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Thymic Lymphoid Follicular Hyperplasia in a Young Woman with Refractory Ulcerative Colitis and Thyroid Autoimmunity: A Case Report and Narrative Review

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Abstract

Introduction and purpose. The thymus is central to T-cell maturation, negative selection and regulatory T-cell development. Disturbance of thymic architecture or function can accompany autoimmune disease, but the clinical meaning of thymic abnormalities in inflammatory bowel disease remains uncertain. The aim of this article is to describe a young woman with long-standing refractory ulcerative colitis (UC), colitis-associated colorectal neoplasia, a

progressive anterior mediastinal lesion and biochemical evidence of thyroid autoimmunity, and to discuss the possible immunological links without assuming a causal relationship.

Materials and methods. A clinical case was analyzed together with a narrative review of the literature relevant to thymic immune tolerance, thymic hyperplasia, ulcerative colitis, colorectal neoplasia, thyroid autoimmunity and thymectomy. The discussion was based primarily on peer-reviewed literature indexed in PubMed/MEDLINE, with particular attention to reviews, observational studies and the limited historical literature directly addressing thymic abnormalities or thymectomy in UC. The review was narrative and was not conducted according to a systematic-review protocol.

Case presentation. A 25-year-old woman with approximately ten years of difficult-to-control UC underwent colectomy with end ileostomy after persistent disease activity despite medical therapy. Histopathology revealed colorectal carcinoma arising in a tubulovillous adenoma with high-grade dysplasia, with active UC elsewhere in the resected colon. Subsequent computed tomography demonstrated anterior mediastinal soft tissue compatible with thymic enlargement. Serial imaging showed progression from approximately 3 cm to 8 cm. Myasthenia gravis was excluded. Thyroid function remained normal, while thyrotropin receptor antibodies were weakly positive (3.0 IU/L; reference <1.5 IU/L). Thymectomy was performed because of lesion progression; histopathology demonstrated benign thymic lymphoid follicular hyperplasia. During subsequent observation, UC remained in remission after colectomy and thyrotropin receptor antibody levels decreased.

Conclusion. This case illustrates the coexistence of severe immune-mediated intestinal disease, thyroid autoimmunity and thymic lymphoid follicular hyperplasia. The combination is biologically compatible with shared immune dysregulation, but it does not establish that the thymic lesion caused UC or that thymectomy induced remission. Progressive anterior mediastinal lesions should be evaluated according to standard mediastinal-mass pathways. Further clinical research is required before thymic imaging or thymectomy can be recommended as part of routine UC management.

Keywords: thymus; thymic lymphoid follicular hyperplasia; ulcerative colitis; autoimmunity; thymectomy; anterior mediastinum; case report

1. Introduction

The thymus is the primary lymphoid organ in which T-cell precursors undergo maturation and selection. Positive selection preserves thymocytes capable of recognizing self-major histocompatibility complex molecules, whereas negative selection removes many clones with high affinity for self-antigens. In parallel, part of the thymocyte repertoire is directed toward the regulatory T-cell lineage. Together, these processes form a major component of central immune tolerance and reduce the number of potentially pathogenic autoreactive T cells entering the peripheral repertoire [1-4].

Medullary thymic epithelial cells are particularly important because they express a broad range of tissue-restricted antigens under the control of transcriptional programs that include AIRE and FEZF2. Presentation of these antigens contributes to deletion or functional redirection of autoreactive thymocytes. Central tolerance is not absolute, and peripheral mechanisms remain necessary, but structural or functional disturbances of the thymic microenvironment can alter the composition of the peripheral T-cell repertoire and may contribute to autoimmunity [2-5]. The thymus undergoes physiological involution after puberty, with progressive replacement of functional tissue by adipose tissue. Nevertheless, substantial thymic tissue can persist in young adults, and thymic enlargement may occur in several settings. The radiological differential diagnosis of an anterior mediastinal lesion includes thymic hyperplasia, thymoma, lymphoma, germ-cell tumors and thyroid lesions, among other entities. Clinical context, morphology on cross-sectional imaging and interval change are therefore essential to interpretation [6-8].

Thymic hyperplasia is not a single entity. True thymic hyperplasia refers to enlargement with preserved architecture, whereas lymphoid follicular hyperplasia is characterized by prominent lymphoid follicles and germinal centers and is classically associated with autoimmune disorders, particularly myasthenia gravis. Thymic enlargement is also well documented in Graves' disease, where both hormonal and immunological mechanisms have been proposed [7-9].

Ulcerative colitis is a chronic inflammatory bowel disease in which genetic susceptibility, epithelial barrier dysfunction, microbiome changes and dysregulated innate and adaptive immune responses interact. T-cell activation and impaired regulation are integral to the inflammatory phenotype, but the disease cannot be reduced to a single failure of thymic selection [10,11]. Long-standing, extensive and persistently active UC also increases the risk of colorectal neoplasia, making the oncological component of the present case clinically important rather than incidental [12-15].

Older literature explored thymic abnormalities and even thymectomy as a possible intervention in UC. These observations are historically interesting but arose before contemporary biologic therapy, modern endoscopic surveillance and current concepts of IBD pathogenesis. They therefore require cautious reinterpretation [16,17]. The present report describes an unusual combination of refractory UC, colorectal neoplasia, progressive thymic enlargement, lymphoid follicular hyperplasia and thyroid autoimmunity in a young woman. The purpose is not to propose thymectomy as UC therapy, but to examine what can—and cannot—reasonably be inferred from this clinical sequence.

2. Case presentation

A 25-year-old woman had been diagnosed with ulcerative colitis approximately ten years earlier. The available clinical history indicated a prolonged, unstable and treatment-refractory course. Multiple medical treatment attempts, including biological therapy, did not achieve durable disease control. Because of persistent inflammatory activity and intestinal complications, colectomy with end ileostomy was ultimately performed.

Histopathological examination of the surgical specimen demonstrated colorectal carcinoma developing in association with a tubulovillous adenoma containing high-grade dysplasia. The remaining colon showed active ulcerative colitis with mucosal ulceration. In a patient with a decade-long inflammatory disease, this finding is clinically consistent with the recognized association between sustained colonic inflammation and colorectal neoplasia, although the precise contribution of colitis to an individual tumor cannot be established from a single case [12-15].

During subsequent diagnostic work-up, computed tomography of the chest and abdomen demonstrated soft-tissue density in the anterior mediastinum. The initial radiological interpretation raised the possibility of enlarged thymic tissue or another lesion of thymic origin. Comparison with previous imaging showed substantial interval progression, from approximately 3 cm to approximately 8 cm.

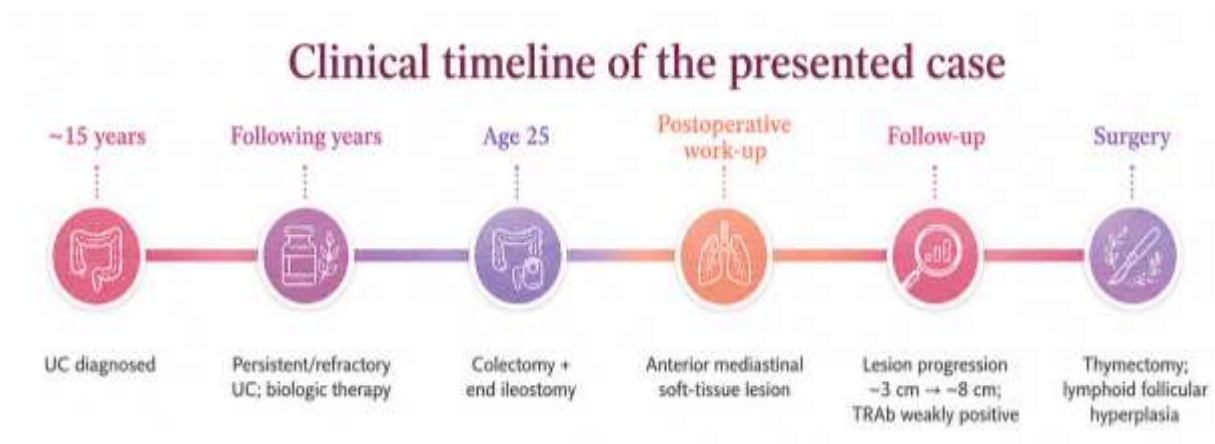
The patient was referred for endocrine evaluation. At the first documented endocrine assessment, the heart rate was regular at 70 beats/min and blood pressure was within the normal range. Thyroid hormone concentrations were normal. Family history did not reveal a known autoimmune disorder. Thyrotropin receptor antibodies (TRAb) were weakly positive at 3.0 IU/L (reference range <1.5 IU/L), without overt biochemical or clinical hyperthyroidism.

Because thymic lymphoid follicular hyperplasia is frequently associated with myasthenia gravis, the patient was assessed for this possibility; myasthenia gravis was excluded. The

progressive size of the anterior mediastinal lesion nevertheless required thoracic surgical assessment. Given the marked interval enlargement and the need to obtain a definitive diagnosis, thymectomy was performed.

Histopathological examination of the resected thymus showed benign thymic lymphoid follicular hyperplasia rather than thymoma or another malignant mediastinal tumor. During subsequent observation, the patient remained in remission from colonic UC after colectomy, and TRAb concentrations decreased. Because the colon had been removed and other treatments and time-related effects may have contributed, the post-thymectomy course cannot be used to infer a therapeutic effect of thymectomy on UC.

Figure 1. Clinical timeline of the presented case. The chronology was reconstructed from the information available in the source manuscript; exact dates were not available for all events.



Source: Authors' own elaboration based on data from the presented case.

Table 1. Key clinical features and their interpretation

Finding	Observed in the patient	Clinical interpretation
Ulcerative colitis	~10-year history; refractory course; biologic therapy; colectomy	Established immune-mediated intestinal disease with substantial inflammatory burden.
Colorectal neoplasia	Carcinoma arising in tubulovillous adenoma	Long-standing UC and persistent inflammation are recognized risk factors for colorectal neoplasia [12-15].

	with high-grade dysplasia	
Anterior mediastinal lesion	Progression from ~3 cm to ~8 cm	Interval growth justified definitive mediastinal evaluation; imaging alone could not establish histology.
Thyroid autoimmunity	TRAb 3.0 IU/L with normal thyroid hormones	Evidence of thyroid-directed autoimmunity without overt Graves' hyperthyroidism.
Myasthenia gravis	Excluded	Important because thymic lymphoid follicular hyperplasia is strongly associated with myasthenia gravis.
Thymic histology	Lymphoid follicular hyperplasia	Benign thymic pathology; does not by itself demonstrate a causal link with UC.
Follow-up	UC remission after colectomy; TRAb decreased	Temporal association only. Colectomy removes the target organ of UC, and TRAb may fluctuate over time.

3. Discussion

The thymus as an organ of central tolerance:

Interpretation of this case depends first on the physiological role of the thymus. Developing T cells encounter self-peptide–MHC complexes presented by thymic epithelial cells and professional antigen-presenting cells. Strong recognition of self-antigens can lead to deletion, while selected thymocytes are diverted into regulatory lineages. The result is not complete elimination of autoreactivity, but a major reduction in the number and pathogenic potential of self-reactive clones entering the circulation [1-5].

This framework is relevant to systemic autoimmunity because abnormalities of thymic selection may have consequences beyond a single peripheral organ. AIRE-dependent and FEZF2-dependent expression of tissue-restricted antigens broadens the range of self-antigens encountered during T-cell development. Defects in these pathways provide strong proof of principle that disturbed central tolerance can produce multi-organ autoimmunity. However, most common autoimmune diseases are polygenic and multifactorial; subtle changes in thymic selection are only one possible component of a much larger network [2-5].

Accordingly, the patient's thymic hyperplasia cannot be regarded as the cause of UC on the basis of the present observations. Lymphoid follicular hyperplasia may be a marker of an activated autoimmune environment rather than its initiating lesion. Conversely, chronic systemic inflammation may influence thymic architecture. The direction of association cannot be resolved from chronology alone.

4. Thymic lymphoid follicular hyperplasia

Lymphoid follicular hyperplasia is characterized histologically by B-cell follicles with germinal centers within the thymic medulla. It differs from true thymic hyperplasia, in which thymic size is increased but normal architecture is retained. The distinction matters because the two patterns have different clinical associations and radiological implications [7-9].

The classic clinical association is myasthenia gravis, which explains why neuromuscular evaluation was appropriate in this patient. Other autoimmune diseases may coexist with thymic abnormalities, but the strength of association varies substantially. A finding of lymphoid follicular hyperplasia should therefore prompt interpretation in the full clinical context rather than automatic attribution to a specific autoimmune diagnosis.

In young adults, thymic tissue can be prominent and may undergo rebound after physiological stress. At the same time, progressive enlargement of an anterior mediastinal lesion cannot be dismissed as physiological. The increase from approximately 3 cm to 8 cm in the present patient created a legitimate diagnostic problem: a benign hyperplastic process was possible, but thymoma, lymphoma and other anterior mediastinal tumors had to remain in the differential until tissue diagnosis.

Table 2. Differential diagnosis of an anterior mediastinal lesion in a young adult

Entity	Typical context	Imaging/clinical clues	Relevance to this case
Thymic hyperplasia	Young adults; autoimmune disease; rebound states	Diffuse or symmetric thymic enlargement; preserved morphology may favor benignity	Final diagnosis: lymphoid follicular hyperplasia.
Thymoma	Usually adults; may be associated with myasthenia gravis or	Focal thymic mass; morphology and invasion determine concern	Needed exclusion because lesion enlarged substantially.

	other paraneoplastic autoimmunity		
Lymphoma	Young patients possible; constitutional symptoms may occur	Bulky mediastinal lymphadenopathy or mass; may involve multiple nodal stations	Important alternative diagnosis in a progressive mediastinal lesion.
Germ-cell tumor	Often young adults	Anterior mediastinal mass; tumor markers and imaging may help	Age made this part of the standard differential.
Ectopic/substernal thyroid tissue	Thyroid disease or goiter	Continuity with thyroid or characteristic enhancement/radionuclide behavior	Thyroid autoimmunity was present, but histology confirmed thymic origin.

5. Ulcerative colitis and immune dysregulation

UC is characterized by chronic mucosal inflammation beginning in the rectum and extending proximally in a continuous pattern. Contemporary models emphasize interaction between host genetics, epithelial barrier function, microbial communities and innate and adaptive immunity [10,11]. T cells participate in this network, but UC is not considered a primary thymic disease. A possible gut-thymus relationship is biologically plausible, although direct human evidence remains limited. Experimental inflammation can influence thymic cellularity and architecture, and peripheral inflammatory signals can alter thymopoiesis. Conversely, the composition of the T-cell repertoire exported from the thymus can influence immune responses at mucosal surfaces. These concepts support bidirectional communication between central lymphoid organs and peripheral inflammatory sites, but human evidence specific to UC remains limited. Older studies reported thymic abnormalities in patients with UC and investigated whether thymectomy altered disease activity [16,17]. Such studies should be interpreted in their historical context. They predate current definitions of disease activity, modern biologic and small-molecule therapy, treat-to-target strategies and contemporary surgical pathways. They cannot establish thymectomy as an evidence-based treatment for refractory UC.

The 1991 series by Tsuchiya and colleagues described experience with thymectomy over a 13-year period [17]. Earlier work from the same group discussed immunological abnormalities involving the thymus and reported therapeutic observations after thymectomy [16]. These reports are valuable for hypothesis generation, but the absence of modern randomized comparisons and the possibility of selection bias, concomitant therapy and natural fluctuation of UC make causal interpretation difficult.

6. Colitis-associated colorectal neoplasia

The colorectal carcinoma found after colectomy is an important component of the case. Chronic colonic inflammation is a recognized driver of dysplasia and cancer in UC. The risk is influenced by disease duration, extent and inflammatory burden, among other factors [12-15]. A meta-analysis of studies evaluating mucosal inflammation found that objective inflammation was associated with a higher risk of subsequent colorectal neoplasia than mucosal healing [12].

Contemporary population-based estimates suggest that the absolute risk of colorectal cancer in UC is lower than in many historical cohorts, likely reflecting better inflammation control and surveillance, but the relative risk remains elevated. A 2025 population-based meta-analysis involving 161,157 patients estimated a standardized incidence ratio of 2.48 (95% CI 1.64-3.76) compared with the general population, with higher risk in extensive colitis (SIR 3.95, 95% CI 2.56-6.09) [15]. This estimate is consistent with broader work on inflammation-associated carcinogenesis and with earlier cohort-based and meta-analytic studies showing an elevated, although declining, colorectal cancer risk in UC [18-22].

The presence of cancer in this patient should therefore primarily be interpreted through the established UC–inflammation–neoplasia pathway. There is no evidence that thymic hyperplasia directly caused the colorectal malignancy. It is more defensible to view the malignancy as a complication occurring in the setting of long-standing inflammatory bowel disease, while acknowledging that systemic immune dysregulation may influence tumor–immune interactions more broadly.

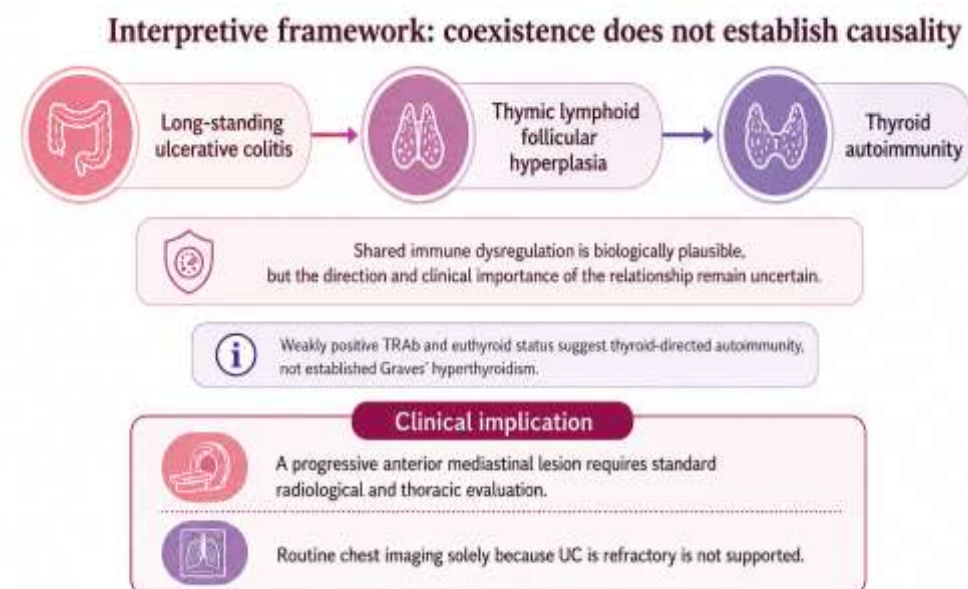
7. Thyroid autoimmunity and thymic enlargement

The weakly positive TRAb result adds a second autoimmune signal. TRAb are strongly associated with Graves' disease, but the patient was euthyroid and did not have overt hyperthyroidism. This distinction is important: the case demonstrates thyroid-directed

autoimmunity, not established Graves' hyperthyroidism. Coexistence of UC and Graves' disease has been described, but such reports do not establish a shared thymic mechanism [23]. The relationship between Graves' disease and thymic enlargement is well described. Reviews and case series indicate that thymic hyperplasia can accompany Graves' hyperthyroidism and often regresses after restoration of euthyroidism [8,9]. Proposed mechanisms include direct trophic effects of thyroid hormones and immunological stimulation within the thymus. Human thymic tissue expresses thyrotropin receptors, and thymic size can decrease in parallel with treatment of Graves' hyperthyroidism [24,25]. Awareness of this association can prevent unnecessary surgery when imaging is reassuring and the thymus regresses with treatment [8,26].

The present patient differs from the typical Graves-associated thymic hyperplasia scenario because thyroid hormone concentrations were normal, TRAb elevation was modest, and the mediastinal lesion showed marked interval progression. A conservative strategy based solely on a presumed Graves' association would therefore have been difficult to justify. Histology ultimately established benign lymphoid follicular hyperplasia.

Figure 2. Interpretive framework for the coexistence of ulcerative colitis, thymic lymphoid follicular hyperplasia, and thyroid autoimmunity. The figure distinguishes biological plausibility from causal inference.



Source: Author's own elaboration based on the presented case and the reviewed literature.

8. Could thymectomy modify autoimmune disease?

Thymectomy is an established component of management in selected patients with myasthenia gravis and is performed for thymic tumors when indicated. Its role in other autoimmune diseases is much less certain. Removal of a pathologically altered thymus could theoretically reduce abnormal antigen presentation or lymphocyte activation, but thymectomy also removes a site of ongoing T-cell production and may have long-term effects on immune homeostasis. Historical UC reports suggested prolonged remission in some thymectomized patients [16,17]. These observations cannot be directly transferred to contemporary practice. Patients selected for surgery may have differed from controls, medical treatment was not standardized according to modern criteria, and spontaneous variation in UC activity complicates before-and-after comparisons. In addition, colectomy itself is definitive treatment for colonic UC, making post-colectomy remission in the present patient unsuitable as evidence of a thymectomy effect. The most scientifically defensible interpretation is therefore that thymectomy was performed for a progressive anterior mediastinal lesion requiring diagnosis, not as treatment for UC. Any subsequent immunological changes are hypothesis-generating observations.

9. Clinical implications

Clinically, the case sits at the intersection of gastroenterology, endocrinology, immunology, radiology and thoracic surgery. In a young patient with autoimmune disease, an anterior mediastinal lesion should not automatically be labeled thymoma, but neither should it automatically be considered benign hyperplasia. Age, symptoms, autoimmune context, imaging morphology and interval growth all matter.

The case should not be used to justify screening the mediastinum in every patient with refractory UC. Current UC management focuses on intestinal disease assessment, complications, cancer surveillance and extraintestinal manifestations. The available evidence does not establish routine chest imaging for thymic pathology as part of standard UC care.

The clinically defensible conclusion is narrower: when chest imaging has already demonstrated thymic enlargement—particularly if it is progressive, atypical or accompanied by symptoms—clinicians should recognize both neoplastic and autoimmune associations and pursue standard evaluation of an anterior mediastinal lesion. In patients with biochemical or clinical thyroid autoimmunity, Graves-associated thymic hyperplasia should be considered, but the degree of thyroid dysfunction and imaging behavior must be taken into account.

10. Relationship between thymic output and peripheral mucosal immunity

A further issue is whether changes in thymic output can be detected at peripheral mucosal sites. Newly generated T cells leave the thymus and enter the circulating naïve T-cell pool before being recruited to secondary lymphoid organs and tissues. In principle, altered thymic selection or output could therefore influence the composition of lymphocyte populations that later participate in intestinal immune responses. This provides a mechanistic bridge between a central lymphoid organ and a disease expressed predominantly in the colonic mucosa. Age-related studies also demonstrate that reduced thymic output changes the naïve T-cell compartment and T-cell receptor diversity, illustrating how thymic biology can shape peripheral immunity [27].

However, the existence of such a bridge does not mean that increased thymic activity is necessarily pathogenic. The thymus can contribute both conventional T cells and regulatory populations, and peripheral expansion, tissue recruitment, antigen exposure and local cytokine signaling can substantially reshape the repertoire after thymic egress. Consequently, peripheral T-cell abnormalities observed in inflammatory bowel disease cannot be assumed to originate in the thymus. Reviews of central tolerance emphasize that thymic antigen presentation and selection are only part of the mechanisms that ultimately determine peripheral autoreactivity [28,29].

This distinction is particularly relevant to the present patient. Lymphoid follicular hyperplasia demonstrates active lymphoid organization within the thymus, but histology alone cannot reveal whether the exported T-cell repertoire was quantitatively or qualitatively abnormal. Studies incorporating T-cell receptor repertoire analysis, recent thymic emigrant markers and longitudinal immune phenotyping would be needed to test that hypothesis.

11. Evidence quality and interpretation of the historical thymectomy literature

The historical literature on thymectomy in UC is unusual because it addresses a therapeutic concept that has largely disappeared from contemporary gastroenterology. Reports from the 1980s and early 1990s described immunological abnormalities involving the thymus and suggested clinical improvement in selected patients after thymectomy [16,17]. These studies deserve citation because they directly concern the hypothesis raised by the present case, but they should not be assigned the same evidential weight as modern controlled therapeutic trials. A later case report describing UC improvement after excision of an invasive thymoma is likewise hypothesis-generating rather than evidence of therapeutic efficacy [30].

Several sources of bias are likely. Surgical selection may have favored patients with particular disease phenotypes; definitions of remission and refractory disease differed from current standards; concomitant corticosteroid or immunomodulatory therapy could confound outcomes; and colectomy or other interventions may have occurred during follow-up. Moreover, biologic agents, therapeutic drug monitoring, endoscopic treat-to-target strategies and contemporary small-molecule therapies were not available. The apparent duration of remission after thymectomy in historical cohorts therefore cannot be interpreted as proof of efficacy in a modern UC population.

The present case should not be framed as a therapeutic replication of those historical observations. The indication for thymectomy was diagnostic and surgical management of a markedly enlarging mediastinal lesion. The subsequent course is clinically noteworthy, but it remains an uncontrolled temporal association.

12. Directions for future research

Several research questions emerge from the case without requiring a causal interpretation. First, imaging-based studies could determine whether thymic size, morphology or rebound patterns differ between young adults with IBD and age-matched controls. Such studies would need to account for corticosteroid exposure, nutritional status, systemic inflammation, recent surgery and other factors known to influence thymic appearance.

Second, immune-phenotyping studies could examine recent thymic emigrants, naïve T-cell subsets, regulatory T cells and T-cell receptor diversity in patients with active versus quiescent UC. If a reproducible thymic signature were found, longitudinal sampling before and after effective intestinal therapy could help determine whether the thymic change precedes disease activity or instead reflects systemic inflammation.

Third, patients with coincident thymic pathology and IBD could be characterized through multicenter registries. Given the rarity of this combination, prospective randomized surgical studies are unlikely to be feasible or ethically justified in the absence of a standard thoracic indication. Carefully documented observational cohorts would be a more realistic first step. They should distinguish true thymic hyperplasia, lymphoid follicular hyperplasia, thymoma and other mediastinal lesions rather than grouping them under the broad label of 'thymic abnormality'.

Finally, thyroid autoimmunity should be treated as a separate variable rather than evidence of a single thymus-driven syndrome. Graves-associated thymic hyperplasia is a recognized entity [8,9], but the patient described here was euthyroid with only weakly positive TRAb. Future

reports should therefore include serial thyroid hormone concentrations, TRAb titers and imaging measurements to clarify whether thymic change parallels thyroid autoimmunity, intestinal inflammation, or neither.

13. Limitations

This report has several limitations. First, it describes a single patient and cannot establish causality. Second, exact dates, complete longitudinal laboratory data, detailed endoscopic activity scores, full medication history and radiological descriptors were not available in the source manuscript. The clinical timeline should therefore be interpreted as approximate where exact dates are not stated.

Third, the postoperative course is difficult to interpret because colectomy, thymectomy, changes in medical treatment and the natural evolution of autoantibody concentrations occurred within the same broad period. Fourth, the historical literature on thymectomy in UC is small and methodologically weak by current standards. Finally, the patient had thyroid-directed autoantibodies without overt hyperthyroidism, so evidence derived from Graves' hyperthyroidism cannot be applied directly.

These limitations are particularly relevant because the clustering of intestinal, thymic and thyroid immune abnormalities is also the feature most liable to overinterpretation. The report is best viewed as a clinically unusual observation that supports further investigation of immune-system interactions rather than as evidence for a new diagnostic or therapeutic pathway.

14. Summary and conclusions

This case describes a young woman with long-standing refractory ulcerative colitis, colorectal neoplasia, a progressively enlarging anterior mediastinal lesion, weakly positive thyrotropin receptor antibodies and histologically confirmed thymic lymphoid follicular hyperplasia. The coexistence of these findings is compatible with a background of multi-organ immune dysregulation, and current knowledge of thymic tolerance provides a biologically plausible framework for discussing such an association.

However, biological plausibility should not be confused with causality. The available evidence does not demonstrate that thymic hyperplasia caused UC, that thymectomy induced UC remission, or that routine mediastinal imaging should be introduced for patients with refractory UC. In this patient, the strongest indication for thymectomy was the substantial progression of an anterior mediastinal lesion requiring definitive diagnosis.

The principal clinical value of the case is to keep thymic pathology in the differential diagnosis of an anterior mediastinal abnormality in a patient with autoimmune disease, while avoiding both under-investigation of a progressive lesion and over-attribution of systemic disease to the thymus. Prospective studies would be required to determine whether specific thymic phenotypes have reproducible clinical relevance in inflammatory bowel disease.

Declaration of the use of generative AI and AI-assisted technologies in the writing process

During the preparation of this manuscript, the author used ChatGPT (OpenAI) to assist with language refinement, improvement of clarity and readability, and manuscript structuring and formatting. After using this tool, the author critically reviewed and edited the content as needed and takes full responsibility for the final content of the manuscript.

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