CASE SERIES

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Hirata Syndrome Clinical Presentation and Management: A Single-Centre Experience

Gurusangappa Mudagall, Uma Kaimal Saikia, Ashok Krishna Bhuyan, Abhamoni Baro

Department of Endocrinology, Gauhati Medical College and Hospital, Guwahati, Assam, India

Abstract

Background. Hirata syndrome, or Insulin Autoimmune Syndrome (IAS), is a rare condition characterized by hyperinsulinemic hypoglycemia due to autoantibodies targeting endogenous insulin. Its rarity and unique presentation make diagnosis and management challenging.

Objective. To analyze the clinical presentation, diagnostic methods, and management of IAS based on a single-center experience

Methodology. This was a case series, conducted at a tertiary care center in northeast India, involving six cases diagnosed between January 2022 and April 2024. Clinical histories, including drug use and comorbidities, were reviewed. Laboratory analyses during hypoglycemic episodes assessed insulin, C-peptide, β-hydroxybutyrate, and cortisol levels. Extended OGTT and 72-hour fasting tests were used when spontaneous hypoglycemia was absent. Insulin autoantibodies (IAA) confirmed the diagnosis.

Results. Six IAS cases were identified, with most linked to α-lipoic acid in multivitamins and one to methimazole. Patients experienced recurrent postprandial hypoglycemia. Elevated insulin, C-peptide, and IAA levels confirmed the diagnosis. Management included discontinuation of causative drugs, dietary changes with frequent complex carbohydrate meals, and pharmacotherapy (acarbose or prednisolone). Follow-up showed resolution of hypoglycemia and normalization of IAA levels in all patients.

Conclusion. IAS should be considered in patients with hypoglycemia and a history of sulfhydryl-containing medications. Early diagnosis using IAA measurement and appropriate management, including drug discontinuation, dietary adjustments, and pharmacotherapy, is essential. Further research is needed to refine treatment strategies and understand the pathogenesis of this rare syndrome.

Key words: Hirata syndrome, insulin autoantibody syndrome, insulin autoantibody, hypoglycemia

INTRODUCTION

Hirata syndrome, also known as Insulin Autoimmune Syndrome (IAS), presents a unique challenge in Endocrinology. First identified by Yukimasa Hirata and colleagues in Japan in 1970, this rare condition manifests as hyperinsulinemic hypoglycemia due to autoantibodies targeting endogenous insulin. The cornerstone of IAS diagnosis is the presence of circulating insulin autoantibodies (IAA), which play a pivotal role in its pathogenesis.[1] Symptoms and signs of hypoglycaemia are nonspecific and are classified into neurogenic and neuroglycopenic symptoms. Neurogenic (autonomic) symptoms include adrenergic (palpitations, tremors and anxiety) and cholinergic (diaphoresis, hunger and paresthesias) symptoms. Neuroglycopenic symptoms are due to glucose deprivation in brain, these symptoms include cognitive impairment, psychomotor abnormalities and in severe cases seizures, and coma.[2]

IAS primarily arises as a result of medication-induced autoimmune responses, with methimazole being the most common trigger. Other implicated medications include captopril, d-penicillamine, propranolol, and α-lipoic acid. Furthermore, genetic predisposition, particularly HLA associations such as HLA-DRB1 *0406, DQB1 *0302, DQA1 *0301, DRB1 *0415, and DRB1 *13:01, have been observed in the majority of IAS cases. This condition can coexist with other autoimmune disorders such as Graves' disease, systemic lupus erythematosus (SLE), rheumatoid

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Corresponding author: Abhamoni Baro, MD, DM (Endocrinology)
Department of Endocrinology,Gauhati Medical College and Hospital Narakasur Hilltop, GMC Hospital Rd, Bhangagarh, Guwahati, Assam 781032, India
E-mail: drabhamonibaro@gmail.com
ORCiD: https://orcid.org/0000-0001-7989-3777

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arthritis, and chronic hepatitis. Additionally, less common associations include ankylosing spondylitis, anti-neutrophil cytoplasmic antibody-associated glomerulonephritis, polymyositis, systemic sclerosis, psoriasis, multiple myeloma, and monoclonal gammopathy.[3] IAS are usually self-limiting once the triggering medication is stopped but immunesuppressants may be required in some.[4]

METHODOLOGY

This case series was conducted in the Department of Endocrinology at a tertiary care center in northeast India (Gauhati Medical College and Hospital, Guwahati, Assam, India). Ethical clearance for the study was obtained from the Institutional Ethics Committee (Approval No. MC No-190/2007/pt-II/oct-2024/7). Our study was conducted over a period of 28 months, from January 2022 to April 2024, covering all cases diagnosed and treated during this period. Our study employed a consecutive sampling approach and all patients presenting with hypoglycemia at the Endocrinology Department of our center between January 2022 and April 2024 were screened. Eligible patients meeting the inclusion criteria were enrolled in the study, ensuring no selection bias.

Inclusion criteria

Patients diagnosed with Hirata syndrome (Insulin Autoimmune Syndrome) based on clinical presentation of recurrent hypoglycemia (blood glucose <55 mg/dl) with elevated insulin autoantibody (IAA) levels confirmed by laboratory testing after excluding other common causes of hypoglycemia, such as insulinoma or exogenous insulin administration.

Exclusion criteria

1. Patients with hypoglycemia secondary to non-autoimmune causes, including insulinoma, critical illness, or malnutrition.
2. Individuals with incomplete diagnostic workup or insufficient data for analysis.
3. Patients who refused or withdrew consent during the study period.

A detailed history of the clinical features, drug intake, and associated comorbidities were assessed. At the height of hypoglycemia (Blood glucose <55 mg/dl), critical samples were collected and sent for analyzing serum insulin, serum C-peptide, plasma β-hydroxybutyrate and serum cortisol. If spontaneous hypoglycemia was not present, then a 72-hour fast test and Extended OGTT tests were done (up to 6 hours after 75 gm OGTT) to induce hypoglycemia. The samples were analyzed by Electrochemiluminescent immunoassay (ECLIA) by Roche Cobas e411 analyzer. Serum levels of insulin >3μIU/ml, C-peptide >0.6 ng/ml, and IAA >10 IU/ml were used as a cut-off for the diagnosis of hyperinsulinemic hypoglycaemia secondary to insulin autoimmune syndrome. A written informed consent was taken from all of the cases. The total number of cases was 6 considering the rarity of the disorder; the descriptive observational data are presented below.

The observations obtained through clinical evaluation, laboratory investigations, and diagnostic procedures are presented descriptively in the Results section. Each case is detailed individually to highlight variations in clinical presentation, diagnostic findings, treatment approaches, and patient outcomes. This case-wise presentation format ensures that the methods employed for diagnosis and management are directly linked to the reported outcomes, offering a clear understanding of how each case was assessed and treated based on the outlined methodology.

RESULTS

During the study period, out of the 25 patients presenting with recurrent hypoglycemia, only 6 were diagnosed with Hirata syndrome. The details of the individual patients are as follows:

Case 1

A 48-year-old male presented with recurrent hypoglycemic episodes for 2 months occurring both in the fasting state as well as four to five hours after a major meal. This man complained of palpitation, tremors, and one episode of altered sensorium requiring hospitalization and intravenous (IV) dextrose therapy. The blood glucose measured during the episodes varied between 27-60 mg/dl. The critical sampling taken at a blood glucose (BG) level of 40 mg/dl revealed serum insulin >1000 μIU/ml, serum C-peptide 32.3 ng/ml, serum cortisol 5.4 μg/dl, serum GH 0.7 ng/ml and absent ketone bodies. The Computed tomography (CT) scan imaging of the abdomen was normal and since suspicion was high, insulin autoantibodies were sent which were more than four times elevated (85.3 IU/ml, normal <18 IU/ml). A diagnosis of Hirata syndrome was made based on the high insulin antibody levels and history of taking α–lipoic acid for a long duration. The patient was started on 2-hourly frequent complex carbohydrate meals along with acarbose (25 mg three times daily with meals) and α-lipoic acid was stopped. The patient did not develop any further episodes of hypoglycemia after the initiation of treatment. On follow-up after 3 months, the insulin antibodies were reassessed and were found to be negative. Acarbose was stopped and there had been no further hypoglycemia to date.

Case 2

A 38-year-old female presented with recurrent hypoglycemia for 1 month, which was typically post-prandial and occurring usually 3-4 hours after taking food. On evaluation, the critical sampling at a blood glucose level of 29 mg/dl revealed serum insulin >1000 μIU/ml, serum C-peptide 23.96 ng/ml, serum cortisol 11.8 μg/dl, and

negative urine ketones. The patient recovered after IV dextrose. A diagnosis of hyperinsulinemic hypoglycemia was made and an MRI (Magnetic resonance imaging) of the pancreas was done which was not suggestive of any tumor. Then anti-insulin autoantibodies were sent and were found to be 10 times elevated (188 IU/ml). Later the patient revealed a history of taking multivitamin tablets for a long duration which contained α-lipoic acid. The patient was started on a frequent complex carbohydrate diet and prednisolone (40 mg/day), which was tapered over 1 month. The patient responded well with no further hypoglycemic episodes even after stopping therapy.

Case 3

A 42-year-old female presented with multiple episodes of hypoglycemia for 5 months, which usually occurred 4-5 hrs after having food and relieved by taking food. The patient underwent an extended oral glucose tolerance test which resulted in hypoglycemia after 4 hours of taking 75 grams of glucose. Critical sampling at BG 20 mg/dl was done and the results showed serum insulin 600 μIU/ml, serum C-peptide 5.5 ng/ml, and serum cortisol 19.6 μg/dl. The CT scan of the abdomen was normal and insulin autoantibody levels were high (78.7 IU/ml). This patient also revealed a history of taking multivitamins containing α-lipoic acid. The patient was started on a complex carbohydrate diet with acarbose (25 mg three times daily with meals) and prednisolone (40 mg/day).

Case 4

A 60-year-old female presented with multiple hypoglycemic episodes for 20 days. The patient had a history of kidney stones for which she received multiple medications along with multivitamins containing α-lipoic acid. Hypoglycemic episodes were mainly post-prandial usually occurring after 2-3 hours after having food. The patient underwent a 72-hour fast test during which the patient did not have hypoglycemia, hence an extended OGTT was done during which the patient developed hypoglycemia after 3.5 hours. During hypoglycemia (BG 20 mg/dl) the serum insulin was >1000 μIU/ml, C-peptide 23.02 ng/ml, cortisol 20 μg/dl and GH 11.54 ng/ml. CT abdomen was normal whereas the anti-insulin autoantibody level was high (98 IU/ml). The patient was started on acarbose (25 mg three times daily with meals) with frequent complex carbohydrate-containing meals. The patient was discharged after 5 days. After 6 months of follow-up patient's antibody levels became negative and the patient did not have any more hypoglycemic episodes even after stopping acarbose.

Case 5

A 64-year-old non-diabetic male presented with recurrent hypoglycemia (lowest BG 34 mg/dl) for 1 month. Whipple's triad was documented when the case was admitted to the hospital. He was hypertensive and was taking telmisartan, amlodipine, and chlorthalidone. All hypoglycemia episodes usually occur after 4-5 hours of taking a carbohydrate meal. He was also taking a multivitamin tablet for 2 months which contained α-lipoic acid. His critical sampling during hypoglycemia (BG 34 mg/dl) revealed adequate cortisol response (20.61 μg/dl) and very high insulin (>1000 μIU/ml) and C-peptide (23.27 ng/ml) levels. His CT abdomen was normal except for a small mesenteric lipoma. The anti-insulin autoantibody levels were high (93 IU/ml: normal range <18 IU/ml). The multivitamin tablet was stopped and he was started on acarbose (25 mg three times daily with meals) with a frequent complex carbohydrate-containing diet. The patient responded after the first dose without any further hypoglycemia episodes. After 3 months, during the follow-up, the patient's insulin autoantibody disappeared and acarbose was tapered off. There was no episode of hypoglycemia thereafter to date.

Case 6

A 60-year-old female presented with recurrent postprandial hypoglycemia for the last 1 month, 4 to 5 episodes in a week. She gave a history of thyroid disease and intake of antithyroid medication (methimazole 10 mg once daily) for the last 2 months, which she had stopped 1 week before presentation. The critical sampling during hypoglycemia (BG 42 mg/dl) revealed high serum insulin (>1000 μIU/ml), high C-peptide (12.35 ng/ml), and serum cortisol of 28.20 μg/dl with absent ketosis. Her thyroid function test was within normal limits (TSH 3.8 mIU/L) and a negative anti-TSH receptor antibody which ruled out Graves' disease. Her abdominal and thyroid imaging were normal. On further evaluation, her insulin autoantibody levels were found to be very high (68 IU/ml). A diagnosis of Hirata syndrome was made with methimazole being the possible culprit. This patient was treated with acarbose (25 mg three times daily) and she responded well without further episodes of hypoglycemia. The acarbose treatment was continued for 3 months and when her insulin antibody levels became negative, acarbose was tapered and stopped.

DISCUSSION

IAS is an autoimmune condition without any gender predilection. The mean age of presentation varies across different studies, ranging from 8 to 62 years. In our series, patients belonged to the age group of 42 to 64 years. Hypoglycemic episodes predominantly occur during the post-absorptive phase, although instances of fasting and exercise-induced hypoglycemia have also been reported. The underlying mechanism of hypoglycemia in IAS is attributed to the presence of substantial levels of insulin autoantibodies (IAA). When a person eats a meal, there is a surge in blood glucose levels which subsequently leads to increased insulin secretion from the pancreatic beta cells. However, when IAA is present, it binds to the insulin molecules and reduces the efficacy of insulin, resulting in postprandial hyperglycemia. This, in turn, stimulates heightened production of insulin and C-peptide to counteract the hyperglycemic state. The complexes formed between

Table 1. Summary of all six patients in tabular form

S.N	Age (Years)	Sex	Timing of hypoglycemia	Triggering drug	Insulin (µIU/ml)	C-peptide (ng/ml)	IAA (IU/ml)	Treatment
1	48	M	Fasting and postprandial	α lipoic acid	>1000	32.3	85.3	Acarbose
2	38	F	Fasting	α lipoic acid	>1000	23.96	188.0	Prednisolone
3	42	F	Fasting	α lipoic acid	>600	5.5	78.7	Acarbose and Prednisolone
4	60	F	Fasting	α lipoic acid	>1000	23.02	98.0	Acarbose
5	64	M	Fasting	α lipoic acid	>1000	23.27	93.0	Acarbose
6	42	F	Fasting and postprandial	Carbimazole	>1000	12.35	68.0	Acarbose

IAA – Insulin autoantibody

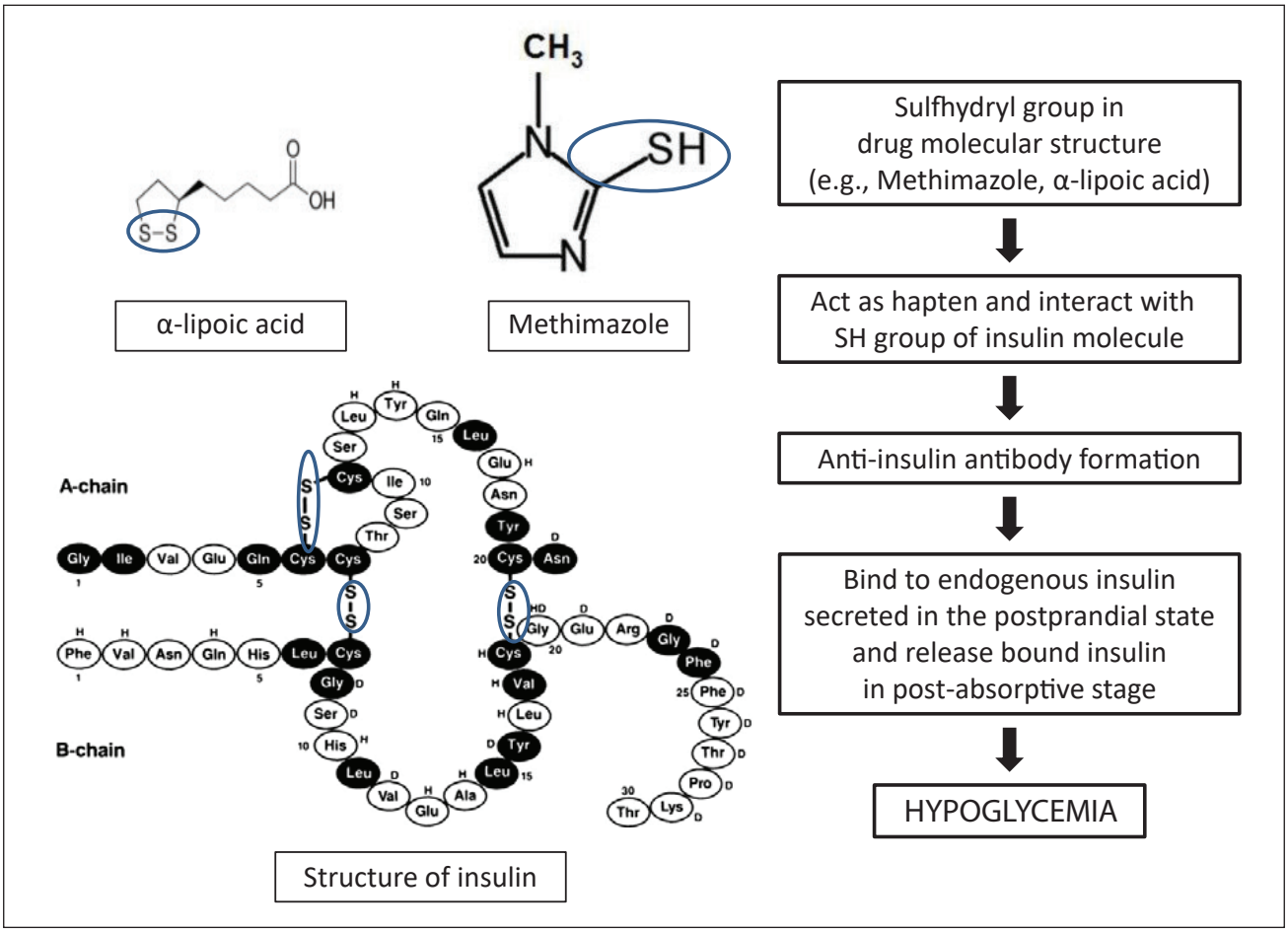

Figure 1. Structure of insulin, methimazole and α-lipoic acid with their sulfhydryl groups.[4,5]

insulin and IAA serve as a reservoir of insulin which upon dissociation at a later post-absorptive phase act as a source of sustained free insulin release leading to prolonged and severe hypoglycemia. Any drug that contains a sulfhydryl group in its molecular structure can act as a hapten because of a similar sulfhydryl group in the insulin chains. These drugs can induce immunogenicity by interacting with the disulfide bonds in insulin and lead to formation of antibodies (Figure 1) Patients usually presented after 4-6 weeks of initiating the offending drug which was similar to other case series.[4,6] In this study, the offending drug other than methimazole causing IAS was *α*-lipoic acid which is usually present in different types of multivitamins and health supplements. This study highlights that prolonged inadvertent use of such medications can lead to a serious side effect like IAS and the use of such supplements without any indication should be avoided. Moreover, this study also emphasizes that IAS should be included in the differential diagnosis in evaluating a case of hypoglycemia. IAS can be differentiated from insulinoma by the insulin levels which are usually very high in IAS (above 1000 µIU/ml) compared to insulinoma (below 1000 µIU/ml). Hiya et al. published a case series on Hirata syndrome, reporting that insulin levels exceeded 1000 µIU/ml in the two cases studied, with the insulin-to-C-peptide molar ratio also being greater than 1.[4] Sehgal et al., published a case of Hirata syndrome which had similar high insulin levels.[7] Hirata Syndrome can also be confused with Type B Insulin Resistance (TBIR), another rare autoimmune disorder causing hyperinsulinemia. However, while Hirata Syndrome is caused by autoantibodies against insulin, TBIR results from autoantibodies targeting insulin receptors, leading to

extreme insulin resistance. TBIR is strongly associated with SLE, Sjogren's, or systemic sclerosis and usually presents with persistent hyperglycemia with occasional paradoxical hypoglycemia. TBIR usually requires aggressive immunosuppression (steroids, cyclophosphamide or rituximab).

The assessment of insulin autoantibody titers is imperative for diagnosing IAS. Nevertheless, a prevailing limitation in many commercially-available assays is their capability to solely detect the immunoglobulin-G class of insulin autoantibodies. The molar ratio of insulin to C-peptide serves as a diagnostic marker for IAS, with values exceeding 1 indicating either IAS or exogenous insulin usage, where C-peptide is suppressed. Most instances of IAS exhibit a self-limiting course, with resolution of symptoms occurring within 3–6 months of initial diagnosis.[8]

In our study, our patients were treated with small frequent meals, acarbose, and steroids and they have proven to be effective as the disease is self-limiting. Ashraf et al. published a case report in which they found frequent small feeds and steroids to be effective in preventing hypoglycemia.[9] Censi et al., found frequent small meals to be effective in preventing hypoglycemia in patients with Hirata syndrome.[10]

Continuous glucose monitoring (CGM) and flash glucose monitoring (FGM) provide real-time glucose tracking, enabling early hypoglycemia detection, trend analysis, and pattern recognition. These tools help prevent severe episodes, guide dietary and pharmacologic adjustments (e.g., acarbose, steroids), and improve patient quality of life by reducing the need for frequent fingerstick testing. Their integration into Hirata syndrome management enhances safety and optimizes treatment strategies. However, in our series, CGM could not be utilized due to cost constraints, though its use would have significantly improved management.

For patients experiencing intractable hypoglycemia, initial treatment involves consuming small, frequent meals low in carbohydrates to mitigate postprandial hyperglycemia and subsequent insulin spikes. Short courses of corticosteroids, such as oral prednisolone (30–60 mg), may be employed as adjunct therapy. Additional therapeutic options encompass acarbose (to diminish carbohydrate absorption), diazoxide, octreotide, partial pancreatectomy (to restrict insulin release), and plasmapheresis (to reduce insulin autoantibody titers). Immunosuppressants like azathioprine, cyclophosphamide, and mycophenolate mofetil have also been explored in IAS management. In refractory cases, rituximab, an anti-CD20 monoclonal antibody, has demonstrated efficacy, particularly when steroid therapy proves ineffective.[3]

CONCLUSION

Our study highlights the diverse clinical presentations and diagnostic challenges encountered in patients with Hirata syndrome. Despite its rarity, clinicians should maintain a high index of suspicion for IAS, especially in individuals presenting with recurrent hypoglycemia associated with autoimmune disorders or a history of sulfhydryl-containing medication use. Prompt diagnosis, through measurement of insulin autoantibodies and comprehensive evaluation is crucial for initiating appropriate management strategies, including dietary modifications, pharmacotherapy, and addressing underlying autoimmune conditions. Further research is warranted to elucidate the underlying mechanisms and optimize therapeutic approaches for this rare but clinically significant syndrome.

Statement of Authorship
All authors certified fulfillment of ICMJE authorship criteria.

CRediT Author Statement
GM: Conceptualization, Methodology, Software, Validation, Formal analysis, Investigation, Resources, Data Curation, Writing – original draft preparation, Writing – review and editing, Visualization, Supervision, Project administration, Funding acquisition; **UKS:** Conceptualization, Methodology, Software, Validation, Formal analysis, Investigation, Resources, Data Curation, Writing – original draft preparation, Writing – review and editing, Visualization, Supervision, Project administration, Funding acquisition; **AKB:** Conceptualization, Methodology, Software, Validation, Formal analysis, Investigation, Resources, Data Curation, Writing – original draft preparation, Writing – review and editing, Visualization, Supervision, Project administration, Funding acquisition; **AB:** Conceptualization, Methodology, Software, Validation, Formal analysis, Investigation, Resources, Data Curation, Writing – original draft preparation, Writing – review and editing, Visualization, Supervision, Project administration, Funding acquisition.

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