World Journal of Pharmaceutical Science and Research
www.wjpsronline.com
Case Report
ISSN: 2583-6579
SJIF Impact Factor: 5.111
Year - 2025
Volume: 4; Issue: 5
Page: 932-939

STEROID-INDUCED DIABETES MELLITUS IN A PATIENT WITH RHEUMATOID ARTHRITIS: A CASE REPORT

Dr. Omer Bin Amer*[1], Vivek Kappali[2], Shaik Abdul Kareem[3], S. Mirudula[4], Harsha Alugula[5], Nandhini P.[6], M. Ragul[7], Dr. U. Kalyan Kumar[8], Dr. Asfia Mohammed Raffi[9], Akuleti Chandra Prakash[10]

Pharm D (Doctor of Pharmacy)
[1]Pharm D (Doctor of Pharmacy), Omerbadawood5069@gmail.com
[2]Pharm D (Doctor of Pharmacy), vivekkappali1950@gmail.com
[3]Pharm D (Doctor of Pharmacy), abdulkareemgmbhh@gmail.com
[4]Pharm D (Doctor of Pharmacy), mirudula.s310@gmail.com
[5]Pharm D (Doctor of Pharmacy), harshaalugula06@gmail.com
[6]M. Pharm, Pharmacy Practice, nandhinimedicine@gmail.com
[7]M. Pharm, Pharmacy Practice, ragulmariyappan143@gmail.com
[8]Pharm D (Doctor of Pharmacy), ukalyan07@gmail.com
[9]Pharm D (Doctor of Pharmacy), ayanayman81@gmail.com
[10]Pharm D (Doctor of Pharmacy), achandraprakash2805@gmail.com

Article Received: 27 September 2025 | | ***Article Revised: 16 October 2025*** | | ***Article Accepted: 08 November 2025***

Corresponding Author: Dr. Omer Bin Amer
Pharm D (Doctor of Pharmacy).
DOI: https://doi.org/10.5281/zenodo.17607113

How to cite this Article: Dr. Omer Bin Amer, Vivek Kappali, Shaik Abdul Kareem, S. Mirudula, Harsha Alugula, Nandhini P., M. Ragul, Dr. U. Kalyan Kumar, Dr. Asfia Mohammed Raffi, Akuleti Chandra Prakash (2025). STEROID-INDUCED DIABETES MELLITUS IN A PATIENT WITH RHEUMATOID ARTHRITIS: A CASE REPORT. World Journal of Pharmaceutical Science and Research, 4(5), 932-939. https://doi.org/10.5281/zenodo.17607113

ABSTRACT

Background: Steroid-induced diabetes mellitus (SIDM) is a well-known metabolic complication of long-term corticosteroid therapy, particularly in patients with autoimmune disorders like rheumatoid arthritis (RA). It is vital to recognize and manage the condition early in order to avoid the complications that are both acute and long-term. **Case Presentation:** A 52-year-old woman with seropositive RA on long-term prednisolone treatment (10–15 mg/day) showed symptoms of polyuria, polydipsia, general weakness, and slight loss of weight. She had no previous diagnosis of diabetes and no family history of metabolic disorders. Laboratory tests showed high fasting plasma glucose (164 mg/dL), postprandial glucose (268 mg/dL), and HbA1c (7.4%), while insulin, C-peptide, and cortisol levels were normal. The patient was diagnosed with diabetes due to steroids. **Management and Outcome:** Prednisolone was reduced gradually, and metformin therapy was started with lifestyle changes including a low-carb diet and regular exercise. After 12 weeks, fasting and postprandial glucose levels were normal (102 mg/dL and 146 mg/dL, respectively) and HbA1c dropped to 6.1%, while RA was still well-controlled with DMARDs. **Conclusion:** This case demonstrates that early detection, proper steroid tapering, drug therapy, and lifestyle changes are all necessary in the effective management of SIDM.

KEYWORDS: Steroid-induced diabetes mellitus, Rheumatoid arthritis, Corticosteroids, Hyperglycemia, Case report, Glucocorticoid therapy, New-onset diabetes, Metabolic complications.

INTRODUCTION

Rheumatoid arthritis (RA) is a persistent autoimmune inflammatory disorder that primarily targets the synovial joints, causing pain, swelling, and ultimately destruction of the joints. The disease is primarily managed with the use of disease-modifying antirheumatic drugs (DMARDs) as well as corticosteroids to keep the flares under control and prevent systemic inflammation. Prednisolone, one of the corticosteroids, is still the most effective agent in managing acute exacerbations due to its strong anti-inflammatory and immunosuppressive properties.[1] Nevertheless, long-term use of steroids is fraught with a variety of side effects, one of which is the aforementioned osteoporosis, hypertension, dyslipidemia, and glucose metabolism disturbances. Among them, one specific effect that is steroid-induced diabetes mellitus (SIDM) is a complication that is both clinically important and frequently overlooked.[2]

The main cause of steroid-induced diabetes is the metabolic actions of glucocorticoids, which increase liver gluconeogenesis, decrease glucose uptake into peripheral tissues, and reduce insulin secretion from pancreatic β-cells. The combination of these processes results in insulin resistance and hyperglycemia, which may occur even in patients who do not have a history of diabetes.[3] The rate of occurrence of SIDM depends on the amount, length of treatment, and type of steroid as well as on the patient's age, body mass index, and genetic factors. It is essential to detect the condition early in order to ward off acute complications such as hyperosmolar hyperglycemic states and long-term cardiovascular risk.[4]

Patients who are on long-term corticosteroid treatment for autoimmune diseases such as rheumatoid arthritis, systemic lupus erythematosus, and polymyalgia rheumatica, are at a higher risk for SIDM. The clinical manifestations usually consist of polyuria, polydipsia, fatigue, and weight loss, however, they can be very mild or misinterpreted as the effects of the disease or the steroids. [5] Laboratory testing most commonly reveals increased fasting and postprandial blood glucose and elevated HbA1c levels. It is very important to differentiate SIDM from other types of diabetes, including type 1, type 2, and latent autoimmune diabetes in adults (LADA), to ensure the correct management.[6]

The management of SIDM is a complex process that includes strategies such as tapering or reducing steroid use, starting pharmacological treatment with metformin, and supporting diet and exercise as lifestyle changes. Metformin is chosen because of its effects on insulin sensitivity and its low potential to cause hypoglycemia. Regular checks of glucose levels, coupled with educating the patient, are important to make sure that the intervention is done at the right time.[7] The cooperation of different specialists, such as rheumatologists, endocrinologists, and clinical pharmacists, leads to better results by maintaining a balance between glycemic control and the continued treatment of the autoimmune disease.[8]

Even though SIDM is common, it remains mostly unrecognized in patients who do not show typical risk factors for diabetes. The knowledge of the time frame of corticosteroid use and hyperglycemia, regular monitoring and early treatment are all necessary to stop the suffering caused by hyperglycemia.[9] The evidence from case reports and observational studies gives the impression that timely recognition and management can result in partial or complete reversal of hyperglycemia. This case presents a patient with RA who developed SIDM during long-term steroid therapy, thus putting stress on the necessity of alertness, multidisciplinary care, and personalized treatment options for the best metabolic and rheumatologic outcomes.[10]

Patient Details

A 52-year-old female, married, a homemaker, living in a city, went to the rheumatology outpatient department of the tertiary care hospital. It was already known that she had seropositive rheumatoid arthritis (RA) for six years. The patient weight was 60 kg and height 158 cm (BMI 23.5 kg/m²).

Chief Complaints

The patient presented with the following complaints for the past two months:

- Increased frequency of urination (polyuria)
- Excessive thirst (polydipsia)
- Generalized weakness and easy fatigability
- Mild blurring of vision

There was no history of fever, nausea, vomiting, or abdominal pain.

History of Present Illness

For the last eight months, the patient had been taking oral corticosteroids continuously for rheumatoid arthritis. At first, the doctor prescribed her a daily dose of 15 mg of prednisolone and later reduced it to 10 mg for maintenance therapy. She was starting to feel increased thirst and urination, particularly in the afternoon and evening, two months before her visit to the hospital. She also faced fatigue all over her body and unintentional weight loss (~2 kg in two months). There was no occurrence of nocturnal hypoglycemia, altered sensorium, or signs suggestive of ketoacidosis. She als said that there were no recent infections, no new medications, or changes in diet. She had neither recent admission to the hospital nor intravenous steroid administration.

Past Medical History

- Rheumatoid arthritis (seropositive) for 6 years
- No history of diabetes mellitus, hypertension, thyroid disorders, renal disease, or dyslipidemia prior to steroid use.
- No previous hospitalizations for metabolic or infectious causes.

Past Medication History

- Prednisolone: 15 mg/day for 8 months, currently on 10 mg/day
- Methotrexate: 15 mg once weekly
- Hydroxychloroquine: 200 mg twice daily
- Folic acid: 5 mg/day
- Calcium and vitamin D supplements regularly
- No use of other drugs associated with hyperglycemia (such as thiazides, antipsychotics, or protease inhibitors).

Family History

- No family history of diabetes mellitus, hypertension, obesity, thyroid disease, or autoimmune disorders.
- Parents were non-diabetic and non-hypertensive.
- No hereditary metabolic or endocrine conditions reported.

Social History

- Non-smoker, non-alcoholic.
- Diet: mixed, with moderate carbohydrate intake.
- Physical activity: light household work; sedentary otherwise.
- Socioeconomic status: middle-class; access to regular healthcare.

General Examination

- **General appearance:** Alert, cooperative, not in distress.
- **Vital signs:** BP 126/80 mmHg; Pulse 84/min; Temp 98.4°F; RR 18/min.
- **Anthropometry:** Weight 58 kg, BMI 23.2 kg/m².
- **Systemic examination:**
  - Musculoskeletal: Swelling and tenderness in small joints of both hands, consistent with active RA.
  - No signs of Cushingoid features (no moon face, truncal obesity, or striae).
  - No pedal edema or lymphadenopathy.
  - Cardiovascular, respiratory, and abdominal examinations were unremarkable.

Laboratory and Diagnostic Investigations

Parameter	Result	Reference Range	Interpretation
Complete Blood Count (CBC)			
Hemoglobin	12.6 g/dL	12–15 g/dL	Normal
Total Leukocyte Count	7,800 /mm³	4,000–11,000 /mm³	Normal
Platelet Count	2.6×10^5 /µL	$1.5–4.0 \times 10^5$ /µL	Normal
Fasting Plasma Glucose (FPG)	164 mg/dL	70–100 mg/dL	Elevated
Postprandial Plasma Glucose (PPG)	268 mg/dL	<140 mg/dL	Elevated
HbA1c	7.4 %	<5.7 %	Consistent with diabetes
Serum Insulin (Fasting)	9.8 µIU/mL	2–25 µIU/mL	Normal
C-Peptide (Fasting)	2.1 ng/mL	0.8–3.1 ng/mL	Normal insulin secretion
Serum Cortisol (Morning)	13.5 µg/dL	5–25 µg/dL	Normal; rules out Cushing's
Thyroid-Stimulating Hormone (TSH)	2.6 µIU/mL	0.4–4.2 µIU/mL	Normal
Liver Function Tests (LFTs)			
Total Bilirubin	0.7 mg/dL	0.2–1.2 mg/dL	Normal
SGOT (AST)	28 U/L	<40 U/L	Normal
SGPT (ALT)	31 U/L	<40 U/L	Normal
Alkaline Phosphatase	82 U/L	44–147 U/L	Normal
Serum Albumin	4.3 g/dL	3.5–5.0 g/dL	Normal
Renal Function Tests (RFTs)			
Serum Urea	24 mg/dL	10–45 mg/dL	Normal
Serum Creatinine	0.8 mg/dL	0.6–1.2 mg/dL	Normal
eGFR	>90 mL/min/1.73 m²	>90	Normal
Electrolytes			
Sodium	138 mmol/L	135–145 mmol/L	Normal
Potassium	4.2 mmol/L	3.5–5.0 mmol/L	Normal
Chloride	102 mmol/L	98–107 mmol/L	Normal
Urinalysis			
Appearance	Clear	—	Normal
Specific Gravity	1.015	1.010–1.030	Normal
Glucose	Positive	Negative	Glycosuria present
Ketones	Negative	Negative	No ketoacidosis
Protein	Negative	Negative	Normal
Microscopy	Normal	—	No infection
Lipid Profile			

Total Cholesterol	202 mg/dL	<200 mg/dL	Slightly elevated
HDL Cholesterol	44 mg/dL	>40 mg/dL	Borderline
LDL Cholesterol	134 mg/dL	<130 mg/dL	Mildly elevated
Triglycerides	168 mg/dL	<150 mg/dL	Mild elevation
Inflammatory/Autoimmune Markers			
Rheumatoid Factor (RF)	Positive (1:80)	Negative	Confirms RA
Anti-CCP Antibody	Positive	Negative	Confirms seropositive RA
ESR	36 mm/hr	<20 mm/hr	Elevated due to RA
CRP	12 mg/L	<5 mg/L	Mildly raised
Other Tests			
Serum Calcium	9.1 mg/dL	8.5–10.5 mg/dL	Normal
Vitamin D (25-OH)	26 ng/mL	20–50 ng/mL	Sufficient
Imaging Studies			
Ultrasound Abdomen	Normal kidneys, liver, pancreas	—	Excluded structural pathology
X-Ray Hands	Erosions in MCP joints	—	Consistent with RA

Diagnosis

The clinical presentation of the patient, history of a long-term use of corticosteroids for rheumatoid arthritis, and laboratory results consisting of raised fasting blood glucose, postprandial blood glucose, and HbA1c all led to the conclusion of steroid-induced diabetes mellitus (SIDM). Normal insulin, C-peptide, and cortisol levels at fasting ruled out both primary diabetes and Cushing's syndrome. The lack of previous hyperglycemia and the connection between steroid treatment and increased glucose levels further pointed to this diagnosis. The patient was diagnosed with seropositive rheumatoid arthritis due to the positive rheumatoid factor and anti-CCP antibodies, and the elevated ESR and CRP showed that the inflammation was still active.

Treatment and Management

The management of the patient aimed at the favorable glycemic control, and at the same time, the rheumatoid arthritis (RA) was kept in remission. Her corticosteroid therapy was assessed on the day of the diagnosis. She had been on prednisolone 10 mg/day, which was slowly reduced to 7.5 mg/day under a rheumatologist's care with the view to avoiding further steroid-induced metabolic consequences while not provoking an RA flare. At the same time, metformin 500 mg was given once daily after breakfast as the primary treatment to enhance insulin sensitivity and lower hepatic glucose output.

The patient was given dietary counseling, focusing on a low-carbohydrate, high-fiber diet that would consist of whole grains, leafy vegetables, and lean proteins. The patient was told not to take refined sugars or processed foods. Besides, it was recommended to have small and frequent meals to avoid glucose spikes caused by steroids after meals. The lifestyle changes were supported, which consisted of brisk walking for 30 minutes a day, drinking enough water (2–2.5 liters a day), and keeping the weight at a healthy level.

At the end of the first week, metformin was well tolerated by the patient and the dose was increased to 500 mg bid (twice a day). Regular monitoring of blood glucose levels was done, and daily fasting and post-lunch capillary readings were recorded. There were no reported episodes of hypoglycemia. Self-monitoring of blood glucose and recognizing warning signs of hyperglycemia were taught to the patient.

At the two-week follow-up, FBS and PPBS levels showed slight improvement, and the prednisolone dose was decreased to 5 mg/day. Because of the mild hyperglycemia (FBS 140 mg/dL, PPBS 210 mg/dL), the metformin dose was increased to 1000 mg bid. The patient's rheumatoid arthritis was under control with no increase in joint pain or stiffness and no signs of corticosteroid withdrawal.

By the end of the fourth week, laboratory values showed further improvement (FBS 120 mg/dL, PPBS 184 mg/dL, HbA1c 6.8%). The patient had been very compliant with her dietary and exercise regimen and reported feeling more energetic and less thirsty. Prednisolone was continued at 5 mg/day, and methotrexate (15 mg weekly) and hydroxychloroquine (200 mg twice daily) were administered for disease management.

At the eight-week follow-up, her glycemic profile became almost normal (FBS 108 mg/dL, PPBS 152 mg/dL, HbA1c 6.3%) and her joint symptoms were still under good control. Prednisolone was systematically reduced in dosage and cessation was planned for the following two weeks. Metformin therapy was maintained to support stabilization of glycemic control.

After twelve weeks, the patient had complete glucose level normalization (FBS 102 mg/dL, PPBS 146 mg/dL, HbA1c 6.1%) and stable rheumatoid arthritis symptoms treated with DMARDs only. The case illustrated the successful reversal of steroid-induced diabetes by means of gradual tapering of steroids, metformin initiation, and strict dietary and lifestyle rules without rheumatologic compromise.

DISCUSSION

Steroid-induced diabetes mellitus (SIDM) is one of the metabolic complications that arise from glucocorticoid administration and is frequently and sometimes under-recognized. Corticosteroids of which systemic agents like prednisolone are the most potent are responsible for influencing glucose homeostasis via multiple mechanisms. These drugs have an effect on the liver where they enhance gluconeogenesis, and at the same time are taking away the use of glucose by the peripheral tissues such as muscle and fat, thus the levels of insulin in the blood will not be effectively reduced. The drugs are also responsible for the promotion of lipolysis which hinders the action of insulin and the resultant increase in blood sugar level. The risk and intensity of SIDM are mostly determined by the dosage and duration of treatment, where higher doses and long exposure are linked to the maximum cases. Patients with factors predisposing them to the condition, for instance, the elderly, those that are overweight or in a state of prediabetes, are more likely to suffer from SIDM, but it can also happen in individuals, like in this case, that do not have any prior metabolic conditions.[11]

In the current case, the patient experienced hyperglycemic symptoms extensive urination, excessive thirst, tiredness, and slight weight loss after 8 months of chronic treatment with prednisolone for rheumatoid arthritis (RA). Initial tests such as fasting plasma glucose and HbA1c done before starting steroids were all normal, and diabetes or metabolic syndrome was not present in the family history, which made it less likely that the patient had pre-existing type 2 diabetes. The lab tests showed high fasting and glucose levels after meals, and HbA1c of 7.4% that confirmed the diagnosis of diabetes as new-onset. The presence of normal serum insulin and C-peptide levels indicated that β-cell function was not affected, which supported the diagnosis of SIDM rather than type 1 diabetes or LADA. In addition, the normal morning cortisol and the lack of Cushingoid features ruled out Cushing's syndrome as a secondary cause of

hyperglycemia. The timing between steroid treatment and hyperglycemia, along with partial improvement after tapering steroids, further supported the diagnosis.[12]

Management of SIDM is requiring a multi-pronged approach. The first step is to minimize steroid exposure to the lowest dose that still keeps the patient comfortable. In this specific situation, prednisolone was reduced from 10 mg/day to 5 mg/day under very close rheumatology supervision while avoiding RA flare-ups. Metformin, which is a pharmacologic agent that is used to lower blood sugar through its insulin-sensitizing effect which is less likely to cause hypoglycemia, was started and increased slowly to 1000 mg twice daily. Meanwhile, lifestyle changes were backed up that included a diet with low carbs, high fiber, regular exercise, and drinking enough water. This combination of measures resulted in a gradual improvement in the glycemic status of the patients. Their fasting plasma glucose, for instance, fell from 164 mg/dL to 102 mg/dL and HbA1c dropped to 6.1% over a period of 12 weeks. These results showed that SIDM is reversible if timely detected and managed with the right strategies.[13]

The case emphasizes the need for proactive monitoring in patients who take corticosteroids for a long time. According to the guidelines, glucose levels should be measured during fasting and postprandially at the beginning of the treatment, 1-3 months later, and then regularly, particularly in patients with autoimmune disorders that need long-term steroid therapy. The early identification and intervention can prevent severe complications like hyperosmolar hyperglycemic state, infections, and slow healing of wounds, and also reduce the risk of cardiovascular problems in the long run. Besides, educating the patient on self-monitoring, spotting hyperglycemia symptoms, and running lifestyle measures is crucial for control that lasts.[14]

The necessity of the multidisciplinary approach for maximum benefit is highlighted in this case. The cooperation among the specialists led to the successful clinical management of steroid tapering, antidiabetic therapy start and titration, and patient counseling. The literature indicates that SIDM is frequently not detected, especially in non-obese individuals or those without previous risk factors, thereby indicating that physicians must be more vigilant. Likewise, the case reports have stated that timely intervention with a combination of lifestyle changes, drug therapy, and steroid dose reduction can completely eliminate hyperglycemia, which was the case with this patient.

CONCLUSION

Steroid-induced diabetes mellitus is one of the serious complications of corticosteroid therapy that could be reversed if recognized early and treated properly. Even patients without prior metabolic risk factors are not spared. The case reviewed here shows that early recognition, prompt steroid tapering, metformin therapy, and adherence to lifestyle changes can successfully restore blood sugar levels while keeping rheumatoid arthritis under good control with disease-modifying antirheumatic drugs. Regular glucose monitoring in patients receiving long-term steroids is crucial in order to avoid both acute complications and long-term cardiovascular risks. A teamwork approach that involves rheumatologists, endocrinologists, and clinical pharmacists is necessary for wide-ranging patient care, education, and better treatment results.

REFERENCES

1. Guo Q, Wang Y, Xu D, Nossent J, Pavlos NJ, Xu J. Rheumatoid arthritis: pathological mechanisms and modern pharmacologic therapies. Bone Res, 2018; 6: 15.
2. Hwang JL, Weiss RE. Steroid-induced diabetes: a clinical and molecular approach to understanding and treatment. Diabetes Metab Res Rev, 2014 Feb; 30(2): 96-102.
3. Kuo T, McQueen A, Chen TC, Wang JC. Regulation of Glucose Homeostasis by Glucocorticoids. Adv Exp Med Biol, 2015; 872: 99-126.
4. Albakr RB. Steroid-Induced Hyperosmolar Hyperglycemic Syndrome in a Young Patient Without Diabetes After Treating Him for Minimal Change Disease-Case Report. Clin Case Rep, 2024 Nov 22; 12(11): e9599.
5. Joseph J. Beyond the Triad: Uncommon Initial Presentations in Newly Diagnosed Type 2 Diabetes Mellitus. Cureus, 2025 Aug 7; 17(8): e89525.
6. Tian Z, Mclaughlin J, Verma A, Chinoy H, Heald AH. The relationship between rheumatoid arthritis and diabetes mellitus: a systematic review and meta-analysis. Cardiovasc Endocrinol Metab, 2021 Feb 19; 10(2): 125-131.
7. Ito S, Ogishima H, Kondo Y, Sugihara M, Hayashi T, Chino Y, Goto D, Matsumoto I, Sumida T. Early diagnosis and treatment of steroid-induced diabetes mellitus in patients with rheumatoid arthritis and other connective tissue diseases. Mod Rheumatol, 2014 Jan; 24(1): 52-9.
8. Donihi AC, Raval D, Saul M, Korytkowski MT, DeVita MA. Prevalence and predictors of corticosteroid-related hyperglycemia in hospitalized patients. Endocr Pract, 2006; 12: 358–362.
9. Gurwitz JH, Bohn RL, Glynn RJ, Monane M, Mogun H, Avorn J. Glucocorticoids and the risk for initiation of hypoglycemic therapy. Arch Intern Med, 1994; 154: 97–101.
10. Papang R, John AS, Abraham S, Rao PS. A study of steroid-induced diabetes mellitus in leprosy. Indian J Lepr, 2009; 81: 125–129.
11. Suh S, Park MK. Glucocorticoid-Induced Diabetes Mellitus: An Important but Overlooked Problem. Endocrinol Metab (Seoul), 2017 Jun; 32(2): 180-189.
12. van der Pol JA, Allaart CF, Lems W, van der Kooij SM, Huizinga TWJ, Bergstra SA. Prednisone use, disease activity and the occurrence of hyperglycaemia and diabetes in patients with early rheumatoid arthritis: a 10-year subanalysis of the BeSt study. RMD Open, 2024 Apr 30; 10(2): e004246.
13. Aberer F, Hochfellner DA, Sourij H, Mader JK. A Practical Guide for the Management of Steroid Induced Hyperglycaemia in the Hospital. J Clin Med, 2021 May 16; 10(10): 2154.
14. Jain AB, Lai V. Medication-Induced Hyperglycemia and Diabetes Mellitus: A Review of Current Literature and Practical Management Strategies. Diabetes Ther, 2024 Sep; 15(9): 2001-2025.