

ORIGINAL ARTICLE

Recognizing the Columnar Cell Subtype of Papillary Thyroid Carcinoma: A Case Report With Histopathologic and IHC Insights

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ABSTRACT

Columnar Cell subtype of Papillary Thyroid Carcinoma (CC-PTC) is an exceptionally rare histologic subtype, accounting for less than 0.5% of all papillary thyroid carcinomas worldwide.^{1,2} Despite its rarity, CC-PTC demonstrates aggressive biological behavior and is classified as a high-risk histologic subtype in the World Health Organization Classification of Tumors of Endocrine Organs, 5th Edition (2022)³, owing to its frequent association with capsular invasion, lymph vascular permeation, extrathyroidal extension, and distant metastasis. The tumor poses significant diagnostic challenges due to its distinctive morphology, particularly on cytology and limited biopsy material.^{4,6}

Case Presentation: A 27-year-old woman presented with a one-year history of anterior neck swelling with recent onset throat pain. Ultrasonography revealed a right thyroid lobe nodule categorized as TI-RADS 4/5. Fine-needle aspiration cytology showed highly cellular smears composed of tall and columnar epithelial cells arranged in papillary fragments and sheets, exhibiting nuclear elongation, hyperchromatic nuclei, marked pseudo stratification, and occasional nuclear pseudoinclusions, consistent with Bethesda Category VI. Total thyroidectomy revealed a 3-cm ill-defined, unencapsulated grey-white tumor.

Histopathological and Immunohistochemical Findings: Histopathology demonstrated circumscribed tumor composed of complex papillary and trabecular architecture lined by pseudostratified columnar cells with elongated

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hyperchromatic nuclei, conspicuous eosinophilic nucleoli, amphophilic cytoplasm, increased mitotic activity, and extensive lymphovascular invasion. Immunohistochemistry confirmed thyroid follicular epithelial origin with tumor cell nuclear positivity for TTF-1 and retained membranous β -catenin expression, while CDX2 was negative. A high Ki-67 labelling index supported the aggressive proliferative nature of the tumor.

Conclusion: This case highlights the importance of recognizing the distinctive cytomorphic, histopathologic and immunohistochemical features of CC-PTC. Accurate diagnosis requires a multimodal approach integrating clinical findings, cytology, histopathology, and immunohistochemistry, particularly in young patients where aggressive behavior may be unsuspected.

KEYWORDS:

• Papillary Thyroid Carcinoma • Columnar Cell Variant • High-Risk Thyroid Carcinoma • Fine-Needle Aspiration Cytology • Immunohistochemistry • TTF-1 • Ki-67 • β -Catenin • CDX2

INTRODUCTION

Papillary thyroid carcinoma (PTC) constitutes the majority of thyroid malignancies and is generally associated with an indolent clinical course and favorable prognosis. Nevertheless, PTC represents a heterogeneous group of tumors encompassing several histologic variants with distinct biological behavior, therapeutic implications, and prognostic outcomes¹. Accurate identification of these subtypes is therefore essential for optimal risk stratification and clinical management.

The columnar cell subtypes of papillary thyroid carcinoma (CC-PTC) is an exceptionally rare subtype, accounting for less than 0.5% of all ptc's.^{2,3} Despite its rarity, CC-PTC is consistently associated with aggressive biological behavior, including early capsular invasion, extensive lymph vascular permeation, extrathyroidal extension, and distant metastasis, particularly to the lungs.⁴ Reflecting this aggressive nature CC-PTC is categorized as a high-risk histologic variant in the world health organization classification of tumors of endocrine organs, 5th edition (2022).³

Histomorphologically, CC-PTC is characterized by papillary, trabecular, or glandular architecture lined by tall to columnar epithelial cells arranged in a pseudostratified fashion, displaying elongated hyperchromatic nuclei, conspicuous eosinophilic nucleoli, amphophilic cytoplasm, and increased mitotic activity.^{5,6} Classical nuclear features of PTC may be focal or subtle or absent, further complicating recognition. Owing to

these features, CC-PTC frequently mimics gastrointestinal adenocarcinoma, particularly on cytology and limited biopsy material, posing a significant diagnostic pitfall.⁷

Given its rarity, aggressive behavior, and potential for misdiagnosis, detailed documentation of CC-PTC cases—especially in young patients—is critical. We report a case of CC-PTC in a 27-year-old woman, emphasizing the cytological, histopathological, immunohistochemical, and diagnostic considerations that underpin accurate diagnosis and prognostic assessment.

CASE PRESENTATION

A 27-year-old woman presented to the surgical outpatient department with a gradually enlarging anterior neck swelling that she had first noticed approximately one year prior. Over the preceding three days, she reported the onset of acute throat pain accompanied by odynophagia. The pain was described as sharp, localized to the midline of the neck, and exacerbated by swallowing. She denied any symptoms of hoarseness, dyspnea, choking sensation, fever, night sweats, or weight loss. There was no history of prior radiation exposure, thyroid disease, autoimmune disorder, or significant family history of thyroid malignancy.

On clinical examination, she appeared well and normotensive. Inspection revealed a visible midline neck prominence that moved with deglutition. Palpation confirmed a firm, non-tender, 3 × 1 cm swelling in the anterior neck,

located near the isthmus-right lobe junction. The mass was mobile with swallowing but not with protrusion of the tongue, confirming thyroid origin. No cervical lymph nodes were palpable. The rest of the systemic examination was unremarkable.



Figure 1: Initial Presentation of Swelling in front of the neck

Laboratory investigations revealed normal thyroid function, with a TSH level of 0.85 MIU/mL and normal free T3 and free T4 levels. Anti-thyroid antibodies were not elevated.

The initial ultrasonography performed at a peripheral center described a hypoechoic heterogeneous nodule in the right thyroid lobe and classified it as TI-RADS III. Given her progression of symptoms, a repeat ultrasound was performed at our institution. This revealed a well-defined, heterogeneously hypoechoic, taller-than-wide nodule measuring $3.5 \times 1.7 \times 2.0$ cm, with minimal intranodular vascularity and absence of microcalcifications. The lesion was categorized as TI-RADS 4/5 due to suspicious sonographic features.

Cytological Findings

Fine-needle aspiration cytology revealed highly cellular smears composed of papillary fragments and cohesive sheets of tall to columnar epithelial cells. The cells exhibited elongated, hyperchromatic nuclei with prominent pseudo stratification, coarse chromatin and conspicuous nucleoli. Occasional nuclear pseudoinclusions, chromatin clearing and nuclear grooves. Focally rosette like structures were also seen. Based on these findings, a diagnosis of Bethesda Category VI (Malignant) was rendered, with features suggestive of a columnar cell variant of papillary thyroid carcinoma.

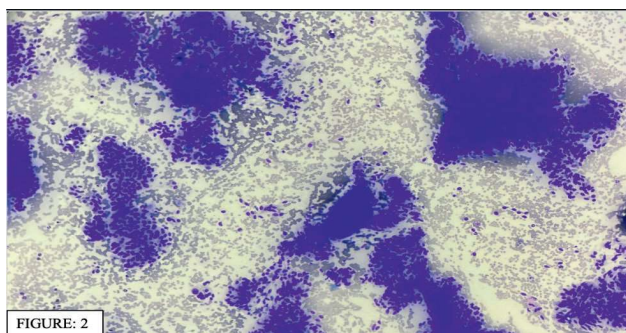


Figure 2: Hypercellular smear with tumor cells arranged in papillary fronds, syncytial sheets and at places rosette like pattern.

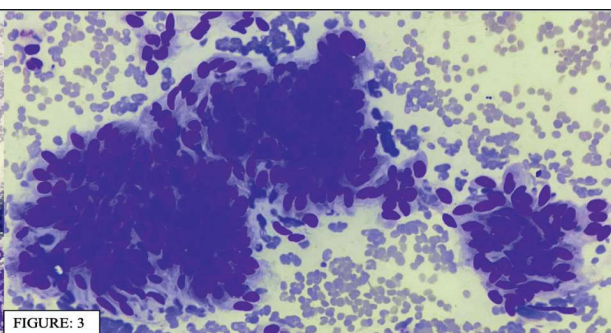


Figure 3: Pseudo stratification of nuclei, cellular crowding, wispy cytoplasm, nucleomegaly, moderate degree of anisonucleosis, dark chromatin and inconspicuous nucleoli.

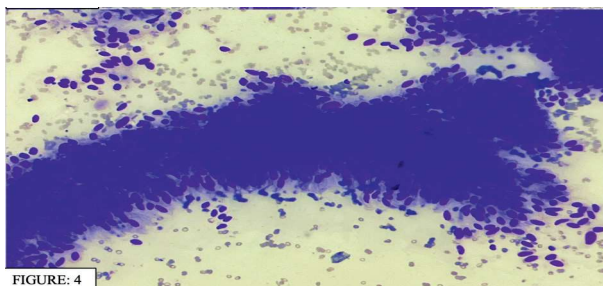


Figure 4: Pseudo stratification with paucity of grooves and inclusions is a cytological clue to CCV-PTC Gross Pathology

Gross Pathology

Total thyroidectomy specimen revealed a 3-cm, ill-defined, unencapsulated grey-white lesion involving the right lobe of the thyroid. The cut surface was solid and firm, without areas of hemorrhage or necrosis. The remaining thyroid tissue appeared grossly unremarkable.

Histopathological Findings

Microscopic examination revealed a circumscribed neoplastic lesion with partial

encapsulation. The tumor was composed of epithelial cells arranged predominantly in

papillary architecture, with additional trabeculae and follicular patterns.



Figure 5 & 6: Gross images

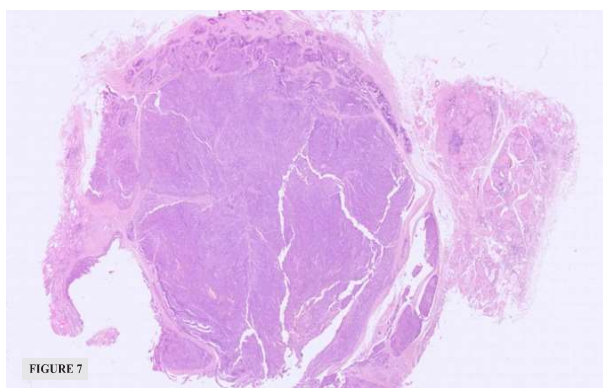


Figure 7: Circumscribed cellular tumor with adjacent thyroid tissue

The tumor cells were tall to columnar, showing marked nuclear pseudo stratification, elongated

hyperchromatic nuclei, coarse chromatin, and prominent nucleoli, with occasional nuclear grooving. The cytoplasm was eosinophilic. Mitotic activity was increased, with 2-3 mitoses per 10 high-power fields. Focally subnuclear vacuolization resembling secretory endometrium was noted. Nuclear clearing, intranuclear cytoplasmic pseudo inclusions, and psammoma bodies were absent.

There was extensive lymph vascular invasion and tumor capsular invasion. No evidence of extrathyroidal extension.

The thyroid parenchyma adjacent to the tumor was unremarkable. The contralateral thyroid lobe showed variably sized colloid-filled follicles without evidence of neoplasia.

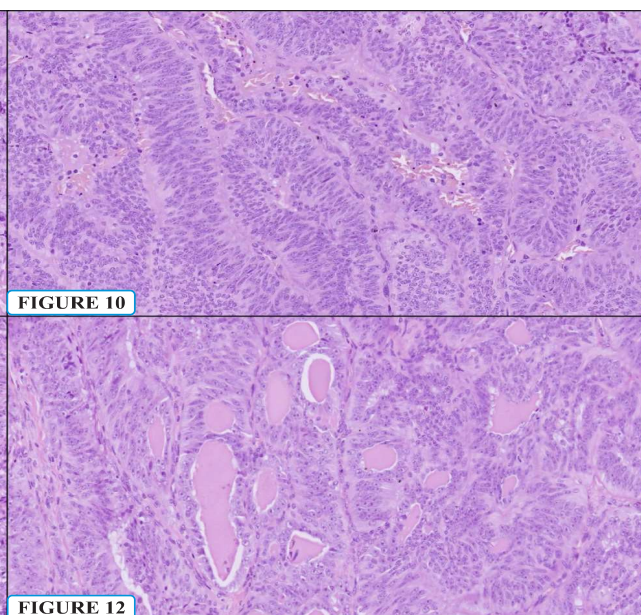
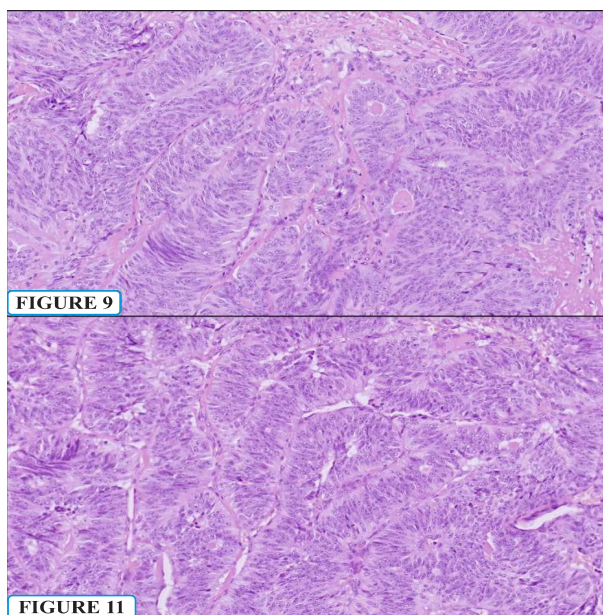


Figure 9: Papillary pattern

Figure 10: Trabecular pattern

Figure 11: Glandular like pattern

Figure 12: Follicular pattern

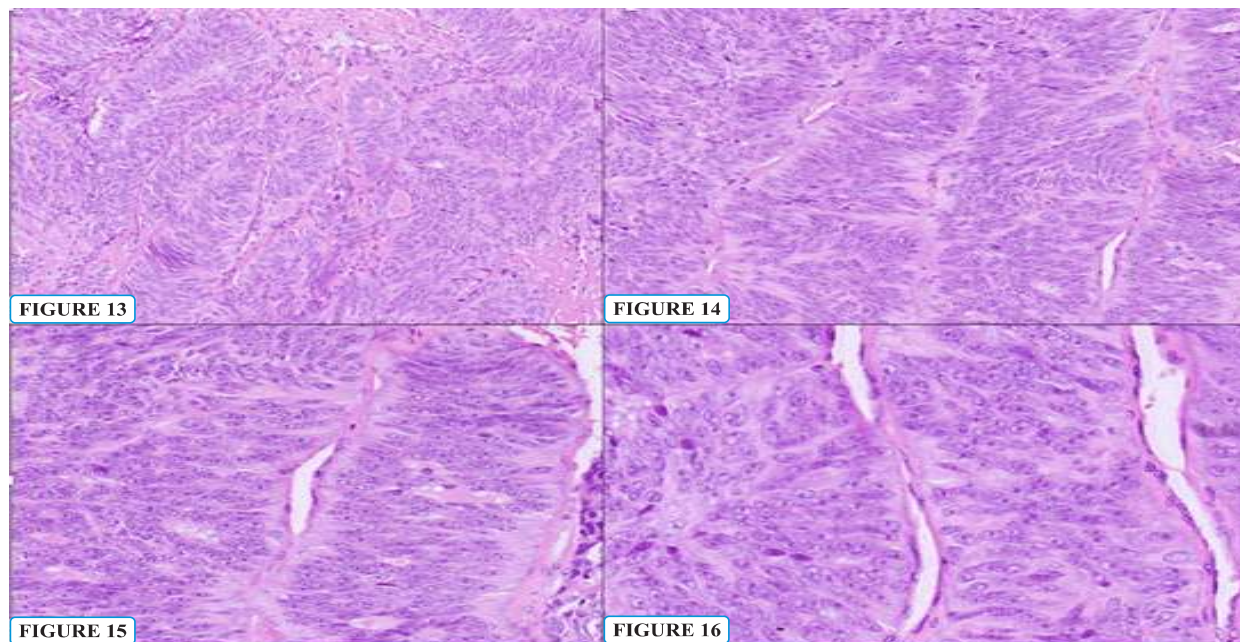


Figure 13: Complex papillae **Figure 14:** Papillae lined by crowded elongated pseudostratified epithelial cells composed of columnar cells with pale eosinophilic to clear cytoplasm, and prominent nuclear pseudo stratification
Figure 15, 16: Tall columnar cells, elongated nuclei with nucleoli and eosinophilic cytoplasm

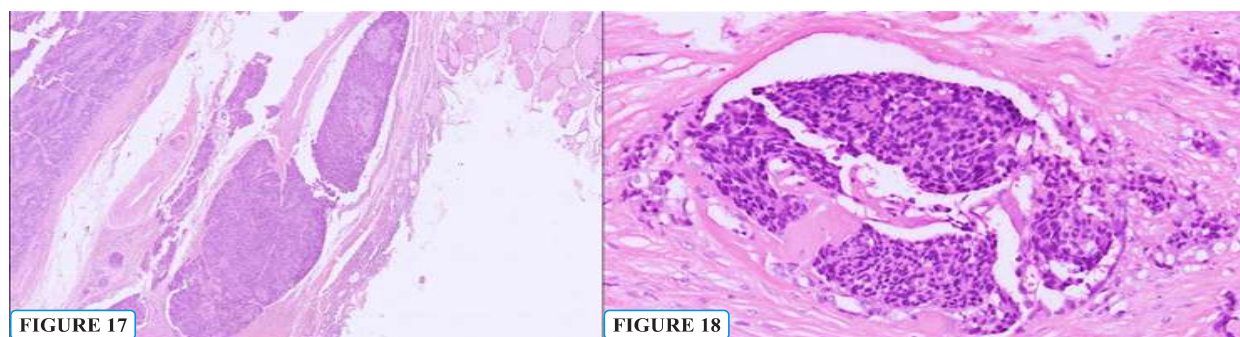


Figure 17: Capsular invasion

Figure 18: Vascular (blood vessel) invasion

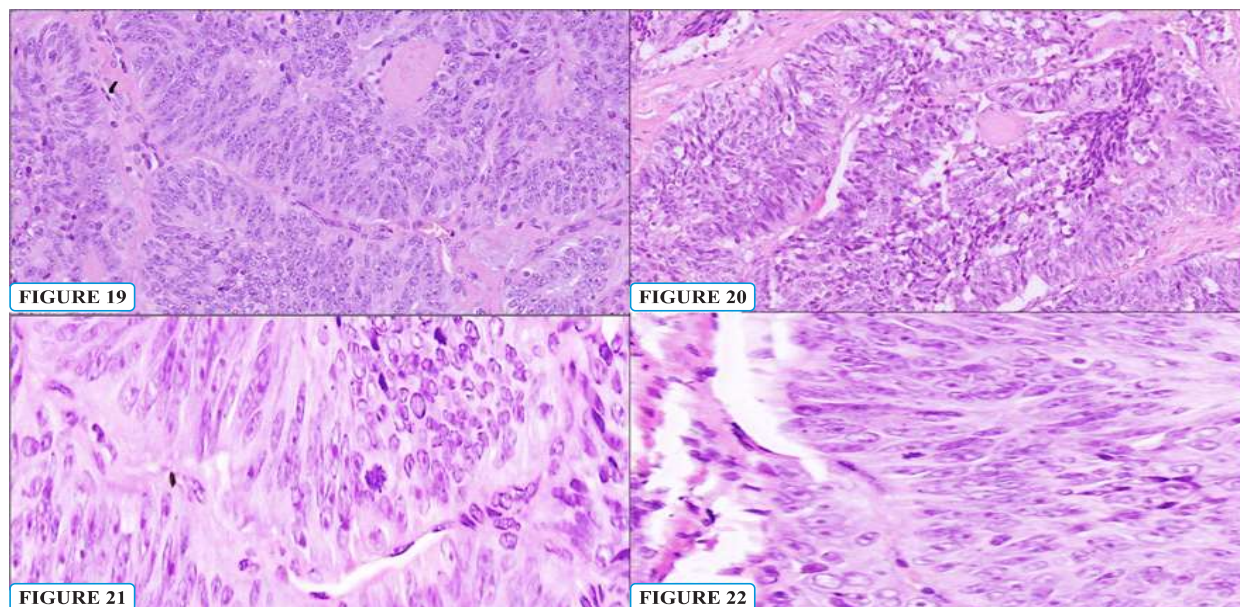


Figure 19: Complex papillae

Figure 20: Subnuclear vacuolization

Figure 21, 22: Mitotic activity 3-4/10hpf

Immunohistochemical Findings

Immunohistochemistry demonstrated tumor cells positive for TTF-1. β -catenin showed retained membranous positivity. Negative staining for CDX2, excluding metastatic gastrointestinal adenocarcinoma. The tumor cells showed an elevated Ki-67 proliferation index, supporting thyroid follicular epithelial

origin and aggressive biological behavior

Based on the combined clinicoradiological,

cytological, histopathological, and immunohistochemical findings, a final diagnosis of Papillary carcinoma of **Thyroid (Columnar cell variant)** Pathologic Stage Classification (ptnm, AJCC 8th Edition)-Pt2NxMx

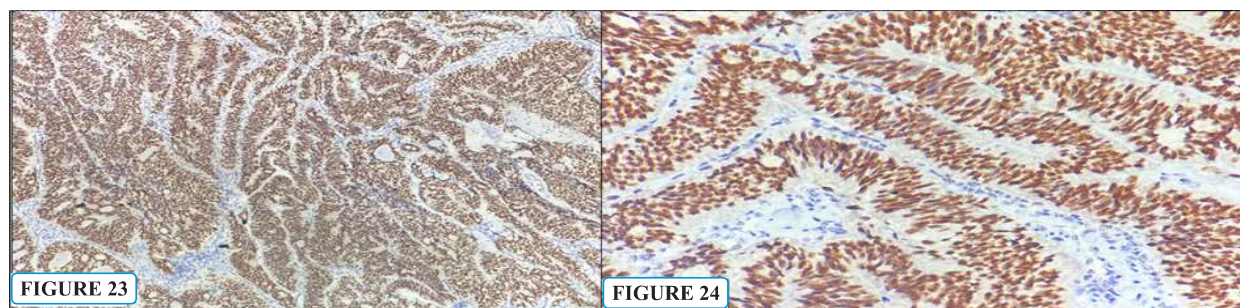


Figure 23 & 24: TTF -1 Positive

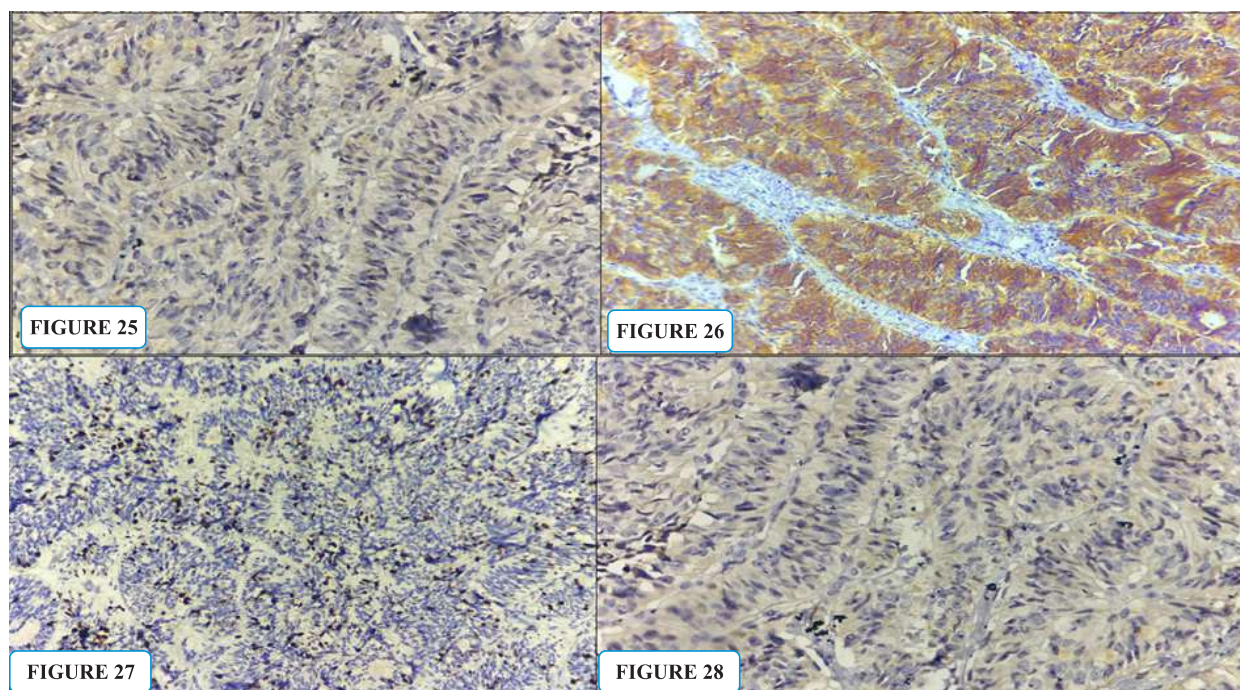


Figure 25: CDX2 negative

Figure 26: Beta catenin - retained membranous and cytoplasmic positivity

Figure 27: Ki-67 (52%)

Figure 28: BRAF (V600E) - negative

Differential Diagnosis

The principal differential diagnoses considered included:

Metastatic gastrointestinal adenocarcinoma - excluded by absence of CDX2 expression and presence of TTF-1 positivity. (Figure 23)

Tall cell variant of PTC - differs by cell height-to-width ratio, more prominent nuclear

clearing, and less pseudo stratification.

Poorly differentiated thyroid carcinoma - excluded by preservation of papillary architecture and absence of solid/insular growth.

Recognition of the characteristic architectural and cytologic features, supported by immunohistochemistry, was critical in establishing correct diagnosis.

DISCUSSION

Columnar Cell subtype of Papillary Thyroid Carcinoma (CC-PTC) is a rare histologic subtype of papillary thyroid carcinoma, accounting for fewer than 0.5% of cases, yet demonstrating aggressive biological behaviour.^{1,2} In recognition of its clinicopathologic characteristics and adverse prognostic implications, CC-PTC is classified as a high-risk histologic variant in the World Health Organization Classification of Tumors of Endocrine Organs, 5th Edition (2022).³

The defining histopathological features of CC-PTC include complex architectural patterns such as papillary, trabecular, and follicular arrangements lined by tall to columnar epithelial cells exhibiting prominent nuclear pseudo stratification. The tumor cells typically show elongated hyperchromatic nuclei, conspicuous nucleoli, eosinophilic cytoplasm, and increased mitotic activity.^{4,5} Classical nuclear features of papillary thyroid carcinoma may be variable or inconspicuous, as observed in the present case.⁶

The present case demonstrated several histologic features associated with aggressive biological behavior, including extensive lymphovascular invasion, tumour capsular invasion, and focal infiltration into adjacent skeletal muscle. These findings are well documented in CC-PTC and correlate with its recognized high-risk status.^{7,8}

Increased Ki-67 (Figure: 27) The elevated mitotic activity and increased Ki-67 labeling index further support the high proliferative nature of this variant.⁹

Immunohistochemistry plays an important role in confirming thyroid follicular epithelial differentiation in CC-PTC. Tumor cell positivity for TTF-1, along with retained membranous β -catenin expression, supports the diagnosis, while CDX2 negativity is consistent with a primary thyroid neoplasm.^{10,11} These immunophenotypic findings, in conjunction with characteristic histomorphology, allow confident diagnosis.

The occurrence of CC-PTC in a young patient, as in the present case, is noteworthy. Although papillary thyroid carcinoma is common in younger age groups, aggressive histologic variants may not be clinically anticipated, underscoring the importance of meticulous pathologic evaluation.¹² Early recognition of

CC-PTC is essential, as it has direct implications for surgical management, risk stratification, and postoperative surveillance.

Based on growth pattern and invasive characteristics, CC-PTC can be divided into encapsulated and infiltrative forms, which show significant differences in biological behavior and prognosis. The encapsulated form is typically well-circumscribed and confined within a fibrous capsule without evidence of invasion into the surrounding thyroid parenchyma or extrathyroidal soft tissue. These tumors are associated with relatively indolent clinical behavior and favorable prognosis when completely excised. In contrast, the infiltrative form demonstrates irregular tumor margins with invasion into adjacent thyroid tissue and may be associated with lymphovascular invasion, extrathyroidal extension, and increased risk of recurrence and metastasis. Therefore, the distinction between encapsulated and infiltrative forms of CC-PTC is important, as tumor invasiveness is a key determinant of clinical outcome and management.¹³

CONCLUSION

Columnar Cell subtype of Papillary Thyroid Carcinoma is a distinctly rare yet biologically aggressive subtype of papillary thyroid carcinoma and is recognized as a high-risk histologic variant in the current World Health Organization classification.¹ The present case exemplifies the characteristic clinicopathologic spectrum of this entity, including tall to columnar tumour cells with prominent nuclear pseudo stratification, increased mitotic activity, and extensive lymphovascular and capsular invasion—features that underpin its adverse biological behaviour.²⁻⁴

This report underscores the critical importance of meticulous histopathological evaluation supported by targeted immunohistochemistry in establishing an accurate diagnosis, particularly in young patients in whom aggressive variants may not be clinically anticipated.^{5,6} Early recognition of CC-PTC has direct implications for risk stratification, surgical decision-making, and vigilant postoperative surveillance. By documenting the salient morphologic and immunophenotypic features of this rare variant, this case adds meaningful evidence to the limited existing literature and reinforces the need for heightened awareness among practicing pathologists to optimize patient outcomes.^{7,8}

REFERENCES

1. Lloyd RV, Osamura RY, Klöppel G, Rosai J, Editors. WHO Classification Of Tumours Of Endocrine Organs. 5th Ed. Lyon: International Agency For Research On Cancer; 2022.
2. Asioli S, Erickson LA, Sebo TJ, Zhang J, Jin L, Thompson GB, et al. Papillary thyroid carcinoma with prominent columnar cell features: a clinicopathologic study of 17 cases. *Am J Surg Pathol*. 2010;34(5):662–669. doi:10.1097/PAS.0b013e3181d96c57.
3. Mete O, Asa SL. Pathological definition and clinical significance of aggressive variants of papillary thyroid carcinoma. *J Clin Endocrinol Metab*. 2011;96(6):1806–1814. doi:10.1210/jc.2010-2877.
4. Wenig BM. Aggressive variants of papillary thyroid carcinoma. *Mod Pathol*. 2011;24(Suppl 2):S18–S27. doi:10.1038/modpathol.2010.131.
5. LiVolsi VA. Papillary thyroid carcinoma: an update. *Mod Pathol*. 2011;24(Suppl 2):S1–S9. doi:10.1038/modpathol.2010.129.
6. Chen JH, Faquin WC. Diagnosis of papillary thyroid carcinoma variants on fine-needle aspiration biopsy. *Cancer Cytopathol*. 2012;120(6):407–414. doi:10.1002/cncy.21225.
7. Baloch ZW, LiVolsi VA. Fine-needle aspiration of thyroid nodules: past, present, and future. *Endocr Pract*. 2004;10(3):234–241. doi:10.4158/EP.10.3.234.
8. Bongiovanni M, Spitale A, Faquin WC, Mazzucchelli L, Baloch ZW. The Bethesda System for Reporting Thyroid Cytopathology: a meta-analysis. *Acta Cytol*. 2012;56(4):333–339. doi:10.1159/000339959.
9. Ordóñez NG. Value of thyroid transcription factor-1 immunostaining in tumor diagnosis: a review and update. *Appl Immunohistochem Mol Morphol*. 2012;20(5):429–444. doi:10.1097/PAI.0b013e318254056b.
10. Xu B, Ghossein R. Evolution of the histologic classification of thyroid neoplasms and its impact on clinical management. *Eur J Surg Oncol*. 2018;44(3):338–347. doi:10.1016/j.ejso.2017.11.016.
11. Rivera M, Tuttle RM, Patel S, Shaha A, Shah JP, Ghossein RA. Encapsulated papillary thyroid carcinoma: a clinicopathologic study of 106 cases. *Hum Pathol*. 2009;40(12):1708–1713. doi:10.1016/j.humpath.2009.04.007.
12. Tallini G, Tuttle RM, Ghossein RA. The history of the follicular variant of papillary thyroid carcinoma. *J Clin Endocrinol Metab*. 2017;102(1):15–22. doi:10.1210/jc.2016-2970.
13. Evans HL. Columnar-cell carcinoma of the thyroid. A report of nine cases of a distinctive type of thyroid carcinoma. *Am J Surg Pathol*. 1986;10(11):737–749.