L). Throat PCR for GAS was positive. His respiratory pathogen testing was positive for non-SARS Coronavirus. A blood culture was drawn, and a lumbar puncture was performed, significant for a cerebrospinal fluid (CSF) glucose of 76 mg/dL (reference range 42–91 mg/dL), protein of 151 mg/dL (reference range 13–43 mg/dL), 559 nucleated cells per mm^3 (88% polymorphonuclear cells (reference range 0–7 per mm^3)), and 31 red blood cells (reference range 0–5 per mm^3) in tube 4. Multiplex meningitis/encephalitis PCR from the CSF was negative, and gram stain showed gram-positive cocci. Ceftriaxone (CTX) and vancomycin were initiated. MRI of the brain with contrast on Hospital Day (HD) 1 revealed circumferential dural enhancement with left cerebral convexity subdural empyema without ventriculitis [**Figure 1A,B**]. CSF and blood cultures both were positive for GAS, and antibiotics were narrowed to CTX monotherapy.

Throughout the first week of hospitalization, the patient initially showed improvement; inflammatory markers downtrended with resolution of fever. On HD 5, left upper extremity pain worsened, and he developed left hand weakness. On HD 7, a contrast-enhanced MRI of the left hand was consistent with extensor tenosynovitis and myositis without abscess formation [**Figure 1C**], and the pain dissipated over time. On HD 8, the patient developed acute worsening of headache and vomiting, agitation, confusion, and global aphasia, and he was transferred to the pediatric intensive care unit (PICU). Methylprednisolone and rifampin were briefly added due to concern for encephalitis and incomplete penetration of the empyema, respectively. On HD 10, he developed generalized seizures and was started on levetiracetam. A repeat MRI/MRV on HD 11 showed new left lateral temporal lobe diffusion restriction concerning for stroke due to vasospasm vs vasculitis. He received two 1g/kg doses of intravenous (IV) immune globulin for possible auto-immune vasculitis and encephalitis, though high-resolution MRI of the vessel walls was normal. He was also started on verapamil for possible vasospasm and amitriptyline for migraine headaches.

The patient did not require surgical intervention and completed six weeks of CTX. Follow-up MRI at six weeks of therapy showed residual diffuse pachymeningeal enhancement with resolution of the subdural empyema and temporal reduced-diffusivity. His left upper extremity tenosynovitis and aphasia have resolved. At the time of this writing, he continues on levetiracetam and amitriptyline, is weaning from verapamil, and continues to follow with Neurology for management of breakthrough seizures and headaches.

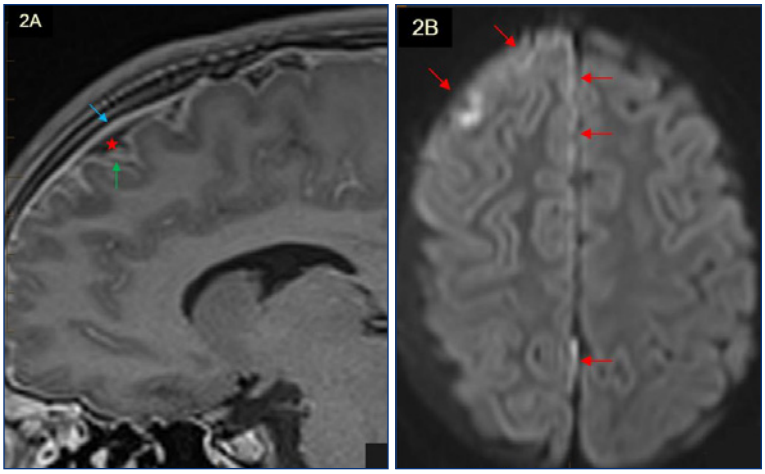
Case 2

A 9-year-old female with no significant past medical history presented to an urgent care center with one week of fatigue, malaise, rhinorrhea, post-tussive emesis, progressive frontotemporal headache and one day of sudden-onset disorientation, difficulty ambulating, and left-sided weakness. There were known sick contacts at home and at school. She had no recent travel, animal bites or scratches, or consumption of raw or undercooked foods. She had all age-appropriate immunizations except for SARS-CoV-2 and influenza.

On physical exam, the temperature was 36.4°C, the heart rate was 110 beats per minute, the respiratory rate was 20 breaths per minute, and the blood pressure was 60/30 mm Hg. She was lethargic with left-sided weakness, left ptosis, right-sided gaze preference, impaired left-hand grip, and left lower extremity weakness.

Upon arrival at our facility, the patient was admitted to the PICU and required an epinephrine infusion. Her laboratory

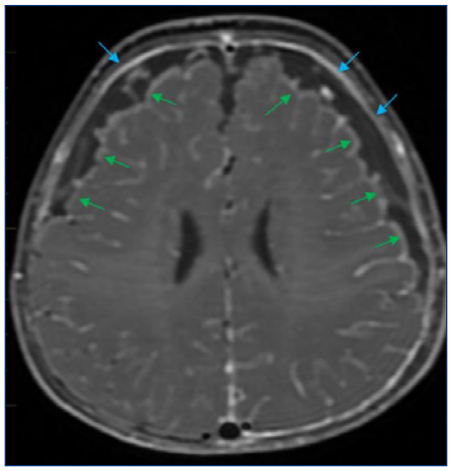
Figure 2. [**A**] Post-contrast T1-weighted parasagittal image through right frontal lobe shows non-enhancing hypointense fluid (red star) between enhancing dura (blue arrow) and leptomeninges (green arrow) over surface of frontal lobe. [**B**] Trace diffusion-weighted MR imaging in transverse axial plane shows hyperintense signal (red arrows) over right frontal convexity and along interhemispheric falx consistent with purulent fluid.



testing revealed a WBC count of 12.5 x 10^9/L with 5% band forms, hemoglobin of 10.6 g/dL, platelet count of 86 x 10^9/L, as well as hyponatremia and hypokalemia to 134 mEq/L and 3.2 mEq/L, respectively. Throat PCR and CRP were not obtained. Respiratory pathogen testing was negative. CT of the head with contrast revealed a right subdural fluid collection, and opacification of the bilateral frontal, left ethmoid, and left sphenoid sinuses. A contrast-enhanced MRI of the brain revealed a right cerebral convexity subdural empyema with leptomeningitis [**Figure 2A,B**]. The patient underwent decompressive right craniectomy with subdural empyema evacuation and endoscopic sinus debridement. She was started on vancomycin, ceftriaxone, and metronidazole. Cultures of the blood and subdural fluid collection grew GAS. Antibiotics were subsequently narrowed to CTX monotherapy. On HD 5, the patient experienced status epilepticus with left-sided tonic-clonic movements and received levetiracetam and lacosamide. A surveillance MRI of the brain after four weeks of therapy showed a possible new abscess vs. post-surgical hemorrhagic changes in the right lateral superior parietal lobe, which prompted the decision to extend her antibiotic course.

Follow-up MRI after six weeks of therapy showed an interval decrease in the size of the lesion attributed to cerebral abscess. The patient completed nine total weeks of therapy for GAS; CTX was stopped at eight weeks and replaced with levofloxacin due to drug-induced liver injury and choledocholithiasis requiring cholecystectomy. At the time of this writing, the patient continues on anti-epileptics with Neurology follow-up and continues to work with Physical Therapy for residual left-sided weakness.

Figure 3. Post-contrast T1-weighted image in transverse axial plane shows abnormal enhancement of both dura (blue arrows) and leptomeninges (green arrows) over surface of the frontal lobes. Diffusion-weighted imaging (not shown) did not demonstrate reduced water-diffusivity.



Case 3

A 3-month-old female with no prenatal or postnatal history was sent to the ED by her pediatrician for "bacteria in the blood." She had a two-day history of fever, increased sleepiness, decreased urine output, and one day of emesis. She had no sick contacts at home. She had no recent travel, animal bites or scratches, or exposure to unpasteurized dairy products. She had received all age-appropriate immunizations.

On physical exam, the temperature was 40.4˚C, maximum heart rate was 218 beats per minute, respiratory rate was 43 breaths per minute, and blood pressure was 96/69 mm Hg. Her exam was notable for an ill-appearing, sleepy infant with a full anterior fontanelle. She had upward gaze deviation and was not tracking. She was minimally responsive and did not cry with IV line placement or urinary catheterization. Review of the laboratory testing obtained by her pediatrician showed a gram-stain of blood positive for gram positive cocci in chains. Her laboratory testing in the emergency room revealed WBC 20.6 x 10^9/L with 5% band forms, CRP 177.01 mg/L, and platelet count of 462 x 10^9/L. Respiratory pathogen testing was positive for Adenovirus. Urinalysis was negative for leukocytes, nitrites, and WBCs. A second blood culture was obtained, and lumbar puncture was performed. CSF studies revealed pink, cloudy fluid with a glucose of less than 5 mg/dL (reference range: 42–91 mg/dL), protein 528 mg/dL (reference range: 13–43 mg/dL), 891 nucleated cells per mm^3, and 12,000 red blood cells per mm^3. A multiplex meningitis/encephalitis PCR was negative, and CSF gram stain showed gram positive cocci. CT head was negative for acute intracranial pathology. Ceftriaxone and vancomycin were initiated, and she was admitted to the PICU.

The first blood culture drawn by her pediatrician and CSF culture grew GAS, and antibiotics were narrowed to ampicillin monotherapy. On HD 3, an MRI brain scan with and

without contrast was obtained, which showed diffuse leptomeningeal and pachymeningeal enhancement consistent with meningitis, bilateral subdural effusions, and no focal cerebritis [**Figure 3**]. The clinical course was complicated by episodes of abnormal movements with apnea on HD 11, with seizure activity noted in the left temporal lobe on electroencephalogram (EEG). She was started on levetiracetam without further seizure or apneic episodes.

The patient ultimately completed a four-week course of IV CTX. Surveillance MRI obtained prior to discharge showed an interval decrease in both bifrontal and left parafalcine effusions and bifrontal leptomeningeal enhancement. The hearing screen was normal. Physical exam throughout admission showed low axial tone with head lag. She was discharged home on levetiracetam with outpatient Neurology follow-up and a referral for Early Intervention services for motor delays.

At the time of this writing, the patient continues to follow with Neurology and at her most recent appointment at 6 months old, she was able to roll over, hold head up and sit with support, but was not yet sitting independently or reaching for objects. Repeat EEG three months after discharge was normal and discontinuation of levetiracetam is being considered.

DISCUSSION

The timing and geographic proximity of our patients are notable. All three presented within the same two-month period in early 2025, and their homes in Massachusetts are located within 20 miles of each other, though there was no known epidemiological link among these three cases. All three presented as ill-appearing and required intensive care during their hospital course. They were all found to have CNS involvement with meningitis and subdural collections seen on brain imaging and required at least four weeks of IV antibiotics. Of the three, one patient underwent surgical intervention (**Case 2**), and one patient had symptoms and imaging suggestive of ischemic stroke (**Case 1**). All three had seizures requiring anti-epileptic agents. At the time of this writing, all are continuing on long-term anti-epileptic therapy. All three patients showed significant improvement although continue with persistent symptoms including headaches and breakthrough seizures (**Case 1**), residual left-sided weakness (**Case 2**), and persistent gross motor delay (**Case 3**).

The global incidence and severity of GAS infections have increased in the post-pandemic years. Although epidemiologic data on iGAS have been published recently in Europe,[10,15] Australia,[11] and Canada,[4] pediatric data are lacking from the New England region in the U.S. The Massachusetts Department of Public Health has reported 148 cases of adult and pediatric iGAS in the first half of 2025, with the highest number of cases in long-term care facilities, but not stratified by age group.[12] Similarly, the Rhode Island Department of Health has shown an increase in the incidence of iGAS cases in the 0–54-year-old population (1.87 to 5.75 per 100,000 persons from 2021 to 2024).[14]

Historically, GAS is a rare etiology of bacterial meningitis, representing 2.2% of cases in 2001–2004.[16] In their 2023 review of case reports from 2001–2020, Hutton and colleagues found 115 cases of intracranial iGAS, with 92% showing meningeal involvement, evidence of intraparenchymal abscess in 8.7%, and a case fatality rate of 10.4%.[17] Of 1272 children with iGAS in 10 U.S. states from 2013 to 2022, 3.5% had meningitis.[18] Recent data looking only at post-pandemic cases reveal that intracranial iGAS remains rare. Of 154 iGAS cases in Southeast Texas from June 2022 to May 2023, 5.8% had CNS involvement.[19] Another study from Italy documented two cases of GAS meningitis out of 34 cases of iGAS from April 2023 to July 2024,[20] and in Ireland, two out of 167 iGAS pediatric cases from 2022 to 2023 had CNS involvement.[9] A recent Dutch cohort study from August 2025 showed increased incidence of meningitis/encephalitis due to GAS after the COVID-19 pandemic, with an incidence ratio of 12.30 in January 2022 to May 2024 compared to January 2015 to March 2020.[21]

Concurrent viral respiratory infections have been seen in 28–52% of patients with iGAS.[1,4,8,22] Some literature demonstrates an association between influenza and iGAS diseases such as toxic shock syndrome, bacteremia, and pleural empyema.[23-25] Of our three patients, two had respiratory viruses identified (non-SARS Coronavirus and Adenovirus).

There are no guidelines specific to the management of iGAS with CNS involvement. GAS is universally susceptible to penicillin with a typical duration of four weeks for uncomplicated disease. However, an extended course or alternative antibiotic choice may be made to broaden coverage, such as in the case of subdural empyema, as seen in our cases. GAS resistance to certain antibiotics has increased over time in the U.S. (27.4%, 26.4%, and 32.1% isolates were resistant to macrolides, clindamycin, and tetracyclines, respectively, in 2023).[26,27]

Given the recent literature and cases presented above, it may be prudent to consider GAS as a culprit when approaching patients presenting with intracranial bacterial infections. Furthermore, because intracranial iGAS infections commonly require intensive care and have a high case fatality rate,[22] prompt identification of clinical symptoms is necessary. Providers should also be aware that GAS is not a target included on many commercially available multiplex CSF PCR assays for meningitis/encephalitis. Dedicated surveillance of pediatric iGAS cases and their associated clinical outcomes will be an important step towards a better understanding of the regional burden of this pathology in the pediatric population.

CONCLUSION

Rates of GAS and iGAS infections have been rising globally in recent years since the COVID-19 pandemic. iGAS infections are often severe and may involve the central nervous system, leading to significant long-term sequelae. Providers evaluating patients with signs of severe invasive bacterial infection should be mindful of GAS as a culprit of increasing prevalence.

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