DOI: 10.7860/JCDR/2026/87287.23482

Case Report

Internal Medicine Section

Adult-onset Still's Disease Masquerading as Pyrexia of Unknown Origin with Severe Hyponatremia and Pleural Effusion: A Case Report

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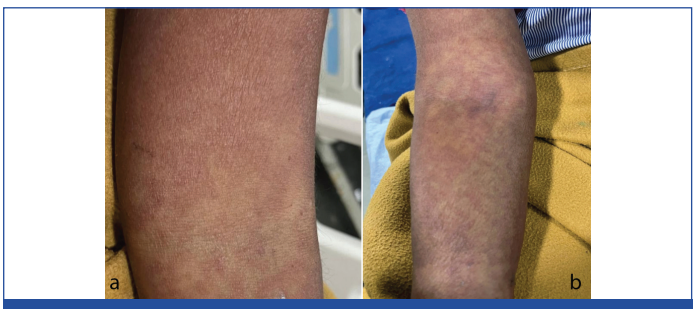
ABSTRACT

Adult-onset Still's Disease (AOSD) is a rare autoinflammatory disorder marked by quotidian fever, evanescent rash, and inflammatory arthritis, often requiring exclusion of infectious and autoimmune causes before diagnosis. Hereby, the authors present a case report of 45-year-old female with a two-and-a-half–month history of daily high-grade fever, salmon-coloured maculopapular rash exacerbated during fever spikes, severe sore throat, and progressive polyarthralgia. Her illness later progressed to bilateral flank pain, vomiting, dysapnoea, and hypotension. Laboratory evaluation revealed marked leukocytosis (27,000/cumm), anaemia, hyponatremia, elevated Serum Glutamic Oxaloacetic Transaminase (SGOT), hypertriglyceridemia, and significantly raised inflammatory markers, including C-reactive Protein (CRP) (162 mg/dL), Lactate Dehydrogenase (LDH) (1612 U/L), and ferritin (1200 ng/mL). Infectious and autoimmune studies were negative. High Resolution Computed Tomography (HRCT) of the thorax showed bilateral pleural effusions and ground-glass opacities, while Contrast-enhanced Computed Tomography (CECT) abdomen demonstrated hepatosplenomegaly and ascites. Despite broad-spectrum intravenous antibiotics, fever and systemic symptoms persisted. Given the prolonged fever, typical rash, sore throat, neutrophilic leukocytosis, abnormal liver function tests, and negative Anti-nuclear Antibodies (ANA) and Rheumatoid Factor (RF), a diagnosis of AOSD was strongly supported, fulfilling Yamaguchi criteria with three major and multiple minor criteria. The patient was treated with indomethacin and intravenous methylprednisolone 1 mg/kg for five days, later transitioned to tapering oral steroids. Within 48 hours, she demonstrated marked clinical improvement with resolution of fever and rapid decline in ferritin, CRP, and White Blood Cells (WBC) count. She was discharged after stabilisation and remained asymptomatic at her 15-day follow-up while continuing a tapering steroid regimen. The present case highlights the importance of early consideration of AOSD in patients with prolonged fever and multisystem involvement, particularly when infections and autoimmune diseases have been excluded. Rapid response to corticosteroids further supports the diagnosis and underscores their central role in initial management.

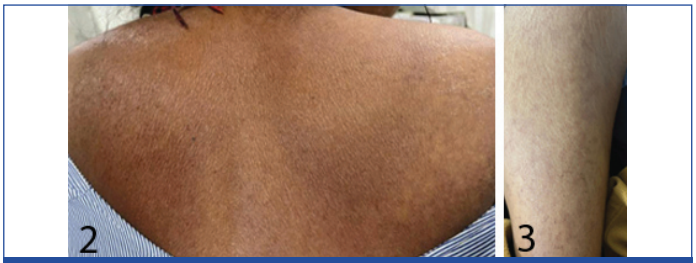
Keywords: Hepatosplenomegaly, Hypertriglyceridemia, Yamaguchi criteria

CASE REPORT

A 45-year-old female presented with a history of daily high-grade fever for the past two and a half months, accompanied by a maculopapular rash over the trunk and limbs that became more prominent during fever spikes, along with sore throat [Table/Fig-1a,b-3]. During the past two months, she gradually developed bilateral knee and elbow joint pain, which was followed by progressive difficulty in rising from a squatting position and lifting her arms overhead. Around 10 days prior to admission, she began experiencing bilateral flank abdominal pain, and one week before presentation she developed vomiting with 1-2 episodes per day. There was no history of recent drug intake known to precipitate drug-induced Still's disease, and no family history of autoimmune or auto-inflammatory disorders. There was no documented weight loss. Symptomatic treatment received at local healthcare facilities did not result in improvement.



[Table/Fig-1]: Both hands showing salmon pink maculopapular rash.



[Table/Fig-2]: Upper back showing salmon pink maculopapular rash. **[Table/Fig-3]:** Thigh showing salmon pink maculopapular rash. (Images from left to right)

On admission, the examination suggested the patient was conscious and oriented. Vital signs revealed tachycardia (112 beats/min), hypotension (90/60 mmHg), tachypnea (20/min), and oxygen saturation of 88% on room air. Respiratory examination revealed bilateral basal crepitations. Cardiovascular examination was unremarkable, with no murmurs or pericardial rub. Abdominal examination revealed tenderness in both hypochondria without guarding or rigidity, along with clinical hepatosplenomegaly. Musculoskeletal examination demonstrated tenderness and restricted movement of bilateral knee and elbow joints, causing moderate functional limitation. No peripheral lymphadenopathy was detected. The patient had an evanescent, non pruritic, salmon-pink maculopapular rash over the trunk and limbs, coinciding with fever peaks and did not accentuated by heat or friction (Koebner phenomenon).

Initial laboratory evaluation showed marked leukocytosis (WBC 21,000/cumm) with neutrophilia, haemoglobin 9.4 g/dL, platelet count 1.5 lac/cumm, and hyponatremia (121 mEq/L) [Table/Fig-4]. Peripheral smear revealed microcytic hypochromic anaemia with neutrophilic leukocytosis. Inflammatory markers were markedly elevated, including CRP 162 mg/dL, LDH 1612 U/L, Erythrocyte Sedimentation Rate (ESR)-90 and serum ferritin 1200 ng/mL. Glycosylated ferritin was not available to support the elevated ferritin levels. Liver function tests showed elevated SGOT (89 U/L) with normal SGPT, bilirubin, and alkaline phosphatase. Lipid profile revealed hypertriglyceridemia (314 mg/dL) with low High-density Lipid (HDL) and Low-density Lipid (LDL). Although hypertriglyceridemia raised concern for MAS, the absence of cytopenias, liver failure, or coagulopathy and normal levels of fibrinogen made Macrophage Activation Syndrome (MAS) unlikely. Serum cortisol and thyroid function tests were normal. Serum procalcitonin was 0.23 ng/dL (<0.05 ng/dL normal). Serum fibrinogen were within normal limits 180 mg/dL (normal range 150-450) Extensive infectious and autoimmune work-up including malaria, dengue, leptospira, brucellosis serology, blood cultures, and ANA profile was negative, effectively excluding common causes of pyrexia of unknown origin.

The patient was initially managed as a case of sepsis with multiorgan involvement, given hypotension, leukocytosis, and hypoxia. The patient was treated with broad-spectrum i.v. antibiotics- meropenem 1 gm tds, teicoplanin 400 mg bd on first day then od, and metronidazole 500 mg tds- along with supportive care and tramadol for joint pain and myalgias. However, persistent fever despite broad-spectrum antibiotics, repeatedly negative cultures, characteristic rash, sore throat, markedly elevated ferritin, hepatosplenomegaly, and inflammatory arthritis prompted consideration of AOSD. She fulfilled the Yamaguchi criteria, with major criteria including fever >2 weeks, typical evanescent rash, and leukocytosis with neutrophilia, along with minor criteria of sore throat, splenomegaly, abnormal liver enzymes, and negative ANA and RF. A diagnosis of AOSD was established after a five-day diagnostic workup that excluded infectious, malignant, and autoimmune aetiologies. Consequently, treatment was initiated on day five of admission with indomethacin at a low dose of 25 mg three times daily, with plans for dose escalation based on patient tolerability. Injectable methylprednisolone 1 mg/kg (60 mg dose, weight -60 kg) for five days, followed by oral steroids at 1 mg/kg in tapering the dose by 5 mg every five days. Along with steroids, patient was started on proton pump inhibitor pantoprazole

Variables (reference range)	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8
Hb (12-16 g/dL)	9.4	8.5	8.6	9	9	9.4	9.5	9.5
CBC (4,000-10,000/cumm)	21000	27000	35000	32000	33000	23500	17000	13000
Platelet count (1.5-4.5 lac/cumm)	1.5	1.5	1.2	1.4	1.42	1.5	1.6	1.8
PT/INR (11-13.5 sec/0.8-1.2)	17/1.2	15/1.09	1.08	1.29	1.25	1.0	1.5	1.2
APTT (25-35 sec)	41	45	52	40	34	40	44	44
Serum creatinine (0.6-1.2 mg/dL)	1	1.1	0.8	0.9	1.2	1.0	1.5	1
Serum urea (15-40 mg/dL)	42	61	40	44	37	24	25	45
Serum bilirubin (0.2-1.2 mg/dL)	0.6 (0.2/0.4)	0.5 (0.2/0.3)	0.4 (0.2/0.2)	0.9 (0.4/0.5)	1 (0.5/0.5)	1 (0.5/0.5)	0.6 (0.4/0.2)	0.6 (0.4/0.2)
SGOT (10-40 IU/L)	89	151	157	161	172	130	88	60
SGPT (7-56 IU/L)	16	27	56	52	54	31	35	36
LDH (140-280 IU/L)	1612	1525		1628		900	700	400
Serum uric acid (2.4-6.0 mg/dL–F)	5.6	6.7						
Serum sodium (136-145 mEq/L)	121	124	125	126	125	129	129	128
Serum potassium (3.5–5.0 mEq/L)	4	4	5	3.4	4	5	4.4	3.9
CRP (<6 mg/L)	162		141		149.6		134	90
Serum iron (60-170 mcg/dL)	57.5							
Serum ferritin (12-150 ng/mL -F)	1200		1500		1780		978	800
TIBC (250-370 mcg/dL)	127							
Serum triglyceride (<150 mg/dL)	314							
S.fibrinogen (200-400 mg/dL)		180		183			170	
ESR (0-30 mm/hour-f)	90					67		
PCT (<0.5 ng/dL)		0.23				0.3		

[Table/Fig-4]: Patient's blood reports during admission.
PT: Prothrombin time; aPTT: Activated partial thromboplastin time; INR: International normalised ratio; CBC: Complete blood count; SGPT: Serum glutamic pyruvic transaminase; TIBC: Total iron

The HRCT thorax demonstrated mild right-sided and moderate left-sided pleural effusion with fissural extension, patchy bilateral ground-glass opacities, and small mediastinal lymph nodes (largest 16×17.5 mm), suggesting inflammatory serositis so pleural tap was not done. Contrast-enhanced Computed Tomography.(CECT) abdomen revealed hepatomegaly, splenomegaly, pericholecystic oedema, and mild to moderate ascites, supporting systemic inflammatory involvement. Pericholecystic oedema was interpreted as part of systemic inflammatory involvement rather than acute cholecystitis, as the patient had no right upper quadrant tenderness, gallstones, or laboratory evidence of biliary infection. Electrocardiogram suggested normal sinus rhythm. Transthoracic echocardiography suggestive of normal ejection fraction with o regional wall motion abnormality and no valualar heart disease with no signs of infective endocarditis.

40 mg od and calcium vitamin d supplementation. Within two days of initiating steroid therapy, the patient demonstrated clear clinical improvement, with cessation of fever spikes and declining trends in WBC count, ferritin and CRP, indicating a favourable therapeutic response to early inflammatory dominant AOSD in absence of chronic arthritis. Serositis-pleural fluid and ascitis reflected high inflammatory burden of AOSD and improved with steroid therapy. Antibiotics were continued briefly after the diagnosis of AOSD to cover a potential occult infection and were discontinued once infection was definitively excluded.

After a one-week stay in the critical care unit, she was shifted to the general ward and subsequently discharged three days later following optimisation of antibiotic therapy and gradual tapering of steroids. On discharge patient was asymptomatic and vitally stable. Patient

was discharge with tablet prednisolone 50 mg od for five days and advised to taper by 5 mg every five days with tablet indomethacin 25 mg tds for 15 days with calcium vitamin supplementation and proton pump inhibitor.

At the 15-day outpatient follow-up, the patient was afebrile, haemodynamically stable and reported marked improvement in joint symptoms with no recurrence of rash. Laboratory parameters showed improving inflammatory markers, and oral corticosteroids were continued in a tapering regimen. At 3-month follow-up, the patient remained clinically stable with no recurrence of fever, rash, or arthritis. Functional status had returned to baseline, and inflammatory markers showed sustained improvement. Corticosteroid dosage was gradually tapered without evidence of disease relapse, and no features suggestive of MAS or other complications were observed. Major complications of AOSD include MAS, chronic destructive arthritis, serositis, hepatic dysfunction, and treatment-related adverse effects were not observed in this period.

DISCUSSION

The AOSD is an uncommon multisystem inflammatory disorder characterised by quotidian high spiking fevers, inflammatory arthritis, and a transient salmon-coloured rash. Although its exact cause remains unclear, AOSD is believed to represent an exaggerated inflammatory response to infectious triggers in genetically predisposed individuals [1]. AOSD may present as monocyclic, polycyclic, or chronic articular patterns, with monocyclic disease characterised by a single self-limited episode, polycyclic by recurrent flares, and chronic articular by persistent arthritis and higher risk of joint damage. The disease is rare with reported AOSD incidence in a range between 0.16 and 0.4/100000 people and estimated prevalence rate between 1 and 34 cases/1 million people with slight female predominance, and exhibits a bimodal age distribution with peaks in early adulthood (15-25 years) and mid-adulthood (36-46 years). Data from India are limited, but available reports suggest that AOSD remains underrecognised and often misdiagnosed due to its nonspecific presentation [2]. The present case report of monocyclic AOSD highlights the diagnostic complexity because of its close clinical mimicry of sepsis, lymphoma and autoimmune disorders and the importance of timely recognition to avoid unnecessary antimicrobial exposure and delayed immunosuppression. Despite fulfilling Yamaguchi criteria, diagnosis was challenging due to initial sepsis-like presentation with hypotension, hypoxia, and multiorgan involvement, necessitating Intensive Care Unit (ICU) admission and empiric broad-spectrum antibiotics.

Clinically, the fever pattern is distinctive, typically occurring once or twice daily, often in the evening, with temperatures returning to near-normal between spikes. The characteristic rash is evanescent, non pruritic, and salmon-pink, commonly appearing on the trunk and limbs during fever peaks and may be elicited by heat or friction (Koebner phenomenon). Joint involvement usually begins as oligoarthritis but may progress to a more persistent and symmetric polyarthritis affecting large and small joints. Additional manifestations include severe sore throat, generalised lymphadenopathy, splenomegaly, and mild hepatic dysfunction [Table/Fig-5]. Serosal involvement, such as pericarditis or pleural effusions, may be seen in up to one-third of patients. A serious but infrequent complication is MAS, suggested by hypertriglyceridemia, hypofibrinogenemia, and cytopenias [3].

Major criteria	Minor criteria	Exclusion criteria
Fever >102° F for >1 week	Sore throat	Infection
Arthralgia for >2 weeks	Lymphadenopathy	Malignancy
Typical rash	Hepatomegaly/splenomegaly	Other rheumatic diseases
WBC >10,000/mL	Abnormal liver function tests	
	Negative ANA and RF	

[Table/Fig-5]: Diagnostic Yamaguchi criteria for adult onset still's disease.

Laboratory abnormalities support the diagnosis, with markedly elevated serum ferritin- often exceeding fivefold the upper limit- being a key feature, along with raised ESR and CRP. Haematologic findings commonly include neutrophilic leukocytosis, anaemia, and thrombocytosis [4]. Diagnosis relies on integrating these clinical and laboratory features using established criteria such as Yamaguchi's. At least five features, with two major and three minor, or three major and two minor criteria should be fulfilled [5].

Treatment of AOSD is guided by disease severity and systemic involvement. Mild cases without organ-threatening features may respond to Non Steroidal Anti-Inflammatory Drugs (NSAIDs) such as indomethacin for fever, rash, and arthralgia. Moderate to severe disease, particularly with systemic inflammation, serositis, or organ dysfunction, requires corticosteroids, typically initiated at 0.5-1 mg/kg/day of prednisone or equivalent, with gradual tapering based on clinical response [6]. But many patients relapse when steroids are tapered, even with the addition of conventional DMARDs such as methotrexate, azathioprine, leflunomide, or cyclosporine. Tumour Necrosis Factor-alpha (TNF-α) inhibitors- including etanercept, infliximab, and adalimumab- may help patients who fail or have sub-optimal responses to steroids and Disease-modifying Anti-rheumatic Drugs (DMARDs), though their benefit is usually greater for joint symptoms than for fevers or rash. IL-1- targeted therapies like anakinra and canakinumab are more effective for systemic features, with canakinumab preferred for long-term management due to its monthly dosing. Rilonacept has also shown benefit in refractory cases, especially when anakinra is ineffective. Tocilizumab, an Interleukin-6 (IL-6) receptor blocker, is effective for both systemic and joint manifestations, and other biologics such as abatacept and rituximab have been used in difficult-to-treat AOSD [7]. A meta-analysis Ruscitti P et al., of 44 studies showed high complete remission rates with biologics in AOSD: TCZ 80%, ANK 73%, and CNK 77%, with significant corticosteroid discontinuation rates. Although three Randomised Controlled Trails (RCTs) did not show significant benefit, pooled analyses favoured Biologic DMARDs despite high heterogeneity. Overall, TCZ, ANK, and CNK are supported, but comparative effectiveness and true magnitude of benefit remain uncertain [8]. A systematic review and metanalysis by Liao J et al., showed JAK inhibitors (tofacitinib, baricitinib, ruxolitinib, upadacitinib) achieved 49% complete and 34% partial remission in mostly refractory AOSD. About 20% experienced relapse or loss of efficacy; ruxolitinib was particularly effective in MAS-associated cases [9]. Supportive measures- including gastrointestinal protection with proton pump inhibitors, calcium and vitamin D supplementation for bone health.

Complications of AOSD include MAS, chronic arthritis, serositis (pleural, pericardial, or ascitic), hepatic dysfunction, haematologic abnormalities, secondary infections, and, rarely, amyloidosis or severe pulmonary and cardiac involvement such as interstitial lung diasease, cardiac tamponade and myocarditis underscoring the need for early recognition and vigilant monitoring. Relapse risk in AOSD varies according to the disease pattern. Patients with polycyclic or chronic articular forms have the highest likelihood of recurrent flares, whereas those with a monocyclic course generally achieve complete remission with minimal risk of relapse. Factors associated with higher relapse risk include persistent arthritis, elevated inflammatory markers at baseline, delayed initiation of immunosuppressive therapy, and prior flares; regular clinical and laboratory monitoring is recommended to detect early recurrence [7].

In case report by Shad I et al., a 12-year-old girl presented with persistent high-grade fever for two weeks, salmon-coloured rash, joint pain, and cervical lymphadenopathy, initially suggesting

infectious, malignant, or autoimmune causes. Extensive testing, including blood and urine cultures, echocardiography, Brucella studies, peripheral smear, and autoimmune serologies, was unremarkable, effectively ruling out these common differentials. Her marked neutrophilic leukocytosis, thrombocytosis, borderline splenomegaly, and reactive lymph node changes pointed toward a systemic inflammatory process. A strikingly elevated serum ferritin level (5506 ng/mL) together with raised CRP provided key diagnostic support and fulfilled Yamaguchi criteria for AOSD. The patient responded promptly to corticosteroid therapy and achieved remission within one month, emphasising the importance of considering AOSD in cases of prolonged fever with unexplained inflammation [10]. In contrast present case report 45-year-old woman presented with prolonged high-grade quotidian fever for 2.5 months, evanescent salmon-coloured rash, sore throat, inflammatory arthritis, hypotension, hypoxia, and marked elevation of inflammatory markers including ferritin, initially managed as sepsis with multiorgan involvement with high grade antibiotics. Extensive infectious, autoimmune, and malignant workup was negative, and the patient fulfilled Yamaguchi criteria for AOSD. Patient was started on indomethacin due to its established efficacy in inflammatory serositis and availability, serving as adjunctive therapy prior to steroid escalation. Corticosteroid therapy resulted in rapid clinical and laboratory improvement within 48 hours. Antibiotics were subsequently discontinued after persistently sterile cultures, lack of biochemical response, and emergence of classical AOSD features, allowing safe transition to immunosuppressive therapy.

In present case report, the differential diagnosis was sepsis, lymphoma, autoimmune rheumatic disorders, drug induced Still's disease and autoimmune inflammatory disorder like MAS/ Hemophagocytic Lymphohistiocytosis (HLH). Lymphoma was considered an important differential diagnosis given the patient's prolonged fever and systemic inflammation. Absence of persistent lymphadenopathy, unremarkable peripheral smear, non significant contrast-enhanced imaging, and rapid response to corticosteroids collectively ruled out lymphoma and invasive diagnostic procedures such as lymph node biopsy were not indicated. Severe sepsis was ruled out by negative cultures ad serology. Autoimmune rheumatic disorders were ruled out by negative ANA and negative history of drug ruled out drug induced Still's disease. Hypertriglyceridemia raised the concern for MAS but normal fibrinogen, absence of cytopenias and coagulopathy ruled out MAS. Glycosylated ferritin, a highly specific marker for AOSD, was not measured due to limited availability, representing a limitation; however, markedly elevated total ferritin (more than 1000 ng/ml) with supportive clinical features strengthened the diagnosis [6].

In case report by Meenatchi R et al., a 38-year-old woman presented to the emergency department with three months of intermittent fever, body pain, swelling of multiple joints, and a one-month history of a generalised itchy macular rash. Examination revealed pallor, fever, upper-body rash, and a swollen, tender left wrist joint, with symptoms previously showing only temporary relief with NSAIDs. Investigations showed elevated serum ferritin, wrist joint erosion on X-ray, and normal echocardiography, while lymph node biopsy and bone marrow aspiration ruled out malignancy or infection. She fulfilled the major and minor Yamaguchi criteria for AOSD after excluding cancer, infectious, and rheumatologic causes. The patient improved with oral prednisolone, supportive care, and was discharged on steroids, antibiotics, antihistamines, and analgesics [11].

In a retrospective study by Krishna Prasad A et al., six patients presented with pyrexia of unknown origin over seven years, all were diagnosed with AOSD after comprehensive evaluation. The patients, with a mean age of 24.6 years, commonly exhibited prolonged fever, rash, arthropathy, hepatosplenomegaly, lymphadenopathy, neutrophilic leukocytosis, serositis, and markedly elevated serum ferritin, while ANA and RF were negative. One patient developed acute respiratory distress syndrome and died, whereas the remaining five responded well to NSAIDs, corticosteroids, and hydroxychloroquine. The study highlights the need for heightened clinical suspicion for AOSD, as early recognition is crucial despite its generally low mortality [12].

In case report by Faxas SM and Nguyen KYT the patient presented with persistent sore throat, generalised weakness, diffuse synovitis, and an atypical urticarial rash, along with the uncommon manifestation of left-sided hearing loss. Laboratory evaluation revealed markedly elevated inflammatory markers and ferritin, with high interleukin-18 levels providing key diagnostic support for AOSD. Following a six-year diagnostic course, the patient progressed from high-dose steroids to biologic therapy, ultimately achieving remission with Canakinumab after intolerance to Anakinra. The present case highlights rare clinical features, the diagnostic value of novel biomarkers, and the role of targeted biologics in managing complex or refractory AOSD [13].

In case report by Thomas S et al., a 32-year-old Nepalese woman presented with two weeks of fever, sore throat, polyarthralgia, and an evanescent rash, with labs notable for leukocytosis and markedly elevated ferritin. Extensive infectious, autoimmune, and imaging workup was negative. Based on her symptoms and laboratory findings, she met Yamaguchi criteria and was diagnosed with AOSD, improving initially with high-dose corticosteroids and NSAIDs. She became steroid-dependent, and anakinra was started but later discontinued due to biopsy-confirmed drug hypersensitivity rash. She was successfully transitioned to tocilizumab and methotrexate, allowing steroid tapering, and has remained in remission with methotrexate now being tapered [4].

Systemic complications of AOSD span multiple organ systems, including life-threatening conditions such as MAS, Disseminated Intravascular Coagulapathy (DIC), Thrombotic Themocytopenic Purpura (TTP), myocarditis, pulmonary hypertension, and fulminant hepatitis. These severe manifestations significantly influence disease management and prognosis, with infection remaining the leading cause of mortality [14].

CONCLUSION(S)

The AOSD remains a challenging diagnosis due to its non specific presentation and overlap with several infectious, autoimmune, and malignant conditions. The present case highlights the importance of maintaining a high index of suspicion in patients with prolonged fever, musculoskeletal symptoms, and elevated inflammatory markers, particularly when routine investigations are inconclusive. Early recognition using established criteria supported by markedly elevated serum ferritin- facilitates prompt initiation of therapy, which can significantly reduce morbidity. Timely diagnosis, appropriate treatment and continued follow-up are essential to improving clinical outcomes and enhancing the patient's quality of life.

REFERENCES

[1] Rao S, Tsang LS, Zhao M, Shi W, Lu Q. Adult-onset Still's disease: A disease at the crossroad of innate immunity and autoimmunity. Front Med (Lausanne). 2022;9:881431.
[2] Giacomelli R, Ruscitti P, Shoenfeld Y. A comprehensive review on adult onset Still's disease. J Autoimmun. 2018;93:24-36.
[3] Efthimiou P, Paik PK, Bielory L. Diagnosis and management of adult onset Still's disease. Ann Rheum Dis. 2006;65(5):564-72.
[4] Thomas S, Kesarwani V, Graber M, Joshi W. Adult-onset Still's disease: A case report and review of current therapeutic options. Cureus. 2022;14(3):e22743.
[5] Yamaguchi M, Ohta A, Tsunematsu T, Kasukawa R, Mizushima Y, Kashiwagi H, et al. Preliminary criteria for classification of adult Still's disease. J Rheumatol. 1992;19(3):424-30.
[6] Sapkota A, Pokhrel N, Adhikari J, Shrestha B, Yadav YK. Adult-onset Still's disease: A case report. JNMA J Nepal Med Assoc. 2020;58(222):115-18.

[7] Efthimiou P, Yadlapati S. Adult-onset still's disease. Auto-Inflammatory Syndromes. 2018:261-76.
[8] Ruscitti P, McGonagle D, Garcia VC, Rabijns H, Toennessen K, Chappell M, et al. Systematic review and metaanalysis of pharmacological interventions in adult-onset still disease and the role of biologic disease-modifying antirheumatic drugs. J Rheumatol. 2024;51(5):442-51.
[9] Liao J, Tang Q, Xie X, Zhou B, Li F, Li S. The efficacy and safety of JAK inhibitors in patients with adult-onset still's disease: A meta-analysis and systematic review. Int Immunopharmacol. 2024;137:112451.
[10] Shad I, Shafique M, Waris SA, Shabbir F, Begum A. Adult-onset Still's disease: A case report. Cureus. 2022;14(1):e21033.
[11] Meenatchi R, Karthy C, Krithika Sri JB. A case report on adult-onset still's disease. Int J Med Pharm Case Rep. 2023;16(4):37-41.
[12] Krishna Prasad A, Srujana A, Subbalaxmi MVS, Shetty M, Upadhyay AC, Rao MN. Adult onset Still's disease: 7 years' experience at a tertiary care centre from South India. J Clin Sci Res. 2014;3:224-27.
[13] Faxas SM, Nguyen KYT. Adult-onset Still's disease with atypical presentation: A case report. Med Rep. 2025;13:100272.
[14] Han ZB, Wu J, Liu J, Li HM, Guo K, Sun T. Adult-onset Still's disease evolving with multiple organ failure and death: A case report and review of the literature. World J Clin Cases. 2021;9(4):886-97.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Jan 11, 2026
- Manual Googling: Mar 23, 2026
- iThenticate Software: Mar 25, 2026 (1%)

ETYMOLOGY: Author Origin

EMENDATIONS: 7

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Jan 09, 2026**
Date of Peer Review: **Jan 27, 2026**
Date of Acceptance: **Mar 27, 2026**
Date of Publishing: **Jun 01, 2026**