Ghana Alternative Medicine Journal (GAMJ)
Abbreviated Key Title: Gha alt Med Jrnl
ISSN 2756-7176 (Print) Open Access
Journal homepage: https://saspublishers.com/journal/gamj/home

OPEN ACCESS

A Type IIIc Autoimmune Polyendocrine Syndrome Associated with Metabolic Syndrome, a Rare Association: Report of a Case

Ibrahima Amadou Dembele[1*], Stéphane Loique Djeugoué[1, 3], Sékou Landouré[1], Mamadou Cissoko[1], Adama Sinayoko[1], Oumou Dembélé[1], Max Kouadio Djé[1], Kévine V Gapaya Dongmo[1], Prudence Adoun[1], Mamga T Célia Reine[1], Gueye Jered Brhaud[1], Yacouba Koné[1], Dodji K Mensah-Zukong[1], Assitan Soumaré[1], Aoua Diarra[1], Nouhoum Koné[1], Samba Camara[1], Moussa Sangaré[1], Romuald Nounga Nyanké[1], Mamadou Mallé[1], Kaly Keïta[1], Djibril Sy[1, 2], Traoré Djénébou[1, 2], Abrar-Ahmad Zulfiqar[4], Didier Mukeba Tshialala[3], Soukho Assétou Kaya[1, 2]

[1]Internal Medicine Department at the University Hospital Center Point G, Bamako, Mali
[2]Faculty of Medicine and Odontostomatology – University of Sciences, Techniques and Technologies of Bamako, Bamako, Mali
[3]Faculty of Medicine, Pharmacy and Public Health - University of Mbuji-Mayi, Mbuji-Mayi, Democratic Republic of Congo
[4]Department of Internal Medicine at the University Hospital Center of Strasbourg, Strasbourg, France

DOI: 10.36347/gamj.2024.v05i03.001 | **Received:** 28.05.2024 | **Accepted:** 01.07.2024 | **Published:** 11.07.2024

***Corresponding author:** Ibrahima Amadou Dembele
Internal Medicine Department at the University Hospital Center Point G, Bamako, Mali

Abstract **Case Report**

Introduction: Autoimmune polyendocrine syndrome (APS) designate the appearance of two or more autoimmune endocrine disorders, associated or not with other non-endocrine autoimmune diseases. ***Observation:*** We describe a case of polyendocrine syndrome type IIIc associating Hashimoto's disease-viligo vulgaris and alopecia areata in a 64-year-old patient with metabolic syndrome, discovered following a myxedematous coma in the initial phase and whose The development was favorable. The article presents the therapeutic particularities specific to this case in our context and also examines the existing literature on the diagnosis and treatment of this condition. ***Conclusion:*** APS type IIIc is a rare polyendocrine picture whose clinical presentation can be severe.

Keywords: Autoimmune polyendocrine syndrome, Autoimmune disorder, Internal medicine.

INTRODUCTION

Autoimmune polyendocrine syndromes (APS) or autoimmune polyendocrinopathies, also called polyglandular autoimmune syndromes, are a heterogeneous group of hereditary diseases characterized by at least 2 endocrine deficiencies linked to an autoimmune mechanism, often associated to other non-endocrine autoimmune diseases (vitiligo, alopecia, celiac disease, Biermer's disease, rheumatoid arthritis, etc.) [1]. APS are syndromic groups which are of obvious medical interest both theoretically and clinically [2]. In addition, they impose on the clinician screening strategies and specific management of patients who suffer from them [2]. Hashimoto's disease is accompanied by a high frequency of autoimmune diseases [2]. APS type 3 is defined by the association of autoimmune dysthyroidism with type 1 diabetes, celiac disease, vitiligo or other autoimmune disease, in the absence of cortico-adrenal damage [3]. It represents the rarest form of associations of autoimmune diseases specific to endocrine organs in adults [3]. The prognosis can be unfavorable for severe forms [4]. Very few reported cases have been published, particularly in the sub-Saharan region. We thus describe the case of a patient suffering from a type IIIc polyendocrine syndrome associated with a metabolic syndrome.

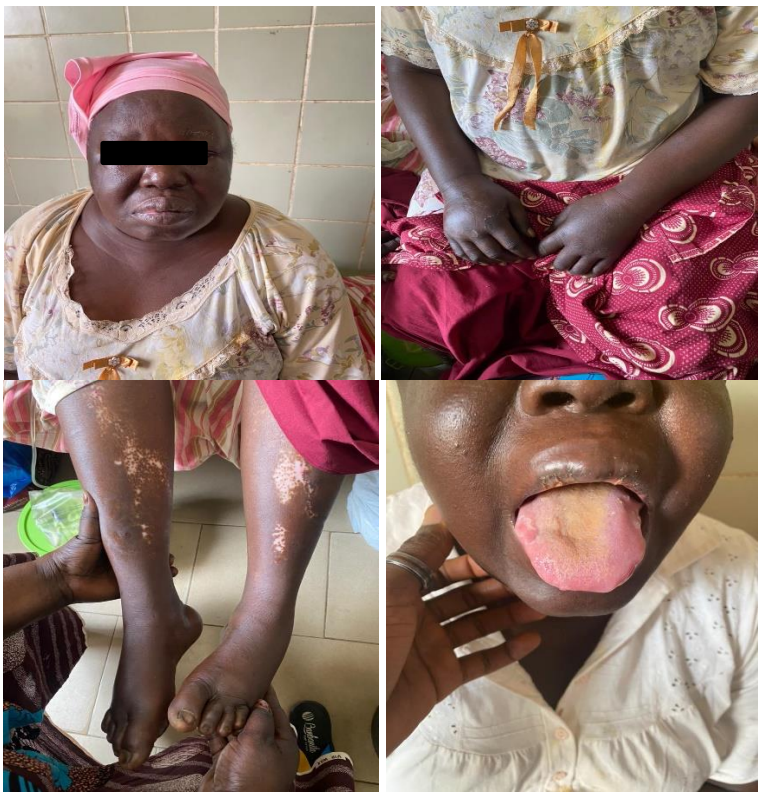
CASE DESCRIPTION

We report the case of a 64-year-old patient of Malian nationality; household. She was seen on February 21, 2024 in the Internal Medicine department at the University Hospital Center Point G in Bamako for non-febrile confusion.

The start of the symptoms reported by the family dates back around a week, marked by the progressive installation of disorders of consciousness such as incoherent comments made of jargonaphasia, without notion of trauma, associated with a psychomotor slowdown, with fluctuation of the disorders and evening-

night accentuation, insomnia when falling asleep; episodes of deep daytime sleepiness; without notion of headaches, ringing in the ears and tinnitus; without notion of loss of consciousness, nor fall from height. A cough with whitish and viscous expectoration. All evolving in a context of uncharacterized physical asthenia, without notion of anorexia and associated with a notion of intermittent unquantified fever, without nocturnal hyper sweating. In addition to the history, she also complained of constipation associated with abdominal bloating with non-nauseating flatulence, heating of the feet, tingling and a painful burning sensation in both lower limbs. No therapeutic attitude was taken; Faced with the persistence of the symptoms, the family decided to consult the Emergency department of the University Hospital Center Point G, which referred them to us for treatment. As antecedents; she has been diabetic for around 11 years, hypertensive for 7 years under irregular care; she has had hypothyroidism for 5 years under irregular care, an undocumented atypical ulcer syndrome. She is the 6th child, 6th parent, 3 living children and 3 deceased children; fetal macrosomia. She would have a diabetic aunt, a hypertensive sister and a diabetic son. She would have taken pantoprazole 40mg per day, amlodipine + valsartan (10/160mg) 1 tablet per day, levothyoroxine 160µg per day, metformin 850mg per day; calcium combined with vitamin D (dosage not documented).

The general condition examination revealed a conscious, bedridden, obese patient; athletic and afebrile to the touch; his Karnofsky index was 60%. Lying blood pressure in the left arm at 123/65 mmHg; a heart rate of 102 bpm; a respiratory rate of 24 cycles per minute; a left axillary temperature of 36.9°C; capillary blood glucose at entry at 0.87g/L. His measurements at entry were, a waist size of 114cm, a weight of 110kg, and height of 160cm for a body mass index of 42.97kg /m². The neurological examination revealed a conscious patient, drowsy with snoring, a Glasgow score of 12/15, disoriented in time and space, superficial sensitivity preserved, muscular strength in the pelvic limbs rated at 4/5 according to the Medical Research Council, no axial stiffness. The remainder of the physical examination noted, at the level of the scalp, small circular areas of well-circumscribed alopecia in patches and small "exclamation point" hairs on the periphery; palmar-plantar conjunctival pallor, fading of the tail of the eyebrows, macroglossia, diffuse myxedema (face – wrists and forearms – feet) ; bilateral crackles in the lower 1/3 of both hemithoraxes; cutaneous xerosis with scratching lesions, hypochromic macules, on the anterior surface of both legs in the region of the tibial crests, asymmetrical distribution, confluent in places, with very distinct irregular contours highlighted by a hyperpigmented border (Picture 1), without itching or pain. Hemorrhoidal marks and cloudy urine in the collection bag of the urinary catheter; whitish and foul-smelling leucorrhoea. The other devices were carefully examined and found no anomalies.



Picture 1: Photographs of the patient – Day 3 (Dr Ibrahima A Dembélé – Dr Stéphane L Djeugoué)

The paraclinical assessment showed:

- Hemogram: hypochromic macrocytic anemia with a hemoglobin level of 8.4 g/dl, the mean corpuscular volume of 77.3fl; a CCMH at 37.8 g/dl. Erythropenia at 2.870.000/mm3
- Inflammatory assessment: an inflammatory syndrome with a CRP of 100 mg/l and a ferritin level of 1000 ng/mL
- Endocrine assessment: TSHus at 13.08µui/mL, FT4 at 12.35pmol/L, baseline cortisol levels were normal; HbA1c at 8.66%.
- Infectious assessment: negative HbsAg, anti-Hbc Ab not done, anti-Hbs Ab not done, HIV1-2 and HCV serologies are negative. The cytobacteriological examination of urine and sputum was sterile, the cytobacteriological examination of vaginal secretions isolated Klebsiella pneumoniae sensitive to ciprofloxacin.
- Biochemical assessment: Serum creatinine at 118 µmol/l with clearance at 48.62 ml/min. Azotemia at 12.68 mmol/L. Hypoosmolar hyponatremia (Na+ =100mEq/L and plasma osmolarity = 234mOsm), hypochloremia at 95.3 mEq/L; and hypocalcemia (corrected) at 1.84 mmol/L. Albuminemia at 38.6g/L, 24-hour proteinuria at 0.229g/24h; transaminases are normal and LDL cholesterol at 1.10g/L
- Immunological assessment: anti-GAD antibodies at 5,49iu/mL (negative), anti-TPO antibodies are positive at 9.41 iu/mL; anti-nuclear antibodies (ANA-Screen) at 0,1093 iu/mL (negative); negative anti-Sm antibodies. Anti-transglutaminase antibodies were requested but not performed
- Morphological assessment: Cervical ultrasound reveals a thyroid gland without detectable ultrasound nodule, of normal volume and echogenicity in favor of a hypothyroid state. The x-ray of the spine revealed a radiographic appearance in favor of moderate lumbar osteoarthritis. The cardiac ultrasound was normal. Abdominopelvic ultrasound revealed a liver of normal size, hyperechoic, homogeneous in relation to a steatotic liver; a gas screen of the colonic frame without functional anomaly related to functional colonopathy.
- Functional assessment: the electrocardiogram was normal, with a regular sinus rhythm at 100bpm

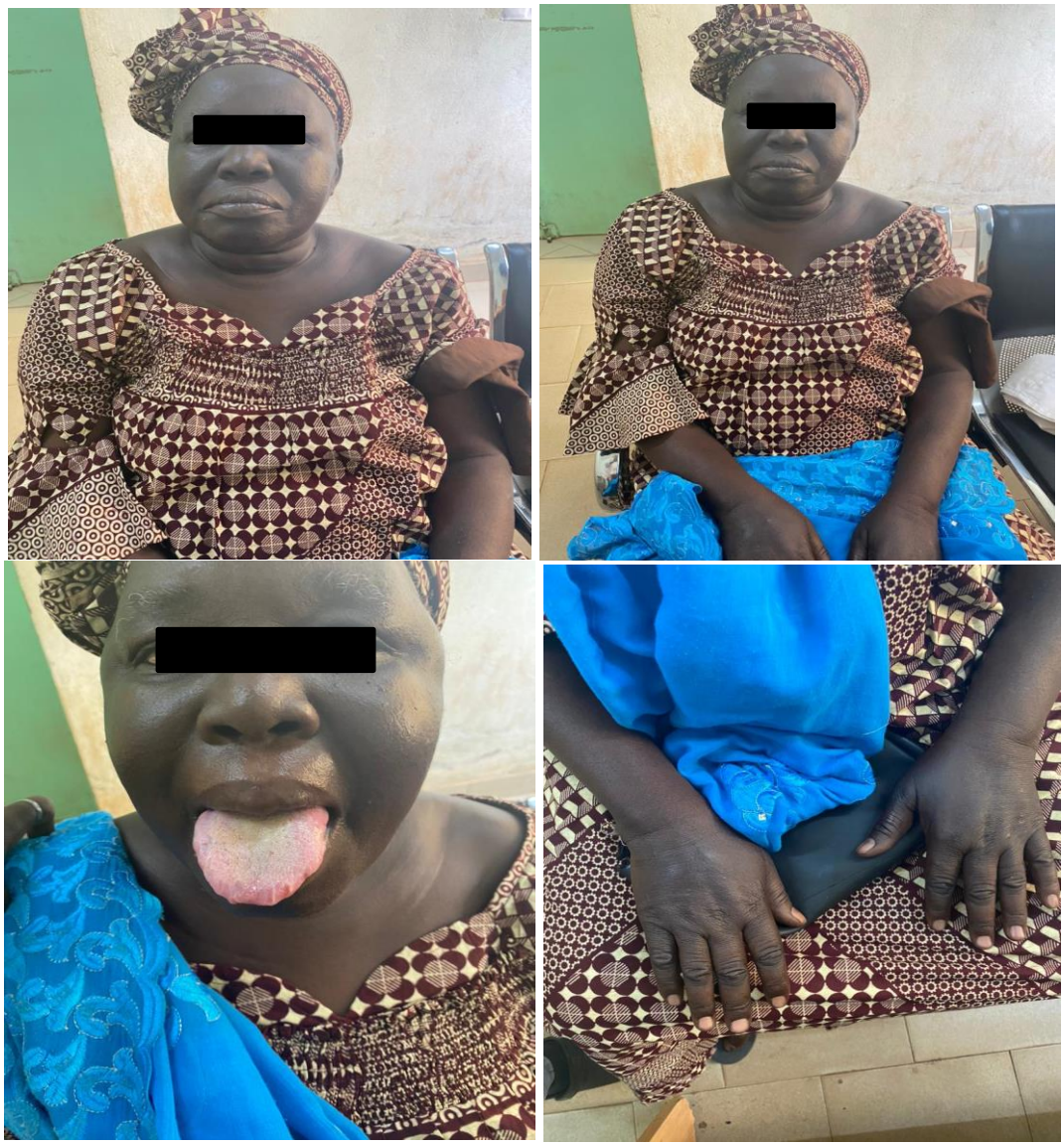
In view of all the clinical and paraclinical elements, we retained the diagnoses of Hashimoto's disease complicated by myxedematous coma in the initial phase following therapeutic non-compliance, vitiligo vulgaris and alopecia areata constituting a type IIIc polyendocrine syndrome on a field of metabolic syndrome; associated with *Klebsiella pneumoniae* vaginosis and moderate lumbar arthritis.

Initial treatment with injectable Levothyoroxine 200mg, one vial slowly intravenously every 8 hours for 3 days, then 100µg tab at a dose of 1.6 µg/kg or 176 µg/day (1 tab + 1/2 tab per day) . Pregabalin 75mg per day; rosuvastatin 10 mg per day, rapid insulin 12 iu every 8 hours, calciparin 5000 iu every 12 hours; 2g NaCl every 12 hours in 200 ml of 0.9% saline to flow for 8 hours until the serum sodium level is greater than 120mEq/L then stop. White vaseline on the skin, one application every 12 hours after washing, chondroitin sulfate 500mg capsule, one capsule every 12 hours. Amlodipine + valsartan (10/160 mg) 1 tab per day associated with a low-sodium diet. Ciprofloxacin 500mg every 12 hours for 10 days. Macrogol 4000, one sachet per day in 50mL of water in the morning for 5 days; beidellitic Montmorillonite 3g, one sachet every 8 hours in half a glass of water before a meal until flatulence stops. She received 2 bags of iso-group and iso-rhesus red blood cells. Hydrocortisone was not administered because cortisolemia was normal. Daily supportive psychotherapy and therapeutic education for the patient and those around her.

The evolution after 8 days was marked by an improvement in her general condition and a reduction in neurological disorders despite persistence of drowsiness and physical asthenia, the Glasgow score at 14/15, the collaborating patient complaining of odynophagia; an improvement in the muscular strength of the lower limbs but with hypotonia and difficulty walking, disappearance of dysesthesias. A disappearance of constipation and reduction of flatulence; disappearance of skin xerosis and scratching lesions, urine appears normal. The intraoral examination reveals lesions covered with a whitish coating, with irregular contours, of different sizes, non-bleeding on contact, painful on the internal surfaces of the cheeks, suggesting oral candidiasis; associated with lingual erosions next to the teeth. Paraclinically, hypochromic normocytic anemia at 8.5 g/dL (MCV at 74.3 fl; CCMH at 33.3 g/dl); a TSHus at 10.96 µui/mL; serum sodium at 117 mmol/L, serum chloride at 89 mmol/L; serum calcium at 2.27mmol/L, azotemia at 6.98 mmol/L; serum creatinine at 137µmol/L and clearance at 40.59mL/min. A renal ultrasound, measurement of serum phosphate, PTH, vitamin D and a swab of the intraoral lesions were requested but were not performed. She received a protocol against oral lesions based on [20 mL of baking soda + 10 mL of Amphotericin B + 30 mL of Chlorhexidine digluconate - Chlorobutanol hemihydrate] in 1 liter of water for mouthwash 4 or 5 times a day after dental cleaning and an application of lidocaine gel 2% in the morning before eating. She received 2 more bags of isogroup isorhesus red blood cells and she continued with the same treatment.

After 1 month of treatment, the patient's evolution was favorable marked by a normal neurological state, a conscious and very collaborative patient, a Glasgow score of 15/15, the disappearance of

drowsiness, the resumption of active mobilization, a marked reduction in diffuse myxedema (Image 2), a cessation of flatulence, a reduction in oral lesions associated with a resumption of oral nutrition. The outlet TSHus was 3.63 µiu/mL; FT4 at 18.74pg/mL; apart from hypocalcemia at 1.46mmol/L, the rest of the plasma ionogram was normal. An appointment was made in dermatology for the treatment of alopecia areata and vitiligo vulgaris. She was discharged after therapeutic education of the patient and her family, with rapid insulin 10iu every 8 hours associated with a blood sugar monitoring sheet, levothyoroxine 100µg per day, rosuvastatin 10mg per day, pregabalin 75 mg per day, chondroitin sulfate 500 mg capsule and amlodipine + valsartan (10/160 mg) 1 tab per day associated with a low-sodium diet.



Picture 2: Photographs of the patient– Day 35 (Dr Ibrahima A Dembélé – Dr Stéphane L Djeugoué)

DISCUSSION

This observation reports a case of autoimmune polyendocrine syndrome type IIIc made of Hashimoto's disease - vitiligo vulgaris - alopecia areata associated with a metabolic syndrome made of android obesity - hypercholesterolemia type of hyperLDLemia - a high blood pressure – type 2 diabetes mellitus. This is a rare combination of a complex endocrine and metabolic picture. Indeed, autoimmune endocrine syndromes or autoimmune polyendocrinopathies correspond to an association of at least 2 endocrine conditions linked to a disruption of the tolerance of the immune system, and often associated with other non-endocrine autoimmune diseases [1]. The concept of polyendocrinopathy is old since it was created by Henri C. J. Claude and Henri Gougerot in 1908 under the term multiglandular endocrine insufficiency [3]. In 1916, Luksh *et al*., reported on a case of illness Addison's syndrome associated with Hashimoto's thyroiditis and 1929, Thorpe *et al*., [3] defined an association of candidates and chronic hypoparathyroidism. Subsequently, the publications relating to these polyendocrinopathies have been multiplied.

To our knowledge, this is one of the rare published case reports of an association of an autoimmune polyendocrine syndrome type IIIc and a metabolic syndrome.

They APS are classified into two large groups: monogenic autoimmune polyendocrine syndromes characterized by diffuse autoimmunity (APS type 1 (APECED) and IPEX Syndrome (Immunodeficiency, Polyendocrinopathy, enteropathy X-linked)) and polygenic autoimmune polyendocrine syndromes [4]. APS 3 is a rare disease defined by the association of autoimmune dysthyroidism with type 1 diabetes, celiac disease, vitiligo or another autoimmune disease, in the absence of cortico-adrenal involvement according to the Neufeld classification [1]. S. Bammou *et al.*, found in his study 17 patients who had type 3 PEAI: 15 patients had Hashimoto's disease and type 1 diabetes, 1 patient had Hashimoto's disease, celiac disease and type 1 diabetes, only one patient had Graves' disease associated with type 1 diabetes [2]. Michel Assane Ndour and his team found in their survey that the majority of patients (95%) had type 3 AP and 5% had type 4 AP. Type 3 AP is subdivided into 4 subtypes. Most (57.9%) patients had subtype 3a. Subtypes 3b, 3c and 3d accounted for 21.1%, 18.4% and 2.6% respectively [3]. Fadel Fikri Suharto and his team reported a case of Autoimmune Polyglandular Syndrome Type 3-D in a 29-year-old patient associating Graves' disease and systemic lupus [4] and Tian S *et al.*, reported a case of autoimmune polyglandular syndrome type III associated with antineutrophil cytoplasmic autoantibody-mediated crescentic glomerulonephritis [10].

Type 1 PGAD:	Chronic mucocutaneous candidiasis Idiopathic hypoparathyroidism Addison's disease (at least two need to be present)
Type 2 PGAD:	Addison's disease Thyroid autoimmune disease and Insulin-dependent diabetes mellitus
Type 3 PGAS:	Thyroid autoimmune disease and other autoimmune disease (excluding Addison's disease and hypoparathyroidism or both a) Insulin-dependent diabetes mellitus b) Chronic atrophic gastritis (with or without pernicious anemia) c) Vitiligo, Alopecia, Myasthenia gravis d.Hypergonadotrophic hypogonadism d) Non-organ specific autoimmune disease (eg. SLE, Sjogren syndrome, rheumatoid arthritis etc).
Type 4 PGAD:	Associations not falling into any of the previous categories (eg. Alopecia and vitiligo or both, IDDM, Myasthenia Gravis and Insulin-Dependent diabetes).

Figure 1: Classification of autoimmune polyendocrine syndromes [5]

Autoimmune polyendocrine syndrome type 1 is rare, mainly affecting young children, the major signs are chronic mucocutaneous candidiasis, hypoparathyroidism, adrenal insufficiency [1]. Minor signs include other endocrinopathies (hypogonadism, insulin-dependent diabetes, autoimmune thyreopathy) and gastrointestinal diseases (gastric atrophy, pernicious anemia), chronic hepatitis, autoimmune skin disease (vitiligo, alopecia) , ectodermal dystrophy (dental enamel defect, nail dystrophy) [1]. The first manifestations begin in childhood and the progression towards polyendocrinopathy sets in during the first 20 years of life [1]. Polyendocrine syndrome type I is also called Blizzard syndrome or Whittaker syndrome. Pëkka Ahonen, in 1990, called it APECED (Autoimmune PolyEndocrinopathy Candidosis Ectodermal Dystrophy) [5]. It is an autosomal recessive genetic disease whose locus has been identified on chromosome 21q22.3 and called the AIRE (Autoimmune Regulator) gene [3].

Syndrome IPEX or X-linked immunodysregulation, polyendocrinopathy, and enteropathy (IPEX) is an extremely rare inherited syndrome characterized by early-onset type 1 diabetes, autoimmune enteropathy with intractable diarrhea and malabsorption, and dermatitis that may be eczematiform, ichthyosiform, or psoriasiform [5]. Eosinophilia and elevated IgE levels are frequently present in patients with IPEX. Kidney disease, most often membranous glomerulonephritis or interstitial nephritis, develops in

some patients. Later manifestations of the syndrome may include autoimmune thyroid disease, alopecia, various [4] autoimmune cytopenias, hepatitis, and exocrine pancreatitis [5]. Many features overlap with APS-1, but they usually develop much earlier in life than in APS-1. IPEX is frequently fatal in the first few years of life unless patients are promptly treated with immunosuppressive agents or, if possible, with allogeneic bone marrow transplantation, which can cure the disease [1].

Autoimmune polyendocrine syndrome type II is an adult polyendocrinopathy. APS type 2 is the most common (80%), also called Schmidt syndrome. It is defined by the presence of 2 elements among the following: Addison's disease (100%), autoimmune thyroid damage (69-82%) and type 1 diabetes (30-52%) [5]. Addison's disease begins after the age of 20 in half of patients. However, diabetes may be present before adrenal insufficiency [3]. The frequency of thyroid damage can reach 70%, taking into account subclinical forms which are common in autoimmune thyreopathies [2].. These thyroid disorders are either Graves' disease or Hashimoto's thyroiditis. Gonadal insufficiency is rarer [1]. Vitiligo, alopecia and Biermer anemia are also more rarely observed.

Autoimmune polyendocrine syndrome type III combines autoimmune thyroid damage (which is the main symptom) and insulin-dependent diabetes (type IIIa), vitiligo and/or alopecia (type IIIc) [1]. There is no adrenal insufficiency. Polyglandular syndrome type IIIa can also combine celiac disease and sarcoidosis (K.I. Papadopoulos, 1994) [1]. Sarcoidosis is also quite frequently associated with an endocrinopathy, in particular thyrotoxicosis, thyroiditis, Addison's disease [1]. Under the name “TASS syndrome”, Paul Seinfeld and Sarah Sharma (1983) described the association of thyroiditis, Addison's disease, Sjögren's syndrome and sarcoidosis [1]. Autoimmune polyendocrine syndrome 3 is considered to be a variant of APS-2. In order to diagnose APS-3 a patient needs to show symptoms of autoimmune thyroid disease without any symptoms of Addison’s disease. Autoimmune thyroid disease often coexists with type 1 diabetes (T1DM), premature ovarian insufficiency and rather rarely with lymphocytic hypophysitis [3]. In our patient, the polyendocrine picture was dominated by Hashimoto's disease complicated by myxedematous coma in the initial phase, associated with vitiligo vulgaris, predominantly in the 2 lower limbs and alopecia areata.

Table 1: Clinical forms of APS type III [5]

Endocrine Glands	Gastrointestinal Tract and Liver	Skin, Muscles, Nervous system, Hematologic system	Autoimmune Rheumatic Disorders Vasculitis
Type 1 DM Hirata’s syndrome Adeno-hypophysitis Neuro-hypophysitis Chronic hypoparathyroidism	Chronic atrophic gastritis Ulcerative colitis Pernicious anemia Celiac Disease Other inflammatory bowel syndrome Primary biliary cirrhosis Autoimmune hepatitis Sclerosing cholangiatis Autoimmune pancreatitis	Vitiligo Alopecia Bullous Skin diseases Myasthenia gravis Stiff Person Syndrome Multiple Sclerosis Autoimmune anemia Autoimmune thrombocytopenia	Systemic Lupus Erythematosus Discoid Lupus Erythematosus Rheumatoid arthritis Mixed connective tissue disease Seronegative arthritis Systemic sclerosis Dermato/polymyositis Vasculitis
3 A	3 B	3 C	3 D

Autoimmune polyendocrine syndrome type IV is defined by the association of auto-immune adrenal insufficiency or more than two autoimmune diseases with another autoimmune pathology excluding the major components of APS 1, 2 and 3 [6].

The goal of treatment is to achieve both remission of clinical signs of hypothyroidism and to balance blood sugar levels; to avoid complications and recurrences [3]. Therapeutic education of the patient and his family must be essential because it is a set of chronic pathologies. Our patient and her family benefited during her hospitalization and during the various check-ups from therapeutic education focused on the physical signs of complications of the different entities constituting her clinical picture and on the goal of the therapy. Given the coexistence of type 2 diabetes mellitus with Hashimoto's disease, there is a risk of hypoglycemia due to decreased insulin demand. After restoring hormonal balance with levothyroxine in a patient with hypothyroidism, the symptoms of hypoglycemia decrease [7].

Patients with hypothyroidism take levothyroxine at a dose of 1.2 to 1.6 µg/kg, checking TSH levels every 4 to 6 weeks initially. Our received levothyroxine at a dose of 1.6 µg/kg. It should be noted that levothyroxine increases cortisol clearance; Therefore, patients with undiagnosed adrenal insufficiency could suffer from acute adrenal crisis. Cortisol levels should be checked in the morning (8:00 a.m.) [7]. Vitiligo vulgaris is initially treated with supportive psychotherapy, cosmetic camouflage if the lesions are very extensive, administration of strong topical corticosteroids and repigmentation of the

epidermis by phototherapy [3]. The management of alopecia areata consists of intralesional infiltrations of corticosteroids, triamcinolone acetonide suspension (in doses of 0.1 mL to 3 mL at concentrations of 2.5 to 5 mg/mL every 4 at 8 weeks) can be injected intradermally if the lesions are small. Potent topical corticosteroids (eg, clobetasol propionate 0.05% foam, gel, or ointment 2 times/day for approximately 4 weeks) can be used [8]. In cases of small alopecic patches, corticosteroids are usually injected subcutaneously near the alopecic area. Minoxidil can also be applied directly to bald patches [9].

Regarding the components of the metabolic syndrome, management is focused on physical activity, a low-sodium diet, taking anti-hypertensives, taking normolipidemic drugs and normalizing blood sugar levels with the administration of anti-diabetic drugs oral or insulin. Our patient benefited from these different therapies.

In perspectives, efforts must be made in terms of research on the different entities of autoimmune polyendocrine syndrome in the sub-Saharan region.

CONCLUSION

APS represent a group of rare concomitant pathologies, which are increasingly diagnosed and can be detected at an asymptomatic stage. The progression may be marked by complications, likely to be life-threatening; especially if it is associated with metabolic syndrome. The early recognition of autoimmune dysthyroidism offers the opportunity to screen for other autoimmune endocrinopathies in the face of any declared Hashimoto's disease given their frequent associations. In any patient with an autoimmune disorder, regular monitoring is indicated to detect the outbreak of possible endocrine syndromes.

Consent: Written informed consent was obtained from the patient to publish this report in accordance with patient consent policies.

Author Contributions: All authors participated in the evaluation and follow-up of the patient, in the writing and correction of the case report. All authors of the manuscript have read and accepted its contents.

Competing Interests: The authors declare no conflict of interest.

REFERENCES

1. Jankowska, K., Dudek, P., Stasiek, M., & Suchta, K. (2023). Autoimmune polyendocrine syndromes associated with autoimmune rheumatic diseases. *Reumatologia*, *61*(4), 225-238.
2. Bammou, S., N'Koua, S. E., Rafi, S., El Mghari, G., & El Ansari, N. Les Polyendocrinopathies Auto Immunes Au Cours Du Diabete Type 1. *Int J Adv Res*, *10*(02), 91-94.
3. Ndour, M. A., Diédhiou, D., Dieng, M., Sow, D., Diallo, I. M., Djiba, B., ... & Mbaye, M. N. (2024). Profile of Autoimmune Polyendocrinopathies at the Medical Clinic II of the Abass Ndao Hospital: About 40 Cases. *Open Journal of Internal Medicine*, *14*(1), 74-82.
4. Suharto, F. F., Anggraini, R. D., Tamin, A., Fitricana, D., Kurniati, N., & Andayani, Y. D. (2022). Autoimmune Polyglandular Syndrome Type 3-D: A Case Report. *AIJR Preprints*.
5. Husebye, E. S., Anderson, M. S., & Kämpe, O. (2018). Autoimmune polyendocrine syndromes. *New England Journal of Medicine*, *378*(12), 1132-1141.
6. Wang, Y., Zhao, J., Zhang, T. T., Ma, X. W., Zhang, J. Q., & Zhang, Z. L. (2020). Autoimmune polyendocrine syndrome type 4 with systemic lupus erythematosus and anti-phospholipid syndrome. *Chinese Medical Journal*, *133*(08), 994-995.
7. Betterle, C., Garelli, S., Coco, G., & Burra, P. (2014). A rare combination of type 3 autoimmune polyendocrine syndrome (APS-3) or multiple autoimmune syndrome (MAS-3). *Autoimmunity highlights*, *5*, 27-31.
8. Kurozumi, A., Okada, Y., Nishio, K., & Tanaka, Y. (2021). Case of autoimmune polyendocrine syndrome type 3 complicated with anti-N-methyl-D-aspartic acid-receptor encephalitis. *Journal of Diabetes Investigation*, *12*(2), 290-292. doi: 10.1111/jdi.13341.
9. Gwoo, S., Choi, Y. S., Kim, B. K., Park, Y. H., Kim, K. T., & Lee, J. S. (2013). A Case of Type III Autoimmune Polyglandular Syndrome. *Kosin Medical Journal*, *28*(2), 167-170.
10. Tian, S., Xu, B., Liu, Z., & Liu, R. (2020). Autoimmune polyglandular syndrome type III associated with antineutrophil cytoplasmic autoantibody-mediated crescentic glomerulonephritis: a case report and literature review. *Medicine*, *99*(7), e19179.
11. Kutwin, M., Orłowska-Orlik, K., Bogaczewicz, J., & Woźniacka, A. (2019). Type 3 autoimmune polyendocrine syndrome (APS) diagnosed in an 87-year-old patient with a concomitant chronic autoimmune urticaria. *Dermatology Review/Przegląd Dermatologiczny*, *106*(4), 396-404.