

Case Report

Adult-onset Still's disease presenting with generalized seizures in the absence of macrophage activation syndrome: a case report with review of the neurological literature

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ABSTRACT

Adult-onset Still's disease (AOSD) is a rare systemic autoinflammatory disorder characterized by quotidian fever, arthritis, rash, and marked hyperferritinemia. Neurological manifestations are uncommon, and generalized seizures are exceptionally rare, particularly in the absence of macrophage activation syndrome (MAS). An 18-year-old man presented with one month of high-grade fever, migratory polyarthralgia, cervical and axillary lymphadenopathy, mild hepatosplenomegaly, and two generalized tonic-clonic seizures preceding diagnosis. Laboratory evaluation showed neutrophilic leukocytosis (21,300/mm³), elevated ESR and CRP, transaminitis, and serum ferritin >2,000 ng/mL. Electrolytes were normal. Blood and urine cultures, HIV testing, tuberculosis evaluation, echocardiography, thoracic CT, and lymph-node cytology were unremarkable. NCCT brain showed no structural lesion, and there was no clinical or laboratory evidence of MAS. After exclusion of infectious, autoimmune, and malignant causes, the patient fulfilled Yamaguchi criteria for AOSD. Treatment with prednisolone (1 mg/kg/day), methotrexate, and levetiracetam resulted in rapid resolution of fever and improvement in arthralgia. Generalized seizures may rarely be the presenting manifestation of AOSD even without MAS or demonstrable CNS pathology. In patients with unexplained seizures accompanied by persistent systemic inflammation and marked hyperferritinemia, AOSD should be considered after exclusion of infectious, autoimmune, and structural neurological causes; early immunosuppressive therapy is associated with favorable outcomes.

Keywords: Adult onset Still's disease, Seizures, Hyperferritinemia, Yamaguchi criteria, Neurological review

INTRODUCTION

Adult-onset Still's disease (AOSD) is a rare, systemic autoinflammatory disorder first described in adults by Bywaters in 1971. AOSD affects approximately 0.16-0.4 per 100,000 individuals annually. It can occur at any age but is most common in young adults aged 16-35 years. There is no clear sex predominance. The precise etiology remains unknown, but environmental triggers such as viral infections (e.g., Epstein-Barr virus, cytomegalovirus) may initiate the disease in genetically predisposed individuals. AOSD is now categorized under autoinflammatory

diseases, reflecting its innate immune system involvement rather than autoimmunity.¹ AOSD is driven by an exaggerated innate immune response. Cytokines such as interleukin (IL)-1 β , IL-6, IL-18, and tumor necrosis factor- α (TNF- α) are significantly elevated and correlate with disease activity. Neutrophil and macrophage activation play key roles in tissue damage.^{2,3} The disease has three main manifestations and may present as systemic, articular or mixed form. Systemic form clinically presents with high-grade spiking fever (usually $\geq 39^{\circ}\text{C}$), sore throat, evanescent non-pruritic rash, and lymphadenopathy. Articular form manifests as polyarthritides, particularly

affecting wrists, knees, and ankles. Mixed form presents with features of both systemic inflammation and chronic arthritis. Other findings may include hepatosplenomegaly, pleuritis, pericarditis, and elevated liver enzymes.⁴

Neurological manifestations occur in only a minority of patients and most often involve aseptic meningitis or encephalitis. Published seizure cases frequently occur with severe systemic disease, MAS, structural CNS abnormalities, or other complications. This patient presented with seizures despite no evidence of MAS, no documented CNS infection, and no obvious structural brain lesion, making the presentation diagnostically challenging.

CASE REPORT

An 18-year-old male, with no known comorbidity, resident of Prayagraj, and a student of B. Pharmacy presented on 19 February 2025 in our tertiary care centre SRN Hospital attached to Moti Lal Nehru Medical College Prayagraj, with complain of multiple joint pains for two months. Joint pain was associated with early morning stiffness for about 30 mins which resolved spontaneously with activity. Joint pain was progressive and migratory, initially involving right wrist followed by left wrist and later right ankle. Joint pain was non additive and resolved with no residual signs of inflammation. Patient also complained of fever for 1 month. Fever was high grade and associated with chills and rigor. It used to subside for 5-6 hours after medication and returned soon after the effect of antipyretics wore off.

Patient had 2 episodes of GTCS seizures a day before for which she got admitted in emergency medicine unit.

There was no h/o conjunctivitis, rashes, illicit behaviour, IV drug abuse, night sweats, weight loss, cough, abnormal dancing movements. Patient had no h/o oral ulcers, malar rash, photosensitivity, any skin rash. There was no h/o headache, blurred vision, double vision. No h/o epistaxis, sinusitis, neuropathy, frothy urine, edema, oliguria. No h/o anemia, blood transfusion.

On general examination, multiple sub-centimetric lymph nodes were palpated in cervical region and axilla which were non tender and firm. No pallor, icterus, cyanosis, clubbing, edema were noted.

On systemic examination neurologically no signs of meningeal irritation were present. Neck rigidity was absent. Plantars were bilateral withdrawal. Deep tendon jerks were brisk and no sensory loss was noted.

Cardiovascular and respiratory systems were normal on examination.

On per abdomen examination- spleen was palpable 1 cm under left costal margin. Liver was just palpable. Bowel sounds were +(6/min). On investigations reports showed leucocytosis, reactive CRP, Raised ESR, raised transaminases, hyperferritinemia and ANA 1+ve nucleolar and fine speckled at 1:100 and splenomegaly as shown in Table 1.

Table 1: Investigation of the case.

Lab parameters	Patient value	Reference range
Hemoglobin (g/dl)	11	12-17
Total leucocyte count (cells/mm ³)	21300	4000-11000
DLC: Granulocytes/lymphocytes (%)	88.2/7.8	40-80/20-40
Platelet count (lac cells/mm ³)	3.34	1.5-4
ESR (mm/1 hour)	70	<10
CRP	Reactive	Non-reactive
S. Sodium/ potassium/ ionized calcium (mmol/l)	135.6/4.97/1.20	135-150/3.5-5.3/1.13-1.32
SGOT/SGPT/ALP (U/l)	64/104/544	<40/<45/<270
S. Protein/albumin/globulin (g/dl)	7.1/3.5/3.6	6.4-8.3/3.5-5.2/2-3.5
S. urea/creatinine (mg/dl)	28/0.88	13-43/0.8-1.3
RA factor	Non-reactive	Non-reactive
ASO titer	Non-reactive	Non-reactive
S. Bilirubin (Total/ direct/ indirect) (mg/dl)	0.31/0.15/0.16	<1.2/<0.2
S. uric acid (ng/dl)	5.5	3.5-7.2
Urine R/M	Pus cells- neg/hpf, RBC- 2-3/hpf Proteins, Glucose-neg	
USG W/A	Splenomegaly (12.2 cm)	
2 D- Echo	Normal study	
Urine C/S	No growth	
Blood C/S	No growth	
S. Ferritin (ng/ml)	>2000	21.8-274.6
S. Fasting lipid profile. S. triglyceride/ cholesterol/ HDL cholesterol / LDL cholesterol (mg/dl)	129/156/33/97	<150/<200/>60/<150

Continued.

Lab parameters	Patient value	Reference range
General blood picture	Normocytic anemia with neutrophilic leucocytosis	
NCCT head	Normal study	
ANA	1+ve nucleolar and fine speckled at 1:100	Negative
C3 (mg/dl)	147	88-180
C4 (mg/dl)	44	16-48
HIV	Non-reactive	Non-reactive
Plasma fibrinogen (mg/dl)	367	200-400
CT thorax	WNL	
FNAC of Rt. axillary lymph node	WNL	

Differential diagnosis

Patient was evaluated for seizures and SLE was ruled out using EULAR criteria 2019. Montoux test was negative and possibility of tuberculosis was dismissed. The patient did not satisfy the criteria for rheumatoid arthritis. The patient continued to have spikes of fever during hospital admission but blood and urine culture were negative, ruling out other infectious etiology like brucellosis and leptospirosis. 2D Echo was within normal limits, ruling out the possibility of infective endocarditis. Blood reports with ferritin >2000, neutrophilic leucocytosis, negative ANA and rheumatoid factor along with h/o fever, arthralgia, lymphadenopathy and splenomegaly satisfied the Yamaguchi criteria.⁵

Patient was diagnosed as AOSD of mixed type, with no features of MAS and was started on prednisolone 1 mg/kg/day and showed symptomatic improvement, with resolution of fever and improvement in arthralgia in 7 days. Although seizures are present in approximately 7-12% of patients with AOSD, it usually occurs in association with MAS secondary to AOSD. Our case is atypical as seizure was a presenting manifestation even without MAS.⁶ Data regarding incidence of seizures in patients with mixed variety of AOSD is lacking in medical literature.

Treatment

Patient was started on tab wysolone 20 mg BD (1 mg/kg/day) with tab. methotrexate 15 mg once weekly and tab folvite 5 mg once weekly. He was started tab. levetiracetam 500 mg BD at the time of seizures which was continued at the time of discharge. Tab wysolone was slowly tapered over a course of 8 weeks. The patient started having spikes of fever following its discontinuation, hence it was started again and methotrexate dose was increased to 25 mg once weekly with folvite being given as 5 mg twice weekly. Disease activity was not controlled on methotrexate and initiation of tab tacrolimus 0.5 mg BD was planned. Due to patient's history of seizures along with possibility of difficulty in monitoring, the patient was started on tab. Upadacitinib 15 mg OD. The patient was lost to follow up.

Initial therapy of AOSD begins with non-steroidal anti-inflammatory drugs (NSAIDs), often ibuprofen or naproxen, or high-dose aspirin in adults, to control fever and mild symptoms.⁷ When disease activity is moderate to severe, corticosteroids such as prednisone (0.5-1 mg/kg/day) or IV methylprednisolone are key to achieving remission. Methotrexate (7.5-20 mg weekly) is commonly used as a steroid-sparing DMARD, helping reduce glucocorticoid exposure in up to 70% of patients.⁸ For patients refractory to steroids or methotrexate, biologic DMARDs have become integral to treatment. Anakinra, an IL1 receptor antagonist, induces complete remission in around 35% of cases and partial response in 41%, significantly lowering steroid dependence.⁹ Canakinumab, a long-acting anti-IL1 β antibody, is FDA-approved for AOSD and consistently demonstrates rapid resolution of symptoms, normalization of ferritin and CRP, and steroid tapering in refractory cohorts.¹⁰⁻¹² Tocilizumab, an IL6 receptor blocker, is effective particularly in chronic articular phenotypes, achieving remission and corticosteroid withdrawal.^{13,14} Meta-analyses show complete remission rates of ~77% for canakinumab, ~73% for anakinra, and ~80% for tocilizumab, with steroid discontinuation in over half of patients.¹⁵ In resistant or life-threatening presentations, TNF inhibitors (e.g., etanercept, infliximab) and cyclosporine A may be used, especially in MAS, though evidence relies largely on case reports.^{16,17} Emerging agents targeting IL-18, IL-17, and JAK pathways are under investigation and may expand future options.¹⁸

DISCUSSION

AOSD shares clinical and pathological features with systemic juvenile idiopathic arthritis (sJIA) seen in children with age at onset less than 16 years.¹

The age of our case was 18 years and young age presentation is extremely rare and the usual age of onset of AOSD is 4th decade. There is no specific diagnostic test for AOSD. The Yamaguchi criteria (1992) is most widely used and includes a combination of major and minor clinical and laboratory features as shown in Table 2. At least five criteria, including two major ones, are needed for diagnosis, after excluding infections, malignancies and autoimmune conditions.⁴

Table 2: Yamaguchi criteria.

Major criteria	Minor criteria
Fever $\geq 39^{\circ}\text{C}$ for ≥ 1 week	Sore throat
Arthralgia/arthritis ≥ 2 weeks	Lymphadenopathy
Evanescant salmon-pink rash	Abnormal liver function tests
Leukocytosis ($>10,000/\text{mm}^3$) with $\geq 80\%$ neutrophils	Negative ANA and RF
	Hepatomegaly or Splenomegaly

The hallmark laboratory finding is marked hyperferritinemia, which is considered a surrogate marker of disease severity and macrophage activation.²

Most patients with AOSD have a monocyclic or polycyclic course with a favourable prognosis. Chronic articular disease may lead to joint damage. Early treatment improves outcome. The most severe complication is MAS, a potentially fatal hyperinflammatory state characterized by cytopenias, liver dysfunction, and coagulopathy. Other complications include chronic erosive arthritis and pulmonary involvement.⁵

Here in this case ANA is reported as: 1+ nucleolar and fine speckled (1:100) fulfilling Classical Yamaguchi criteria expect ANA negativity. Low-titer ANA positivity has occasionally been described in 2.9%-25.8% of patients of AOSD.¹⁹

Neurological manifestations of AOSD are uncommon, occurring in approximately 7-12% of patients.^{20,27,28} Seizures are among the rarest neurological presentations and have been described almost exclusively in isolated case reports, making their true incidence difficult to determine.²¹⁻²⁴ Most reported neurological manifestations in AOSD are:^{20,27,28} Secondary to MAS, encephalitis, aseptic meningitis, cerebral vasculitis, or metabolic disturbances. Seizures-cranial nerve palsy, hearing loss, cerebral infarction, confusion, cranial neuropathy, peripheral neuropathy and Miller Fisher syndrome.

Our patient had-Younger age onset, two seizure episodes, normal NCCT brain, no electrolyte abnormality, no evidence of infection, no evidence of MAS and seizures preceding diagnosis of AOSD.

Only one recent report in which seizures occurred with MAS and myocarditis is seen and our patient differs because seizures occurred without MAS, making it clinically interesting.^{22,29}

In a cohort study among 187 AOSD patients 14 (7.5%) had neurological involvement, the most common manifestation was aseptic meningitis while other manifestations included encephalitis, cranial nerve palsy, and cerebral infarction. Neurological involvement was

frequently associated with MAS and higher ferritin levels.²⁰

From a very old article the neurological entities occurrence has been projected in Table 3.²⁸

Table 3: Occurrence of neurological manifestation in AOSD neurological manifestation.

Manifestation	Frequency
Aseptic meningitis	Most common
Encephalitis	Rare
Confusion/delirium	Rare
Psychiatric symptoms	Rare
Generalized seizures	Very rare
Status epilepticus	Extremely rare
Cranial nerve palsy	Rare
Sensorineural hearing loss	Rare
Cerebral infarction	Rare
Guillain-Barré syndrome/ Miller Fisher syndrome	Very rare
Cerebral microhemorrhages	Exceptional

The exact mechanism underlying seizures in AOSD remains poorly understood. Current evidence suggests that excessive systemic inflammation, driven by cytokines such as interleukin (IL)-1 β , IL-6, IL-18, and tumor necrosis factor- α , results in disruption of the blood-brain barrier and neuroinflammation, thereby lowering the seizure threshold.^{20,24,27} Hyperferritinemia, a hallmark of active AOSD, has also been associated with severe inflammatory disease and neurological involvement.^{20,24}

Our systematic review identified only a limited number of published cases of AOSD presenting with seizures between 1990 and 2026, highlighting the exceptional rarity of this manifestation.²¹⁻²³ Although patients varied widely in age and sex, the majority developed seizures during periods of active systemic inflammation, while a few presented with seizures as the initial manifestation before fulfilling the diagnostic criteria for AOSD.^{21,22}

Generalized tonic-clonic seizures were the most frequently reported seizure type, although focal seizures, recurrent seizures, and altered consciousness associated with meningoencephalitis have also been described.^{21,22,26} Neurological involvement frequently coincided with severe systemic disease, including MAS, suggesting that overwhelming cytokine activation may play a central role.^{22,25,26}

Neuroimaging findings were heterogeneous. While several patients had normal magnetic resonance imaging (MRI), others demonstrated reversible cortical abnormalities, cerebral edema, diffuse white matter lesions, or cerebral microhemorrhages.^{22-24,26} Kim et al recently reported cerebral microhemorrhages in a patient with AOSD, expanding the spectrum of radiological manifestations associated with severe systemic inflammation.²³ Similarly,

cerebrospinal fluid (CSF) findings ranged from normal to mild lymphocytic pleocytosis with elevated protein levels, usually in the absence of infectious organisms, supporting an inflammatory rather than infectious process.^{24,26,27}

Because seizures are an uncommon manifestation of AOSD, diagnosis requires meticulous exclusion of alternative etiologies, including viral or bacterial encephalitis, autoimmune encephalitis, metabolic abnormalities, cerebrovascular disease, drug-induced seizures, and primary epilepsy.^{21,24,27} Negative infectious investigations together with characteristic systemic features—including quotidian fever, arthritis, evanescent rash, marked hyperferritinemia, neutrophilic leukocytosis, and elevated inflammatory markers—strengthen the diagnosis of AOSD.^{20,24,27}

The closest publication is the BMJ case report by Young et al which reviewed the seizure cases available up to 2020 and found only five previously published cases of seizures associated with AOSD.³⁰

Treatment across published cases consistently emphasized rapid suppression of systemic inflammation. High-dose intravenous corticosteroids constituted first-line therapy in nearly all reported patients, while refractory cases required additional immunosuppressive agents or biologic therapies targeting IL-1 or IL-6 pathways.^{20,22,24–27} Antiepileptic drugs were generally administered for acute seizure control but were not required long term once systemic inflammation was adequately controlled.^{21,22}

Clinical outcomes were generally favorable when immunosuppressive therapy was initiated promptly.^{21–24} However, delayed diagnosis or concomitant MAS was associated with increased morbidity, prolonged hospitalization, and greater neurological complications.^{22,25,26} These observations underscore the importance of recognizing seizures as a possible manifestation of severe AOSD rather than attributing them solely to primary neurological disorders.

The present review reinforces several clinically important observations. First, seizures may rarely represent the initial presentation of AOSD before the classical triad of fever, arthritis, and rash becomes fully apparent.^{21,22} Second, unexplained seizures accompanied by markedly elevated ferritin and persistent systemic inflammation should prompt consideration of AOSD after exclusion of infectious and autoimmune causes.^{20,24,27} Finally, successful neurological recovery depends primarily on early recognition and aggressive control of systemic inflammation rather than prolonged antiepileptic therapy.^{20–22,24}

The principal limitation of this review of neurological aspect of AOSD is the reliance on individual case reports like this one and small case series, resulting in heterogeneity of clinical presentation, diagnostic evaluation, treatment, and outcome reporting. Publication

bias is also likely, as unusual or severe neurological presentations are more likely to be reported than uncomplicated cases.^{20–28}

CONCLUSION

AOSD remains a diagnosis of exclusion, with management tailored to symptoms and severity. Long-term outcomes are favourable, though vigilant monitoring is crucial to prevent complications such as MAS. Individual “treat-to-target” strategies and biomarker-guided therapy will further optimize care.

Overall, seizures represent an exceptionally rare but potentially life-threatening neurological manifestation of AOSD. Increased awareness among physicians may facilitate earlier diagnosis, prompt initiation of immunosuppressive therapy, and improved neurological outcomes. In our review of all reported cases, seizures occurred in middle-aged adults with severe systemic inflammation. With exclusion of other etiologies and early immunosuppressive therapy, outcomes were excellent. Physicians should include AOSD in the differential for adult-onset febrile seizures, and vice versa consider neuroimaging and lumbar puncture in AOSD with neurologic signs.

Our patient presented with seizures despite no evidence of MAS, no documented CNS infection, and no obvious structural brain lesion, making the presentation diagnostically challenging.

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