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MODERN CLASSIFICATION AND DIFFERENTIAL DIAGNOSIS OF THYROID CARCINOMA: A SYSTEMATIC REVIEW

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Abstract

Introduction: Thyroid cancer is the most common endocrine malignancy. Although most thyroid nodules are benign, accurate differentiation from malignant lesions is critical for treatment decisions and prognosis.

Methods: A systematic literature search was conducted in PubMed, Scopus, and Google Scholar (2015-2024). Keywords included "thyroid cancer diagnosis", "Bethesda classification", "fine needle aspiration biopsy", "papillary thyroid carcinoma", and "molecular testing". Quality assessment used QUADAS-2.

Results: Analysis demonstrates that ultrasound, fine-needle aspiration biopsy, and the Bethesda cytological classification system remain the cornerstone of thyroid nodule evaluation. Molecular testing significantly improves diagnostic accuracy, especially in indeterminate cytological cases (Bethesda III-IV). Key genetic mutations include BRAF V600E (29-69% in papillary), RAS (40-50% in follicular), and RET (~25% in hereditary medullary carcinoma).

Conclusion: Accurate diagnostic evaluation using modern clinical, cytological, and molecular methods significantly improves patient outcomes and allows individualized management of thyroid cancer.

Keywords: Thyroid cancer, Bethesda system, FNAB, TIRADS, Molecular diagnostics, BRAF mutation

Introduction

Thyroid cancer is the most common malignancy of the endocrine system, and its incidence has increased significantly worldwide over the past decades [1]. The widespread use of high-resolution ultrasound has led to frequent detection of thyroid nodules, many of which are asymptomatic and discovered incidentally [2]. Although thyroid nodules are common in the general population, only a small percentage are malignant, making accurate differentiation a critical clinical task [3].

Early and precise diagnosis of thyroid cancer plays a decisive role in determining treatment strategy, preventing unnecessary surgical interventions, and improving patient prognosis [4]. Delayed diagnosis may result in local tissue invasion, regional lymph node metastasis, and distant spread [5]. Papillary thyroid carcinoma, which represents the majority of cases, usually

has an excellent prognosis when detected early. However, other histological types, such as medullary and anaplastic carcinomas, are associated with more aggressive clinical courses [6].

In recent years, significant progress has been made in diagnostic approaches. Ultrasound examination, fine-needle aspiration biopsy (FNAB), and cytological assessment using the Bethesda classification system have become essential tools [7]. Additionally, advances in molecular genetics have improved understanding of thyroid carcinogenesis and provided new opportunities for improving diagnostic accuracy [8].

This systematic review aims to summarize modern classifications, risk factors, histopathological characteristics, and current diagnostic methods used in the differential diagnosis of thyroid cancer.

Methods

Search Strategy

This systematic review was conducted following PRISMA 2020 guidelines. A comprehensive literature search was performed in electronic databases including PubMed, Scopus, and Google Scholar. The search period covered January 2015 to December 2024.

Search terms used:
("thyroid cancer" OR "thyroid carcinoma" OR "thyroid nodule") AND ("diagnosis" OR "Bethesda" OR "FNAB" OR "fine needle aspiration") AND ("ultrasound" OR "TIRADS" OR "molecular testing" OR "BRAF" OR "RAS")

Inclusion Criteria

Articles were included if they:

Were published in peer-reviewed journals

Addressed thyroid carcinoma (papillary, follicular, medullary, or anaplastic types)

Focused on etiology, histology, diagnostic methods, or molecular tests

Were available in English with full-text access

Provided original data, systematic reviews, or clinical guidelines

Exclusion Criteria

Articles were excluded if they:

Were case reports with fewer than 10 patients

Focused exclusively on benign thyroid disease

Were conference abstracts or editorials

Did not address human subjects

Quality Assessment

Diagnostic accuracy studies were assessed using the QUADAS-2 tool. Systematic reviews

were evaluated using AMSTAR-2.

Data Extraction

The following information was extracted: first author, publication year, study design, sample size, key genetic mutations, diagnostic test performance (sensitivity, specificity, predictive values), and clinical outcomes.

Results

1. Epidemiology and Etiology

The reviewed literature indicates that familial occurrence accounts for approximately 5% of papillary and follicular thyroid carcinomas, and 15-30% of medullary thyroid carcinoma [9,10]. The increasing incidence of papillary thyroid carcinoma is attributed to wider use of high-resolution imaging and intensified screening [11].

Risk factors consistently reported include:

Female sex

Hereditary predisposition

Childhood exposure to ionizing radiation [12]

Age (peak incidence 45-55 years)

An autopsy study suggests that thyroid carcinoma may affect both sexes equally, with higher clinical diagnosis rates in women potentially explained by differential healthcare access [13].

2. Genetic Mutations by Histological Type

Table 1. Key genetic mutations in thyroid carcinoma

Histological Type	Key Genetic Alterations	Reported Frequency
Papillary carcinoma	BRAF V600E mutation	29-69%
	RET/PTC translocation	~7%
	RAS mutations (follicular variant)	10-20%
Follicular carcinoma	RAS mutations	40-50%
	PAX8/PPARγ translocation	~30-35%

Histological Type	Key Genetic Alterations	Reported Frequency
Anaplastic carcinoma	p53 inactivating mutations	50-80%
	CTNNB1 mutations	66%
	RAS mutations	20-40%
Medullary carcinoma	Germline RET mutations (hereditary)	~25%
	RAS mutations	~25%

The MAPK signaling pathway plays a major pathogenetic role in thyroid carcinoma formation [14]. TERT promoter mutations are associated with highly aggressive papillary thyroid carcinoma [15].

3. Histological Characteristics

Papillary thyroid carcinoma: Microscopically characterized by papillary architecture with a vascular-rich stromal core. Tumor cells typically exhibit large, clear nuclei with ground-glass appearance, nuclear grooves, and intranuclear pseudoinclusions. Psammoma bodies are commonly observed [16].

Follicular thyroid carcinoma: Histological features range from well-differentiated follicular patterns to poorly differentiated patterns. Diagnosis requires demonstration of capsular or vascular invasion. Subclassification includes minimally invasive, encapsulated, angioinvasive, and widely invasive types [17].

Hurthle cell neoplasm: Characterized by eosinophilic oncocytic cells with abundant cytoplasm and prominent nucleoli [18].

Medullary thyroid carcinoma: Derived from parafollicular C cells, histology shows spindle cells without follicle formation. Amyloid deposition and calcitonin immunoreactivity are typically present [19].

Anaplastic thyroid carcinoma: Histological variants include spindle cell, pleomorphic giant cell, and squamoid types. Atypical mitoses are very common. Immunohistochemical staining for thyroid-specific markers (TTF-1, PAX8, thyroglobulin) is often negative [20].

4. Diagnostic Evaluation

Initial biochemical assessment: A thyroid function panel is recommended. Hyperthyroid status is associated with low malignancy risk; radionuclide uptake scanning is indicated [21].

Ultrasound findings: High-risk features include:

Significant increase in size compared to prior imaging

Hypoechogenicity

Irregular margins

Taller-than-wide shape

Microcalcifications

Solid internal structure

Extrathyroidal extension

Central vascularity

Low-risk features include purely cystic nodule, spongiform appearance, comet-tail artifact, and peripheral vascularity [22].

TIRADS Classification:

TIRADS Category	Malignancy Risk	Recommended Action
TR1 (Benign)	<2%	No FNAB
TR2 (Not suspicious)	<2%	No FNAB
TR3 (Mildly suspicious)	5%	FNAB if ≥2.5cm
TR4 (Moderately suspicious)	10-20%	FNAB if ≥1.5cm
TR5 (Highly suspicious)	>35%	FNAB if ≥1cm

5. Bethesda System for Reporting Thyroid Cytopathology

Table 2. Bethesda classification and management recommendations

Bethesda Category	Interpretation	Malignancy Risk	Recommended Action
Category I	Non-diagnostic	5-10%	Repeat FNAB

Bethesda Category	Interpretation	Malignancy Risk	Recommended Action
Category II	Benign	0-3%	Clinical follow-up with periodic ultrasound
Category III	AUS/FLUS	10-30%	Repeat FNAB, molecular testing, or lobectomy
Category IV	Follicular neoplasm	25-40%	Molecular testing or lobectomy
Category V	Suspicious for malignancy	50-75%	Surgery
Category VI	Malignant	97-99%	Surgery

Limitations of FNAB: Diagnostic accuracy ranges from 70-97%, depending on operator and pathologist skill. Of samples, 17-20% are deemed diagnostically inadequate. FNAB cannot detect capsular or vascular invasion in follicular carcinoma; therefore, definitive diagnosis requires postsurgical pathology [23,24].

6. Molecular Testing

Molecular testing is typically applied to Bethesda III and IV categories [25].

Current molecular diagnostic approaches:

Test	Sensitivity	Specificity	NPV	PPV
7-gene mutation panel	75%	97%	90%	92%
167-gene expression classifier	91%	68%	94%	51%
ThyroSeq v3 (multi-gene)	94%	82%	96%	68%

The 7-gene panel detects BRAF V600E, HRAS codon 61, KRAS codons 12/13, NRAS codon 61, RET/PTC1, RET/PTC3, and PAX8/PPARγ [26].

7. Postoperative Risk Stratification

ATA risk stratification system classifies patients into low, intermediate, or high risk based on tumor size, histological type, vascular or lymph node involvement, local invasion, distant metastases, resection extent, and postoperative thyroglobulin levels [27].

TNM-AJCC system (8th edition): Considers tumor size, presence and extent of extrathyroidal invasion, number of nodal metastases, and presence of distant metastases. Age at diagnosis is a significant prognostic factor: patients diagnosed before age 55 are staged at most as stage II [28].

8. Medullary Thyroid Carcinoma

Surgical therapy (total thyroidectomy with resection of local and regional metastases) is the primary treatment [29]. Prophylactic central lymph node dissection should be performed during total thyroidectomy in patients without preoperative evidence of cervical lymph node metastases.

Key points:

Serum calcitonin and carcinoembryonic antigen should be measured

Radioactive iodine ablation and TSH suppression are NOT relevant due to non-follicular origin

Kinase inhibitors (RET-specific inhibitors for RET mutation-positive patients) are beneficial for refractory disease [30]

9. Anaplastic Thyroid Carcinoma

BRAF V600E mutation testing and staging are performed. Resectable disease is surgically removed, followed by targeted BRAF kinase inhibitors in BRAF V600E-mutated patients [31]. Other patients receive postoperative radiotherapy and cytotoxic chemotherapy.

Prognosis: Very poor (low 1-year survival), as distant metastases develop rapidly and local invasion often makes the tumor unresectable [32].

10. Radioactive Iodine (RAI) and Management of Persistent Disease

RAI uses oral iodine-131 or iodine-123 to assess thyroid function. For recurrent minimal iodine-avid disease, RAI ablation is preferred [33]. Invasive neck disease requires surgical resection. Radiofrequency ablation has been used for small distant metastases (bone or lung) [34].

11. Dynamic Risk Stratification

Following initial postoperative risk stratification, patients are re-stratified into one of four clinical response categories at each follow-up visit [35]:

Response Category	Definition	Management
Excellent response	No evidence of disease	Reduced follow-up frequency

Response Category	Definition	Management
Biochemical incomplete	Elevated thyroglobulin	Monitor, consider additional therapy
Structural incomplete	Structural disease	Active treatment
Indeterminate response	Non-specific findings	Further testing

Monitoring schedule:

First year: Thyroid ultrasound every 6-12 months

TSH/thyroglobulin every 3-6 months

High-risk patients require additional imaging (CT, MRI, FDG-PET)

Discussion

This systematic review demonstrates that thyroid carcinoma remains a clinically significant pathology, with increasing diagnostic rates driven by improved imaging technologies.

Key Findings

Genetic Landscape: The identification of specific genetic mutations represents a major advancement. The MAPK signaling pathway, particularly BRAF V600E in papillary carcinoma (29-69%) and RAS mutations in follicular carcinoma (40-50%), provides diagnostic targets and therapeutic opportunities [36].

Diagnostic Algorithm: The Bethesda System provides a standardized framework that has improved communication between clinicians and pathologists. However, persistent challenges remain: 17-20% of FNAB samples are diagnostically inadequate, and the procedure cannot diagnose follicular carcinoma definitively [37].

Molecular Testing Impact: Multi-gene panels (e.g., ThyroSeq v3) achieve >90% negative predictive value, meaning a negative test effectively rules out malignancy. This can safely avoid diagnostic surgery in Bethesda III-IV nodules [38].

Risk Stratification: The coexistence of ATA (predicting recurrence) and TNM-AJCC (predicting mortality) systems reflects the multidimensional nature of thyroid carcinoma management [39].

Limitations

This review has several limitations:

Heterogeneity in ultrasound criteria across studies

Molecular testing not universally available

Potential publication bias

Most studies from high-income countries

Clinical Implications

For Bethesda III-IV nodules, molecular testing with high NPV (≥94%) can safely avoid diagnostic surgery in benign cases [40]. This reduces unnecessary thyroidectomies and associated complications.

Conclusion

Thyroid carcinoma remains a significant endocrine malignancy where early diagnosis directly determines patient outcomes. The integration of high-resolution ultrasound, the Bethesda System for cytopathology reporting, and molecular testing has substantially improved diagnostic accuracy.

Recommendations:

Universal adoption of standardized TIRADS and Bethesda reporting

Expansion of molecular testing access for Bethesda III-IV nodules

Dynamic risk-adapted follow-up rather than static staging

Hereditary screening programs for medullary carcinoma (RET mutations)

Investment in anaplastic carcinoma research

Statements

Author Contribution

Absamatov Abror Sherzod o'g'li: Conceptualization, literature search and review, data extraction and synthesis, manuscript writing and drafting, revision and editing, final approval of the version to be published.

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The author declares no conflicts of interest.

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Ethical Approval

Not required. This research is exclusively based on published literature.

AI Statement

During the preparation of this study, AI tools were used only for reference formatting and grammar checking. No AI tools were used for content generation or scientific writing.

Data Sharing Statement

All data extracted from published articles are included in this manuscript. Full reference list is available.

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