Case Report

Open Access Full Text Article

A Rare Case of Sjögren's Syndrome Associated with Autoimmune Hemolytic Anemia, Palindromic Rheumatism, and Hepatitis B

Errol Pinto[1], Durga Prasad Verma[1]

[1] Medicine, Hind Institute of Medical Sciences, Sitapur, Uttar Pradesh, India.
Corresponding Author: Errol Pinto; Email: errolpinto99@gmail.com

Abstract

Background: Sjögren syndrome (SS) is a chronic systemic autoimmune disease that mostly causes dryness in moist areas such as the mouth or eyes, although it may include other parts of the body. Hematological abnormalities such as autoimmune hemolytic anemia are infrequent but can be confusing if they are dominant at the onset of the disease. We encountered a case of SS presenting with profound autoimmune hemolytic anemia (AIHA) and hepatitis B virus (HBV) infection. Immunosuppression therapy could theoretically increase the risks for reactivation of the chronic HBV infection. **Case Description**: A 32-year-old woman came with severe general malaise for 15 days, yellow staining of the eye and the body and migratory joint pain. At presentation, pallor, icterus, heart rate 110 beats/min and hepatosplenomegaly were documented. Laboratory tests revealed Hb 2.7 g/dL with remarkable reticulocytosis, bilirubin levels elevated at 18 mg/dL with indirect bilirubin 14 mg/dL, transaminases elevated with aspartate transaminase AST 228 U/L and alanine transaminase ALT 235 U/L and a direct Coombs test was positive for IgG and C3d. Laboratory testing revealed a positive ANA and strong positive results for SS-A, SS-B and Ro52 antibodies. Screening for hepatitis viruses showed HBsAg positive, but the HBV DNA load was only moderate (2261 IU/mL). She was diagnosed with SS associated with AIHA, palindromic rheumatism and chronic hepatitis B infection. She was given pulse methylprednisolone for 3 consecutive days, followed by oral prednisolone at a taper dosage. She was also started on tenofovir and hydroxychloroquine. Hb and liver enzymes slowly improved. **Conclusion:** SS can cause significant AIHA as the first prominent symptom, especially in young women without classical dryness, leading to the delayed diagnosis of the autoimmune disorder. Autoimmune screening in parallel with screening for chronic viral infection is recommended to identify potential risk factors before any initiation of immunotherapy, among hepatitis B carriers.

Keywords: Sjögren's Syndrome; Anemia, Hemolytic, Autoimmune; Hepatitis B, Chronic; Palindromic Rheumatism; Autoantibodies; Coombs Test; Hyperbilirubinemia; Glucocorticoids

Introduction

Sjögren's syndrome is a chronic, systemic, autoimmune disease predominantly characterized by xerostomia and xerophthalmia, though it can affect various organs and systems, including the musculoskeletal, pulmonary, renal, nervous, hepatobiliary and hematopoietic organs, making it difficult to diagnose when the presentation differs greatly from these two classical features [1].

The American College of Rheumatology/European League Against Rheumatism classification criteria were updated in 2016 to reflect the relative diagnostic importance of positive antibodies for anti-SSA/Ro, focal lymphocytic sialadenitis (an average of ≥1 lymphoid focus/4 mm²), objective ocular staining with rose bengal or fluorescein, Schirmer testing and salivary flow rate measurement. Anti-Ro/SSA and anti-La/SSB antibodies continue to be of major clinical importance in Sjögren's syndrome and these antibodies are typically related to the systemic manifestation of disease than the isolation of gland manifestations. Hematologic features are also important extraglandular manifestations that can arise in patients with primary Sjögren's. These are most often represented by anemia, leukopenia, thrombocytopenia, lymphopenia, monoclonal gammopathy, and, less commonly, autoimmune cytopenias such as Coombs' positivity and/or autoimmune neutropenia or thrombocytopenia [2].

Primary Sjögren's patients may have anemia, cytopenias, monoclonal gammopathies and lymphoproliferative disorders, emphasizing the need to test patients with an abnormal or unexplained cytopenia for Sjögren's [3]. In earlier clinicopathological studies, hematologic abnormalities were described in as many as 11-47% of patients with Sjögren's syndrome, with 20-34% testing positive by direct antiglobulin test (DAT), some of whom did not show clinical evidence of hemolysis [4].

Autoimmune hemolytic anemia is an uncommon but clinically significant hematological manifestation of Sjögren's syndrome. It may precede, accompany, or follow the diagnosis of Sjögren's syndrome and can sometimes be the first major clue to an underlying systemic autoimmune disease [5,6]. Wen et al., in a large-scale cross-sectional study of hospitalized patients with primary Sjögren's syndrome, reported autoimmune hemolytic anemia as an uncommon manifestation, with a prevalence of 2.8% among 565 patients [5]. Earlier reports also described autoimmune hemolytic anemia preceding the clinical

recognition of Sjögren's syndrome, suggesting that patients initially labelled as having "idiopathic" autoimmune hemolytic anemia may later demonstrate systemic autoimmune features [6]. Therefore, when severe anemia is accompanied by jaundice, reticulocytosis, hepatosplenomegaly, or a positive direct antiglobulin test, evaluation for an autoimmune connective tissue disease becomes clinically relevant. In case reports of Sjögren's syndrome presenting with autoimmune cytopenia, patients often require close integration of hematological and rheumatological evaluation because the initial clinical presentation may be dominated by hematological abnormalities rather than sicca symptoms [7].

The diagnosis of autoimmune hemolytic anemia depends on the demonstration of hemolysis and immune-mediated red cell destruction. The direct antiglobulin test remains central to diagnosis, and warm autoimmune hemolytic anemia is typically associated with IgG-mediated red cell sensitization, with or without complement deposition [8]. International consensus recommendations state that corticosteroids remain the first-line treatment for warm autoimmune hemolytic anemia, although the addition of rituximab may be considered early in severe cases or when response is inadequate [8].

Another complication for these patients is palindromic rheumatism, a self-limiting inflammatory arthritis or periarthritis with periods of remission in between [9]. While palindromic rheumatism usually is related to Rheumatoid Arthritis (RA), it might occur in association with, precede or occur alongside various autoimmune connective tissue diseases. Because joints are only intermittently involved in this condition, its development in these patients may delay its diagnosis, especially if more acute manifestations like anemia or jaundice dominate their clinical presentation. Recurrent migratory arthralgia should thus be considered a manifestation of connective tissue disease in patients positive for RF or any of the various systemic autoantibodies than a sole issue involving the joints. A further problem is concomitant hepatitis B infection. Several immunosuppressive therapies that may be employed for autoimmune hemolysis (e.g., systemic corticosteroids and B-cell depleting therapy) can lead to reactivation of hepatitis B. American Association for studies in Liver Diseases (AASLD) suggests that patients positive for HBsAg should be given prophylactic antiviral therapy prior to initiation of immunosuppression or cytotoxic therapy [10]. We consider tenofovir and entecavir the optimal choices due to their effectiveness and the high bar to the development of resistance to these antivirals. Thus, for patients who must be treated with corticosteroids for autoimmune hemolytic anemia, HBsAg screening followed by timely prophylaxis are essential management components. We present this case of a patient who had severe anemia, jaundice, hepatosplenomegaly, repeated joint pains, a positive direct Coombs test, strongly positive SS-A and SS-B antibodies and also had an infection with hepatitis B. This is clinically interesting as it created an overlap of autoimmune disease with viral issues. This case brings attention to the necessity of a comprehensive autoimmune workup in the setting of an unexplained severe anemia with systemic symptoms in patients not presenting with the classic clinical spectrum of Sjögren syndrome. Additionally, it highlights the challenge of finding a therapeutic regimen to adequately treat immune-mediated hemolysis while protecting against reactivation of hepatitis B virus during immunosuppressive treatment.

Case Presentation

The patient was a 32-year-old female admitted to the Department of Medicine with complaints of generalized weakness lasting 15 days, jaundice of the sclera and skin for about 15 days, and intermittent joint pain in the same period **(Figure 1)**. Her blood pressure was 98/56 mm Hg, pulse rate was 125 beats/min, respiratory rate was 24 cycles/min, and temperature was 100.1°F. General examination revealed marked pallor and icterus. Abdominal examination showed hepatosplenomegaly.

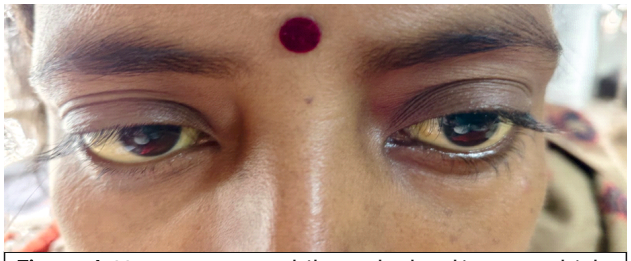

Figure 1: Here we can see bilateral scleral icterus which is consistent with marked hyperbilirubinemia.

Initial laboratory evaluation revealed severe anemia with hemoglobin of 2.7 g/dL. The total leukocyte count was 5,400/mm³ and platelet count was 1.40 lakh/mm³. Peripheral smear showed markedly reduced red blood cells with anisopoikilocytosis, macrocytes, microcytes, and pencil cells, while white blood cells were normal and platelets were adequate. Reticulocyte count was markedly elevated at approximately 35%. Liver function tests were deranged, with total bilirubin 23.4 mg/dL, direct bilirubin 14.6 mg/dL, SGOT 1117 U/L, SGPT 953 U/L, and alkaline phosphatase 466 U/L. Renal function was preserved, with urea 72 mg/dL and serum creatinine 0.68 mg/dL. Ultrasonography of the abdomen confirmed hepatosplenomegaly. Screening for HIV, HCV, dengue, typhoid, and malaria was negative, while HBsAg was reactive with detectable viral load. A summary of the patients laboratory reports is shown in **Table 1.**

During admission, further evaluation was performed. Direct Coombs test was positive **(Figure 2),** with IgG 3+

Table 1: A summary of patients laboratory reports

Test	Result	Test	Result
RA Factor	~29.8 IU/mL	HIV	Non -reactive
CRP	~7 mg/L	HBsAg	Reactive (Viral Load detected)
Procalcitonin	24.4 ng/mL	HCV	Non -reactive
Ferritin	1622 ng/mL	Dengue	Negative
LDH	557 U/L	Typhi IgM	Negative
Vitamin B12	~673 pg/mL	Malaria	Negative
TSH	~0.99	USG Abdomen	Hepato -spleenomegaly
RBC	Moderately reduced	Special cells	Pencil cells seen
Morphology	Macrocytes + microcytes	WBC	Normal
Anisopoikilocytosis	Present	Platelets	Adequate
		Reticulocyte count	~35% ↑

and C3d 4+. ANA by immunofluorescence was positive. ENA profile showed strong positivity for SS-A, SS-B, and Ro-52, along with positive histone antibodies **(Figure3).** Rheumatoid factor was approximately 29.8 IU/mL, CRP was approximately 7 mg/L, procalcitonin was 24.4 ng/mL, ferritin was 1622 ng/mL, LDH was 557 U/L, vitamin B12 was approximately 673 pg/mL, and TSH was 0.99. Based on the clinical presentation and investigations, she was diagnosed as a case of Sjögren's syndrome with autoimmune hemolytic anemia, palindromic rheumatism, and chronic hepatitis B infection. She was managed with pulse methylprednisolone for 3 days, followed by oral prednisolone at discharge with a planned taper. Tenofovir was started in view of hepatitis B infection, and hydroxychloroquine was added for joint symptoms. Supportive care and close monitoring of hematological and

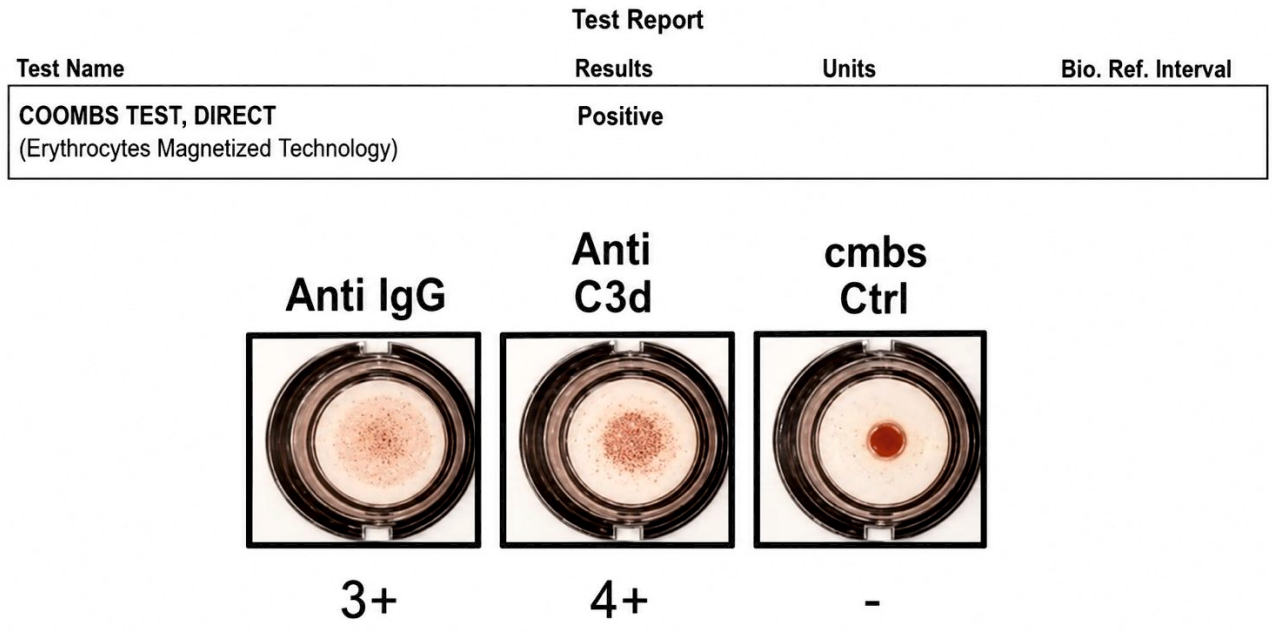

Figure 2: Positive direct Coombs test supporting autoimmune hemolytic anemia

SEROLOGY AND IMMUNOLOGY REPORT

Anti Nuclear Antibody (ANA) By IFA (HEP-2)

Anti Nuclear Antibody by IFA	Positive	Negative
Pattern	Nucleus	
Grade	++	
Estimated Titre	1:100	

Interpretation:
Guidelines (Sample screening Dilution - 1:100):

Negative : No Immunofluorescence
+ : Weak Positive
++ : Moderate Positive
+++ : Strong Positive
++++ : Very strong Positive

Test Report

Test Name	Results	Units	Bio. Ref. Interval
EXTRACTABLE NUCLEAR ANTIGENS (ENA)/ANTI NUCLEAR ANTIBODIES (ANA), QUALITATIVE PROFILE (LIA)			
U1-nRNP/Sm	Negative		
Sm	Negative		
SS-A	**Strong Positive +++**		
Ro- 52	**Strong Positive +++**		
SS-B/La	**Strong Positive +++**		
Scl - 70	Negative		
PM-Scl	Negative		
Jo - 1	Negative		
CENP-B	Negative		
PCNA	Negative		
ds-DNA	Negative		
Nucleosomes	Negative		
Histones	**Positive ++**		
Rib- P Protein	Negative		
AMA- M2	Negative		

Figure 3: Autoimmune serological profile supporting Sjögren's syndrome.

biochemical parameters were continued during hospitalization.

The patient showed gradual clinical and laboratory improvement. By discharge, hemoglobin had improved to 7.2 g/dL, total leukocyte count was 5,300/mm³, and platelet count was 1.44 lakh/mm³. A comparison of intial and later laboratory parameters is shown in **Table 2.** Total bilirubin decreased to 6.0 mg/dL, direct bilirubin to 4.7 mg/dL, SGOT to 74 U/L, SGPT to 257 U/L, and alkaline phosphatase to 230 U/L. Renal function remained stable, with urea 50 mg/dL and serum creatinine 0.61 mg/dL. She was discharged in stable condition on oral medications with advice for regular follow-up, serial complete blood counts, reticulocyte counts, liver function monitoring, and specialist follow-up.

At 1-month follow-up, she reported clinical improvement. Her hemoglobin had increased to 11.2 g/dL, total leukocyte count was 7200/mm³, platelet count was 2.26 lakh/mm³, and MCV was 93.1 fL. Liver function tests also improved, with total bilirubin 2.1 mg/dL, direct bilirubin 0.8 mg/dL, indirect bilirubin 1.3 mg/dL, SGOT 92 U/L, SGPT 117 U/L, and alkaline phosphatase 208 U/L. Renal function and electrolytes remained within acceptable limits, with urea 40 mg/dL, creatinine 0.81 mg/dL, sodium 137 mmol/L, and potassium 4.3 mmol/L. The post treatment evaluation of the patient is shown in **Table 3**.

Table 2: A comparison of laboratory parameters on presentation and discharge

Parameter	Initial Value	Final Value
Hemoglobin (Hb)	2.7 g/dL	7.2 g/dL
TLC	5,400	5,300
Platelets	1.40 lakh	1.44 lakh
MCV	117	138
MCH	69	43
RDW	10.8	14.8
DLC	P70 L21 M04 E05	P81 L16 M1 E1
Urea	72	50
Creatinine	0.68	0.61
Sodium	132	137
Potassium	3.9	4.2
Total Bilirubin	23.4 mg/dL	6.0 mg/dL
Direct Bilirubin	14.6 mg/dL	4.7 mg/dL
SGOT (AST)	1117 U/L	74 U/L
SGPT (ALT)	953 U/L	257 U/L
ALP	466 U/L	230 U/L
Total Protein	5.8 g/dL	—
Albumin	3.2 g/dL	—

Table 3: 1 month Post Treatment evaluation of the patient

INVESTIGATION	FINDING
Hb TLC PC MCV	11.2 g/dL 7200 2.26 lakh 93.1
T. Bilirubin Direct Bilirubin Indirect Bilirubin SGOT SGPT ALP	2.1mg/dL 0.8 1.3 92 117 208
Urea Creatinine	40 0.81mg/dL
Sodium Potassium	137 4.3

Discussion

This case demonstrates the difficulty in diagnosis that can be expected when there are severe blood abnormalities seen alongside systemic autoimmunity and chronic virus infections. Sjögren's syndrome is traditionally diagnosed through the presence of sicca symptoms, but it is currently accepted as a systemic autoimmunity condition which can affect several organs and organ systems. According to the American college of Rheumatology/European League Against Rheumatism classification criteria for Sjögren's syndrome in 2016, there is considerable emphasis placed on the presence of anti-SSA/Ro antibodies and an objective evaluation of glands, but in actual clinical settings, patients can manifest extra-glandular symptoms first before eye and mouth dryness [1]. In the present case, the patient's

dominant clinical problems at admission were profound generalized weakness, jaundice, hepatosplenomegaly, and joint pain, with laboratory evaluation showing life-threatening anemia, marked hyperbilirubinemia, transaminitis, positive direct Coombs test, and strong positivity for SS-A, SS-B, and Ro-52 antibodies. These findings made the case diagnostically challenging, as the initial presentation was led by hematological and hepatobiliary abnormalities rather than classical sicca complaints.

Hematological manifestations of Sjögren's syndrome are well described but remain under-recognized in routine clinical practice. Manganelli et al. reported that anemia, leukopenia, thrombocytopenia, monoclonal gammopathy, and lymphoproliferative disorders may occur in primary Sjögren's syndrome [3]. Similarly, Ramakrishna et al. highlighted that hematological abnormalities may form an important part of the disease spectrum and may occasionally dominate the clinical presentation [4]. In this patient, the degree of anemia was severe, with an initial hemoglobin of 2.7 g/dL, and the presence of jaundice, hepatosplenomegaly, markedly elevated reticulocyte count, raised LDH, and direct antiglobulin test positivity supported immune-mediated hemolysis. The case therefore reinforces the importance of considering an autoimmune etiology in young patients presenting with unexplained severe anemia, especially when anemia is accompanied by systemic symptoms such as fever, arthralgia, jaundice, or organomegaly.

It is rare but significant when autoimmune hemolytic anemia occurs alongside Sjögren's syndrome. A minority of patients with primary Sjögren's syndrome in the study by Wen et al. Experienced autoimmune hemolytic anemia, although the authors stated the disorder occurred in only a few patients. Early studies report on Sjögren's syndrome cases occurring years after a diagnosis of autoimmune hemolytic anemia, indicating that hematological disorder can serve as the first evidence of an underlying connective tissue disorder [6]. This is evident in the current case study where anemia and jaundice were presenting symptoms. Likewise, primary Sjögren's syndrome presented with autoimmune cytopenia in Meena and Bohra's description [7], showing that the signs of autoimmune hematologic disease may precede or overshadow classic symptoms in some individuals with Sjögren's syndrome. Because of these potential correlations, patients who present with autoimmune hemolytic anemia should be investigated for underlying autoimmune disorders if they have any features such as joint pain, a positive ANA, positive rheumatoid factor or a positive ENA profile.

Positive results on the direct Coombs test, including the reactivity for both IgG and C3d, helped establish immune-mediated hemolysis. As recommended in guidelines based on international consensus, the diagnosis of autoimmune hemolytic anemia includes correlation of clinical, biochemical and laboratory evidence for hemolysis and autoimmune hematologic evidence. Corticosteroids are recommended as the first line of treatment in cases of warm autoimmune hemolytic anemia [8]. The patient in this case responded to pulse methylprednisolone followed by oral prednisolone treatment with an improvement in her hemoglobin and liver parameters from 2.7 g/dL on admission to 7.2 g/dL at discharge and 11.2 g/dL at the 1-month follow-up visit.

The patient had joint pain recurrently, which opened the clinical perspective of the case. Palindromic rheumatism consists of attacks of joint pain or arthritis that appear and disappear and that can be a distinct clinical entity or the manifestation of an overlap in systemic autoimmune disease [9]. In this patient, joint manifestations, rheumatoid factor and strong autoimmune serologic results pointed to the need for evaluation by a rheumatologist. Hydroxychloroquine was prescribed to manage her palindromic rheumatism. The autoimmune nature of the arthralgia and other components of her presentation was evidenced by the serologic findings and the positive rheumatoid factor. Thus, musculoskeletal manifestations in this patient were part of a wider autoimmune spectrum.

Coexistence with chronic hepatitis B infection represented a major management difficulty because immunosuppression could trigger hepatitis B virus reactivation. According to the AASLD 2018 recommendations, antiviral therapy should be administered for HBsAg-positive patients before immunosuppression [10]. In this case, tenofovir was initiated together with corticosteroids. The immediate need to treat autoimmune hemolytic anemia needs to be weighed against the risks associated with hepatitis B virus reactivation and worsened hepatitis. This approach mitigated the risks while providing effective therapy for her autoimmune condition. Liver function test results improved with follow-up.

Anti-Ro/SSA and anti-La/SSB antibodies are the most useful autoantibodies for Sjögren's syndrome, especially concerning those with systemic disease expression [2]. The strong positive staining of SS-A, SS-B and Ro-52 was a plausible autoimmune reason for the symptoms experienced in this patient. The positive histone antibodies further reflected broad autoimmune reactivity, though the interpretation and significance need to be considered carefully with her medications and other features of autoimmunity. These findings suggest that ENA testing can be helpful for an unexplained cytopenia with a broader presentation.

This case report is strong because of the uncommon presentation and complete evaluation, as well as clear

clinical improvements at admission to the follow-up stage. It provides an example of a rare association of Sjögren's syndrome with autoimmune hemolytic anemia, palindromic rheumatism and hepatitis B and underscores the need for hepatitis B screening in patients before initiating therapy with immunomodulators. There are limitations because this is an anecdotal experience, the objective sicca assessment with the Schirmer test, ocular staining score, salivary flow rate testing or minor salivary gland biopsy findings are not reported in the study and the long-term outcome of the patient is not yet established. Additionally, it is not possible to find out the distinct effects of autoimmune hemolytic anemia, chronic hepatitis B virus infection and inflammation in the liver about the jaundiced presentation and deranged liver functions from the clinical data available.

CLINICAL, ETHICAL AND SOCIAL IMPLICATIONS

1. Unexplained severe anemia with jaundice and positive Coombs test should prompt autoimmune evaluations.
2. Viral screening should be mandatory before starting immunosuppressive therapy.
3. Treatment should balance benefit and harm.
4. Young women with nonspecific symptoms such as fatigue, jaundice and joint pains often face diagnostic delay.
5. Access to advanced diagnostic tests matters in resource limited settings where autoimmune presentations are frequently missed.

Conclusion

This report documents a case of Sjögren's Syndrome characterized by severe autoimmune hemolytic anemia, as well as the presence of palindromic rheumatism and chronic infection with hepatitis B. The patient's primary complaints were significant anemia, jaundice, enlargement of the spleen and liver and joint pain, which overshadowed any typical dryness-related complaints, tests revealed a positive Coombs test and significant presence of SS-A, SS-B and Ro-52 antibodies. Management was complicated by the coexistence of chronic hepatitis B and was further enhanced by antiviral treatment initiated to prevent viral reactivation during treatment. Prompt assessment led to thorough testing, diligent observation and marked improvements both clinically and from laboratory markers.

Declarations

Acknowledgment: None

AI Declaration: No artificial intelligence tools were used in making this manuscript.

Author Contribution: EP and DPV conceptualised, prepared initial draft, performed literature review and administered this case report. Both authors also approve the final manuscript and EP agrees to be accountable for all aspects of this work.

Patient Consent : Appropriate written consent for the publication was obtained for the publication of this case report.

Conflict of interest : None to declare

Funding : None

How to cite: Pinto E, Verma DP. A Rare Case of Sjögren's Syndrome Associated with Autoimmune Hemolytic Anemia, Palindromic Rheumatism, and Hepatitis B. Ann Med Med Ethics. 2026;1(1):13-20.

DOI: https://doi.org/10.66765/amme.2026.004

Submission received: 3rd May 2026
Submission accepted: 13th May 2026
Submission published: 13th June 2026

ORCID:
Errol Pinto
http://orcid.org/0009-0004-1365-780X
Durga Prasad Verma
http://orcid.org/0009-0008-8433-2550

Reference

1. Shiboski CH, Shiboski SC, Seror R, Criswell LA, Labetoulle M, Lietman TM, Rasmussen A, Scofield H, Vitali C, Bowman SJ, Mariette X. 2016 American College of Rheumatology/European League Against Rheumatism classification criteria for primary Sjögren's syndrome: a consensus and data-driven methodology involving three international patient cohorts. Annals of the rheumatic diseases. 2017 Jan 1;76(1):9-16.
2. Fayyaz A, Kurien BT, Scofield H. Autoantibodies in Sjögren's syndrome. Rheumatic diseases clinics of North America. 2016 Jun 21;42(3):419.
3. Manganelli P, Fietta P, Quaini F. Hematologic manifestations of primary Sjögren's syndrome. Clinical and experimental rheumatology. 2006 Jul 1;24(4):438.
4. Ramakrishna R, Chaudhuri K, Sturgess A, Manoharan A. Haematological manifestations of primary Sjögren's syndrome: a clinicopathological study. QJM:

An International Journal of Medicine. 1992 Jul 1;83(4):547-54.

5. Wen W, Liu Y, Zhao C, Sun X, Zhang C, Li Z. Clinical and serologic features of primary Sjögren's syndrome concomitant with autoimmune hemolytic anemia: a large-scale cross-sectional study. Clinical rheumatology. 2015 Nov;34(11):1877-84.
6. Schattner A, Shtalrid M, Berrebi A. Autoimmune hemolytic anemia preceding Sjögren's syndrome. The Journal of rheumatology. 1983 Jun 1;10(3):482-4.
7. Meena DS, Bohra GK. Primary Sjögren's syndrome presenting as autoimmune cytopenia. Clinics and Practice. 2019 Nov 6;9(4):1190.
8. Jäger U, Barcellini W, Broome CM, Gertz MA, Hill A, Hill QA, Jilma B, Kuter DJ, Michel M, Montillo M, Röth A. Diagnosis and treatment of autoimmune hemolytic anemia in adults: Recommendations from the First International Consensus Meeting. Blood reviews. 2020 May 1;41:100648.
9. Sanmartí R, Haro I, Cañete JD. Palindromic rheumatism: a unique and enigmatic entity with a complex relationship with rheumatoid arthritis. Expert Review of Clinical Immunology. 2021 Apr 3;17(4):375-84.
10. Terrault NA, Lok AS, McMahon BJ, Chang KM, Hwang JP, Jonas MM, Brown Jr RS, Bzowej NH, Wong JB. Update on prevention, diagnosis, and treatment of chronic hepatitis B: AASLD 2018 hepatitis B guidance. Hepatology. 2018 Apr;67(4):1560-99.