Case Report

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Precursor B-Cell Lymphoblastic Leukemia Presenting as Thyrotoxic Goiter: A Case Report and Narrative Review of Evidence

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Abstract

Here, we report a case to emphasize the importance of recognizing the rare association between acute leukemia and hyperthyroidism. These may arise from direct leukemic infiltration of the thyroid gland, leading to structural and functional alterations, or from adverse hematologic effects of antithyroid medications such as agranulocytosis and, in extreme cases, acute leukemia. Our case describes a 32-year-old woman with previously diagnosed but untreated hyperthyroidism who presented with classic thyrotoxic symptoms as well as hematological findings that led to a diagnosis of precursor B-cell lymphoblastic leukemia. Overlap in symptomatology can delay diagnosis and complicate management, emphasizing the necessity for heightened clinical vigilance. By drawing attention to these possibilities, this report stresses the importance of careful evaluation and awareness when managing thyroid abnormalities in patients with hematologic malignancies.

Keywords: precursor B-cell lymphoblastic leukemia; grave's disease; thyrotoxic goiter

1. Introduction

Acute leukemia, encompassing acute myeloid leukemia (AML) and acute lymphoblastic leukemia (ALL), represents a heterogeneous group of rapidly progressive hematologic malignancies marked by the uncontrolled proliferation of immature hematopoietic blasts in the bone marrow. AML is the most common acute leukemia in adults, with a median age at the time of diagnosis being 68. Furthermore, males have a higher incidence compared to the female population, with a ratio of 5:3 [1]. ALL predominates in the pediatric population, the risk for developing ALL is highest in children younger than 5 years, further the risk then declines slowly until the mid-20s, and begins to rise again gradually after the age of 50. Overall, about 4 of every 10 cases of ALL are in adults [2]. Clinically, patients present with manifestations of bone marrow failure—fever, fatigue, recurrent infections, and bleeding tendencies—resulting from cytopenias. Diagnosis relies on morphology, immunophenotyping, cytogenetics, and molecular testing to differentiate it from other marrow disorders.

Thyrotoxicosis, most frequently secondary to Graves' disease, has a prevalence in the general population that is estimated to range roughly from 0.2% to 1.2% globally. Subclinical hyperthyroidism, a related mild form, affects about 0.7% to 1.4% of people, increasing to 2-3% among those aged 65 and older. Thyrotoxicosis affects women disproportionately, with female-to-male ratios between 7:1 and 10:1 [3]. It is characterized by excessive thyroid hormone levels, causing tachycardia, weight loss, tremors, and heat intolerance. Causes include Graves' disease, toxic multinodular goiter, subacute thyroiditis, and drug-induced thyroid dysfunction. Accurate diagnosis necessitates a combination of biochemical thyroid function tests and imaging modalities.

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Although rare, an association between acute leukemia and hyperthyroidism has been documented. These may arise from direct leukemic infiltration of the thyroid gland, leading to structural and functional alterations, or from adverse hematologic effects of antithyroid medications such as agranulocytosis and, in extreme cases, acute leukemia. Such overlap in symptomatology can delay diagnosis and complicate management, emphasizing the necessity for heightened clinical vigilance.

2. Case Presentation

A 32-year-old woman with a 2-year history of hyperthyroidism and goitre, with poor medication adherence, presented with 15 days of progressive odynophagia and increased swelling in the front of the neck. Dysphagia was more pronounced for solids than liquids, and a moderate-grade fever was also reported, which was relieved with antipyretics. She reported worsening palpitations and tremors over the past 45 days, during which she was started on carbimazole; low-grade fever, throat pain, and painful swallowing developed shortly thereafter. She noted a recent increase in the size of the neck swelling. Family history was negative for thyroid or other endocrine disorders. She was taking a mixed diet, with reduced appetite and disturbed sleep, with normal bowel and bladder habits.

On systemic examination, the patient was tachycardic and well-saturated with normal vesicular breath sounds bilaterally. Examination of the neck revealed a midline swelling, more prominent on the right side than the left, with the lower border visible. The right lobe swelling measured approximately 6 × 3 cm, while a discrete swelling on the left measured approximately 3 × 2 cm. The swelling moved with deglutition. On palpation, there was increased local temperature; the swelling was non-tender, firm in consistency, and had a smooth surface. A palpable thrill was appreciated at the right lower pole. Auscultation revealed a bruit over the right inferior pole.

Initial laboratory evaluation revealed anemia, thrombocytopenia, and marked leukocytosis with 80% blasts on the differential count. Red cell indices showed microcytosis and hypochromia. Peripheral smear demonstrated markedly elevated WBCs with large blast cells, three to four times the size of mature lymphocytes, having a high nuclear–cytoplasmic ratio, irregular nuclear membranes, open chromatin, scant cytoplasm, and one to two prominent nucleoli. Liver function tests and urinalysis were unremarkable except for trace proteinuria as seen in Tables 1 and 2. Thyroid profile showed elevated FT4 (2.99 ng/dL) and FT3 (5.50 pg/mL) with suppressed TSH (<0.005 µIU/mL). Abdominal ultrasonography demonstrated hepatomegaly (17.5 cm) and splenomegaly (17.9 × 6.5 cm) without intrahepatic biliary dilatation.

A radioiodine uptake scan and a 111 MBq pertechnetate thyroid scan demonstrated a nodular goitre with a hyperfunctioning nodule replacing the right lobe and a hypoplastic left lobe, consistent with a nodular Graves' pattern. ENT evaluation showed normal vocal cord mobility; upper gastrointestinal endoscopy was advised for dysphagia. Review of prior investigations (15/12/2022) showed T3 2.64 ng/mL, T4 18.30. µg/dL, TSH < 0.008 µIU/mL, and thyroid ultrasound revealing a 4.4 × 3.6 × 3.7 cm right lobe and a 1.9 × 1.4 × 1.6 cm left lobe, suggestive of nodular goiter.

During the hospital stay, the patient developed bilateral shin pain and subsequently a fever of 104 °F. Blood cultures were obtained, which were negative. Bone marrow biopsy and flow cytometry were performed following oncology consultation. Flow cytometry of bone marrow aspirate demonstrated medium- to large-sized blasts with a high nuclear–cytoplasmic ratio, round to oval or notched nuclei, and 1–3 nucleoli as shown in Figure 1. Immunophenotyping revealed a distinct CD45+ blast population characterized by low side scatter, positivity for CD34, TdT, HLA-DR, CD19, CD10, CD22, cytoplasmic CD79a, CD38, and CD58, and aberrant expression of myeloid markers CD13 and CD33. Following flow cytometry results, a diagnosis of precursor B cell lymphoblastic leukemia with CD 13 and CD 33 was made.

The patient, diagnosed with precursor B-cell acute lymphoblastic leukemia, was initiated on the MCP-841 induction protocol with rituximab on January 16, 2023. Induction therapy consisted of vincristine, daunorubicin, intrathecal methotrexate, L-asparaginase, and oral prednisolone, along

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with prophylactic allopurinol and supportive antimicrobial agents (acyclovir, fluconazole, and cotrimoxazole). Baseline laboratory investigations showed anemia with preserved platelet and leukocyte counts, and serial monitoring of hematologic and biochemical parameters was planned. During hospitalization, she tolerated induction chemotherapy with scheduled doses of rituximab and L-asparaginase and was discharged on January 21, 2023, with continuation of methimazole and propranolol for coexisting thyrotoxicosis. She was advised to follow up for thyroid function tests, fine-needle aspiration cytology of the thyroid, and repeat hematologic and biochemical investigations.

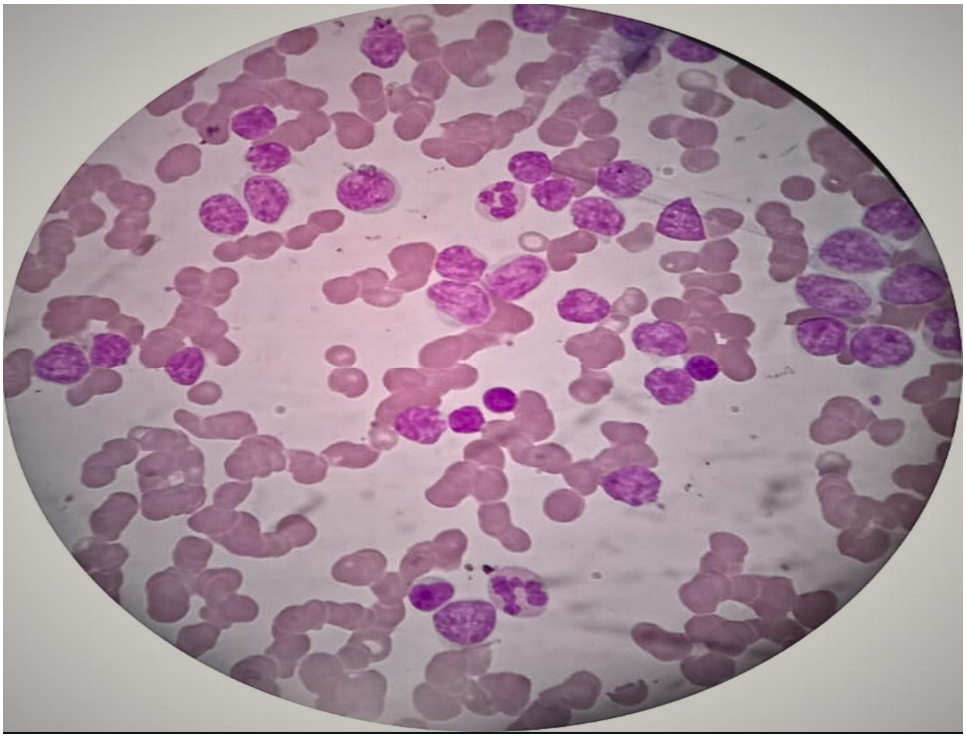

Figure 1. Peripheral Smear.

Table 1. Laboratory Results.

Parameter	Value	Units	Normal Range
Hemoglobin	8.9	g/dl	12–16 (F), 13–17 (M)
Total Leukocyte Count (TLC)	185,290	/mm^3	4,000–11,000
Red Blood Cells (RBC)	4.05	million/mm^3	4.2–5.4 (F), 4.7–6.1 (M)
Packed Cell Volume (PCV)	28.9	%	36–46 (F), 40–54 (M)
Mean Corpuscular Volume (MCV)	71.4	fL	80–100
Mean Corpuscular Hemoglobin (MCH)	22	pg	27–32
Mean Corpuscular	30.8	g/dl	32–36

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Hemoglobin Concentration			
Red Cell Distribution Width (RDW)	22.7	%	11.5–14.5
Erythrocyte Sedimentation Rate	40	mm/hr	<20 (M), <30 (F)
Total Bilirubin	0.7	mg/dl	0.3–1.2
Direct Bilirubin	0.4	mg/dl	<0.3
AST (SGOT)	22	U/L	5–40
ALT (SGPT)	11	U/L	5–45
Alkaline Phosphatase (ALP)	96	U/L	44–147
Total Protein	7.7	g/dl	6.0–8.3
Albumin	3.5	g/dl	3.5–5.0
Gamma Glutamyl Transferase(GGT)	31	U/L	9–48 (M), 8–35 (F)
Urea	20	mg/dl	15–40
Creatinine	0.9	mg/dl	0.6–1.3
Uric Acid	9.2	mg/dl	3.5–7.2 (M), 2.6–6.0 (F)
Sodium (Na^+)	131	mmol/L	135–145
Potassium (K^+)	3.7	mmol/L	3.5–5.0
Chloride (Cl^-)	99	mmol/L	98–106
Test	**Result**	**Reference/Normal Range**	
Iron Profile			
Serum Iron	42	60 – 170 µg/dL	
TIBC	220	240 – 450 µg/dL	
UIBC	177	150 – 375 µg/dL	

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Transferrin saturation	19%	20 – 50%	
Ferritin	Not reported		
Vitamin Profile			
Vitamin B12	481	200 – 900 pg/mL	
Folic Acid	1.4	2.7 – 17 ng/mL	
Hematology			
Reticulocyte count	0.8 %	0.5 – 1.5 %	
Corrected Reticulocyte Count	0.34 %	0.5 – 1.5 %	
Platelet	Pending	150 – 400 $\times 10^9$/L	
PCV	Pending	36 – 46 %	
Serology			
HIV	Negative	Negative	
HBsAg	Negative	Negative	
HEV	Negative	Negative	
VDRL	Pending	Negative	

Table 2. Urinalysis:.

Test	Result	Normal/Reference range
Urinalysis		
Specific Gravity	1.010	1.005 – 1.030
pH	7	4.6 – 8.0
Protein	Trace	Nil / Negative
Glucose	Nil	Nil / Negative
Ketone	Nil	Nil / Negative
Urobilinogen	Normal	Normal

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Bilirubin	Nil	Nil / Negative
Microscopy		
Pus cells	1 /hpf	0–5 /hpf
Epithelial cells	1 /hpf	0–5 /hpf
RBC	Nil	0–2 /hpf
Casts	Nil	Negative
Crystals	Nil	Negative
Nitrite	Nil	Negative
Leukocyte esterase	Nil	Negative
Other tests		
Dengue	Negative	Negative
Malaria	Negative	Negative

Table 3. Flow Cytometry Results.

Test Name	Results	Units	Bio. Ref. Interval
LEUKEMIA DIAGNOSTIC PANEL (Flow Cytometry)			
MARKERS	**RESULT (%)**	**INTENSITY**	**INTERPRETATION**
T cell markers			
CD3 (cyto)	3.8	Negative	Negative
CD5	20.2	Dim pos	Positive
CD7	11.9	Negative	Negative
B cell markers			
CD19	94.0	Moderate	Positive
CD20	4.6	Negative	Negative
CD22	52.6	Dim to mod	Positive
CD22 (cyto)	23.4	Dim pos	Positive
CD79a (cyto)	96.8	Mod to bright	Positive
CD58	71.1	Dim to mod	Positive
Myeloid markers			
CD13	64.0	Partial dim to mod	Positive
CD15	0.4	Negative	Negative
CD33	56.0	Partial dim to mod	Positive
CD66c	0.0	Negative	Negative
MPO	5.1	Negative	Negative
Precursor markers			
CD34	36.8	Dim to mod	Positive
CD117	0.2	Negative	Negative

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TdT	89.6	Moderate	Positive
CD99	98.8	Moderate	Positive
Other markers			
CD45	99.3	Dim pos	Positive
CD9	3.1	Negative	Negative
CD10	97.9	Mod to bright	Positive
HLA-DR	96.4	Mod to bright	Positive
CD38	94.1	Dim to mod	Positive

3. Discussion

The coexistence of hyperthyroidism, especially Graves' disease (GD), and acute leukemia is rare but increasingly reported in the literature. Our case describes a 32-year-old woman with previously diagnosed but untreated hyperthyroidism who presented with classic thyrotoxic symptoms as well as hematological findings that led to a diagnosis of precursor B-cell lymphoblastic leukemia. This case provides an opportunity to review potential associations, shared mechanisms, and important clinical considerations in such presentations.

Previous studies have demonstrated an increased incidence of thyroid diseases, predominantly autoimmune disorders such as Graves' disease and Hashimoto's thyroiditis, among patients with acute leukemia compared to the general population. One such study found a threefold higher prevalence of thyroid disease in patients with acute leukemia, with most cases involving acute myeloid leukemia (AML), but also acute lymphoblastic leukemia (ALL) and chronic myeloid leukemia in the accelerated phase. [4]

Table 4 summarises reported cases worldwide, describing the coexistence of hyperthyroidism and acute leukemias. Graves' disease was the most frequent thyroid condition, though toxic goiter, autoimmune or infectious thyroiditis, and thyroid carcinomas treated with radioiodine were also reported. The leukemias observed encompassed both lymphoid and myeloid lineages, including ALL, AML subtypes (M1, M2, M3/APL, M6), and myelomonocytic or monocytic variants. Treatments for hyperthyroidism frequently involved thionamides or radioiodine, both of which have been implicated in marrow toxicity or genotoxicity, and several cases developed acute leukemia following prolonged drug exposure or repeated radioiodine therapy.

Table 4. Summary of Reported Cases.

Sl no.	AUTHOR	Country	Age	Type of Hyperthyroidism	Treatment Received	Type Of Acute Leukemia	Treatment Received For Leukemia
1	Jiang et al. [5]	China	42/M		The patient was initially treated with propylthiouracil (PTU) at the time of diagnosis; however, due to drug-induced leukopenia, the therapy was switched to methimazole (MMI) in 2010	M3 leukemia harboring the FMS-like tyrosine kinase 3-internal tandem duplication.	ATRA (25 mg/m^2/d, *per os*)
2	Bishnoi et al. [6]	India	52/F	Follicular thyroid carcinoma	The patient underwent a total thyroidectomy in 2011. Between 2012 and 2016, she received 8	AML with myelodysplasia-related changes (WHO category II) as	N/A

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					cycles of radioactive iodine (RAI) therapy (200 mCi per cycle), with a cumulative dose of 1600 mCi. Subsequently, she was given palliative radiotherapy foran L4 spinal metastasis	per the 2017 revised WHO classification	
3	Nehara et al. [7]	India	26/ M	Graves disease	The patient was started on carbimazole 20 mg twice daily and propranolol 40 mg three times daily, followed by radioiodine ablation after achieving remission from acute lymphoblastic leukemia (ALL)	ALL with 85% blasts	Multicentric protocol (MCP 841) chemotherapy for 2.5 years (Oka et al. 2006)
4	Oka S et al. [8]	Japan	35/ M	D/t to metastatic involvement	N/A	Philadelphiaa-chromosome-PPositive– acute lymphoblastic leukemia (ALL)	chemotherapy for ALL with lenograstim 5g/kg per day by subcutaneous injection
5	Tsabouri et al. [9]	Greece	24/ F Pre gna nt	Graves' disease	She was initially managed with carbimazole, which was later switched to propylthiouracil 50 mg/day during her pregnancy	Acute lymphoblastic leukaemia (ALL)	Chemotherapy and 18 months later, achieved complete haematologic remission. Her current maintenance therapy for ALL
6	Niles D et al. [10]	USA	15/ M	Candia tropicalis thyroiditisisis	Started on methimazole, followed by propylthiouracil and eventually thyroidectomy after 9 weeks of treatment.	Acute lymphocytic leukemia	Induction chemotherapy with vincristine, daunorubicin, polyethylene glycol-asparaginase, and intrathecal methotrexate
7	Fadlalbari et al. [11]	SUDAN	16/ F	Graves' disease	The patient was initially started on carbimazole, and after 14 months, was transitioned to levothyroxine due to the development of hypothyroidism	ALL	N/A(Perillat-Menegaux et al. 2003)
8	Perillat-Menegaux F et al. [12]	France		Autoimmune thyroid diseases (Graves'	N/A	ALL ANLL	N/A

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				disease and/or hyperthyroidism and Hashimoto's disease, and/or hypothyroidism)			
9	Thomson [13]	Edinburgh	40/F	Thyrotoxicosis	Radioiodine therapy	Acute leukemia	treated with prednisone 40 mg daily and 6-mercaptopurine
10	Al-Anazi et al. [14]	Riyadh, Saudi Arabia	25/F	Thyrotoxic crisis occurring in a patient with Graves' disease induced by the course of chemotherapy given earlier.	The patient was started on carbimazole 20 mg twice daily, atenolol 100 mg per day, and hydrocortisone 100 mg intravenously, followed by 100 mg IV every 6 hours. By September 7, the thyrotoxic features had slightly improved; however, tachycardia had worsened with a pulse rate of 130/min. Consequently, the atenolol dose was increased to 600 mg/day, while the carbimazole dose was adjusted to 40 mg/day.	Acute myeloid leukaemia (AML, M2 type)	Induction course of chemotherapy (3+7 protocol) composed of cytarabine (Ara-C, Cytosar) 100 mg/m2 i.v. daily for 7 days and daunorubicin 60 mg/m2 i.v. daily for 3 days.
11	McBride [15]	Edinburgh	64/F	N/A	Carbimazole followed by radioactive iodine	Acute leukemia	Prednisolone 60mg followed by 6 6-mercaptopurine 100mg daily
12	Aksoy et al. [16]	Istanbul, Turkey	74/F	Grave's disease	Propylthiouracil	Acute myeloblastic leukemia	Corticosteroid, mercaptopurine, vincristine
13	Johnson et al. [17]	Texas	55/M	Diffuse toxic goiter with congestive heart failure.	Methimazole and reserpine, followed by radioactive iodine	Acute myelomonocytic leukemia	N/A
14	Imai et al. [18]	Niigata, Japan	11/M	Acute suppurative Thyroiditis with bacterial etiology	Clindamycin	AML (FAB classification: M1)	High-does cytarabine (AraC) and etoposide (ETP) with intrathecal injection of methotrexate (MTX), AraC, and hydrocortisone (HDC), pirarubicin, vincristine, and 5

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			14/ F	Acute suppurative Thyroiditis with bacterial etiology	Clindamycin	AML (FAB classification: M2)	days' continuous infusion of AraC
15	Kolade et al. [19]	New york	47/ M	Graves disease	Propylthiouracil, propranolol, followed by radioactive iodine	Acute promyelocytic leukemia	All trans retinoic acid with anthracycline-based chemotherapy
16	Fadilah et al. [20]	Kuala lumpur	18/ M	Transient hyperthyroidism	No anti-thyroid therapy	Acute lymphoblastic leukemia	L asparginase
			52/ M	Transient hyperthyroidism	No anti-thyroid therapy	Acute lymphoblastic leukemia	L asparginase
17	Laurenti et al. [21]	Italy	48/ F	Nodular thyroid	Radioiodine therapy	AML M2,	Mitoxantrone 12 mg/m2 days 1, 3, and 5, VP16 100 mg/m2 days 1–5, and cytosine arabinoside 100 mg/m2 days 1–10. Complete remission was achieved, and consolidation chemotherapy-motherapy with mitoxantrone 12 mg/m2 days 4, 5, and 6 and cytosine arabinoside 500 mg/m2 days 1–6.
			44/ F	Medullary thyroid carcinoma	Radioiodine therapy	AML M6	Not considered eligible for aggressive chemotherapy
18	McCormack et al. [22]	San Francisco	48/ M		Radioiodine therapy	Acute myelomonocytic leukemia	N/A
19	Burns et al. [23]	Missouri	65/ M	Graves disease	Radioiodine therapy	Acute monocytic leukemia	Prednisone, 20 mg per day,

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			64/ F	Goiter			
20	Kennedy et al. [24]	North Carolina	38/ M		Radioiodine therapy	Acute granulocytic leukemia	N/A
21	Mittal et al. [25]	India	34/ M	Thyrotoxicosis	Carbimazole	Acute myeloid leukemia	Induction chemotherapy (daunorubicin and cytarabine) for 7 days and three cycles of high-dose cytarabine chemotherapy as consolidation chemotherapy.
22	Khanna et al. [26]	India	58/ F	Clinically and biochemically euthyroid. Midline neck swelling that moved with deglutition.		Mixed phenotypic Acute leukemia (mixed myeloid/B/B/cell) with myeloid sarcoma, involving the thyroid gland	Hoelzer's protocol, comprising daunorubicin, vincristine, methylprednisolone, and L-asparaginase, along with intrathecal methotrexate.
23	Dana Goldenberg et al. [27]	USA	48/ F	Myeloid sarcoma of the thyroid	Cladribine, cytarabine, and filgrastim with mitoxantrone	Acute myeloid leukemia (AML)	Allogeneic stem cell transplant

5. Conclusions

The concurrent occurrence of hyperthyroidism, particularly autoimmune Graves' disease, and acute leukemia, though rare, represents a clinically significant intersection that warrants careful attention. The overlap of shared autoimmune and molecular mechanisms, coupled with the potential adverse effects of antithyroid therapy, highlights the intricate interplay between endocrine and hematological disorders. Recognizing these associations is essential, as it not only aids in timely diagnosis and comprehensive management but also opens avenues for further research into the pathophysiological links and therapeutic strategies at this intersection.

6. Limitations

The patient didn't have any additional workup, like imaging with FDG PET or surgical biopsy, which was not pursued due to a lack of any severe compressive symptoms. Also, the patient didn't undergo scheduled FNAC as she improved with chemotherapy; therefore, the possibility of infiltration of the thyroid by blast cells could not be ruled out.

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