Revista Española de Cirugía Oral y Maxilofacial
Publicación Oficial de la SECOM CyC Sociedad Española de Cirugía Oral y Maxilofacial y de Cabeza y Cuello

Artículo Aceptado para su pre-publicación / Article Accepted for pre-publication

Título / Title:
Espectro clínico del síndrome de hiperparatiroidismo-tumor mandibular: revisión exploratoria / Clinical Spectrum of Hyperparathyroidism–Jaw Tumor Syndrome: Scoping Review

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DOI: 10.20986/recom.2026.1758/2026

Instrucciones de citación para el artículo / Citation instructions for the article:
Berríos Juan Pablo, Matus-Miranda Gustavo , Padilla María, Tapia Pedro, Puente Benjamín. Espectro clínico del síndrome de hiperparatiroidismo-tumor mandibular: revisión exploratoria / Clinical Spectrum of Hyperparathyroidism–Jaw Tumor Syndrome: Scoping Review. j.maxilo 2026. doi: 10.20986/recom.2026.1758/2026.

INSPIRA NETWORK

CLINICAL SPECTRUM OF HYPERPARATHYROIDISM–JAW TUMOR SYNDROME: SCOPING REVIEW

ESPECTRO CLÍNICO DEL SÍNDROME DE HIPERPARATIROIDISMO-TUMOR MANDIBULAR: REVISIÓN EXPLORATORIA

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Recibido: 19-04-2026

Aceptado: 07-07-2026

ABSTRACT

Background: Hyperparathyroidism–jaw tumor (HPT-JT) syndrome is a rare autosomal dominant disorder caused by pathogenic variants in the *CDC73* tumor suppressor gene. It is characterized by pleiotropic manifestations, including primary hyperparathyroidism (PHPT), parathyroid neoplasms, fibro-osseous jaw lesions, and renal and uterine abnormalities, resulting in diagnostic challenges.

Objective: To comprehensively describe the clinical spectrum, laboratory findings, and imaging features of HPT-JT syndrome, with emphasis on maxillofacial manifestations, through a scoping review of the literature.

Methods: A PRISMA-ScR–guided scoping review of MEDLINE/PubMed was conducted, analyzing demographic characteristics, clinical manifestations, biochemical findings, imaging features, genetic testing results and organ involvement.

Results: 35 studies comprising 154 patients were included. Mean age at presentation was 30 years, with a slight female predominance. Elevated serum calcium and parathyroid hormone levels were present in over 95 % of cases. PHPT was common, often associated with parathyroid adenomas or carcinomas. Maxillofacial involvement, most commonly ossifying fibromas of the mandible and/or maxilla, was identified in approximately 25 % of patients. Renal and uterine manifestations were also frequently reported. Most cases had confirmed *CDC73* mutations.

Conclusions: Early recognition of maxillofacial manifestations combined with biochemical and genetic assessment is essential for accurate diagnosis and optimal management of HPT-JT syndrome.

Keywords: CDC73 protein human, hyperparathyroidism, jaw neoplasms.

RESUMEN

Antecedentes: El síndrome de hiperparatiroidismo-tumor mandibular (HPT-JT) es un trastorno autosómico dominante poco frecuente causado por variantes patogénicas en el gen supresor de tumores CDC73. Se caracteriza por manifestaciones pleiotrópicas, que incluyen hiperparatiroidismo primario (HPTP), neoplasias paratiroideas, lesiones fibroóseas mandibulares y anomalías renales y uterinas, lo que plantea dificultades diagnósticas.

Objetivo: Describir exhaustivamente el espectro clínico, los hallazgos de laboratorio y las características de imagen del síndrome HPT-JT, con énfasis en las manifestaciones maxilofaciales, mediante una revisión exploratoria de la literatura.

Métodos: Se realizó una revisión exploratoria guiada por PRISMA-ScR de MEDLINE/PubMed, analizando las características demográficas, las manifestaciones clínicas, los hallazgos bioquímicos, las características de imagen, los resultados de las pruebas genéticas y la afectación orgánica.

Resultados: Se incluyeron 35 estudios con 154 pacientes. La edad media al momento del diagnóstico fue de 30 años, con un ligero predominio femenino. En más del 95 % de los casos se observaron niveles elevados de calcio sérico y hormona paratiroidea. El hiperparatiroidismo primario (HPTP) fue frecuente, a menudo asociado con adenomas o carcinomas paratiroideos. La afectación maxilofacial, principalmente fibromas osificantes de la mandíbula y/o el maxilar, se identificó en aproximadamente el 25 % de los pacientes. También se informaron con frecuencia manifestaciones renales y uterinas. La mayoría de los casos presentaban mutaciones confirmadas en el gen CDC73.

Conclusiones: El reconocimiento precoz de las manifestaciones maxilofaciales, junto con la evaluación bioquímica y genética, es fundamental para un diagnóstico preciso y un manejo óptimo del síndrome HPT-JT.

Palabras clave: Proteína CDC73 humana, hiperparatiroidismo, neoplasias mandibulares.

INTRODUCTION

Primary hyperparathyroidism (PHPT) is the third most common endocrine disorder and the leading cause of hypercalcemia in the outpatient setting[1]. Although robust epidemiological data are limited, it is estimated that approximately 10-15 % of PHPT cases correspond to hereditary forms of the disease[1].

Hyperparathyroidism-jaw tumor syndrome (HPT-JT) is a rare diagnostic entity and is considered an uncommon variant of familial PHPT[2]. It is inherited in an autosomal dominant pattern with incomplete penetrance[1-10] and is associated with pathogenic variants in the tumor suppressor gene *CDC73* (also known as *HRPT2*), which encodes the parafibromin protein[4,11,12]. Approximately 90 % of mutation carriers develop hyperparathyroidism secondary to parathyroid tumors[4].

HPT-JT syndrome was first described by Jackson in 1990, who reported multiple cases of hyperparathyroidism and mandibular tumors occurring across three generations of the same family, although the condition had been previously investigated by the same author in 1958[10,13]. The estimated prevalence of HPT-JT is approximately 12 % among patients suspected of having hereditary forms of PHPT[1], however, its true prevalence remains difficult to determine due to its rarity[3].

The absence of formal clinical guidelines, the scarcity of data and the variable phenotypic expression of the syndrome make diagnosis particularly challenging[3]. HPT-JT encompasses multiple pleiotropic manifestations, including PHPT associated with parathyroid adenomas or carcinomas, fibro-osseous tumors of the mandible and maxilla, as well as renal and uterine pathologies[2,3]. It is estimated that approximately 35 % of affected individuals may develop ossifying fibromas of the jaws[4].

When HPT-JT syndrome is suspected, genetic testing is considered the gold standard for diagnosis and should be offered to all potentially affected family members[5]. Surgical intervention represents the mainstay of treatment for PHPT[3]. Ossifying fibromas of the jaws often require surgical management to prevent functional impairment and local destruction[3].

Given the rarity of this syndrome and its clinical relevance for oral and maxillofacial surgeons, the aim of this review is to describe the spectrum of clinical manifestations and laboratory and imaging findings associated with HPT-JT syndrome, with particular emphasis on maxillofacial involvement, through a scoping review of the literature.

MATERIALS AND METHODS

Study design

This scoping review was conducted in accordance with the PRISMA-ScR recommendations[14]. The research question addressed was: *What is the reported spectrum and frequency of clinical manifestations,* laboratory abnormalities and imaging findings *associated with Hyperparathyroidism–jaw tumor syndrome in the scientific literature?*

Eligibility criteria

Eligible studies included full-text articles, without language restriction, reporting patients diagnosed with HPT-JT syndrome, with or without maxillofacial tumor involvement. Cohort studies, prospective and retrospective observational studies, clinical studies (randomized or non-randomized), case series, and case reports published within the last 20 years were included.

Animal studies, narrative reviews, systematic reviews, and in vitro studies were excluded.

Information sources and search strategy

The MEDLINE/PubMed database was searched to identify potentially relevant articles. MEDLINE/PubMed was selected as the sole electronic database as it provides comprehensive coverage of the peer-reviewed biomedical literature and is considered the principal source for publications on rare endocrine and maxillofacial disorders. Given the rarity of HPT-JT syndrome and the predominance of case reports and small observational studies, this database was considered sufficient to capture the majority of clinically relevant evidence. To reduce the likelihood of missing eligible studies, a manual search of the reference lists of all included articles was also performed. The literature search was independently conducted by two authors (G.M.M. and B.P.M.) between December 1 and December 8, 2025, using the following terms: "CDC73 protein, human", "Hyperparathyroidism", and "Jaw Neoplasms".

Study selection

Article selection was independently carried out by two reviewers (G.M.M. and J.B.B.). Titles and abstracts were screened, and potentially eligible articles underwent full-text review. Disagreements were resolved through discussion and consensus, with the involvement of a third reviewer when necessary.

Data extraction and synthesis

Data extracted included demographic characteristics, sex distribution, biochemical findings (serum calcium and parathyroid hormone levels), parathyroid, maxillofacial gynecological and renal manifestations. Data were tabulated and analyzed descriptively.

Risk of bias

In accordance with PRISMA-ScR[14] recommendations, no formal risk-of-bias assessment was performed, as the objective of this review was to map available evidence rather than evaluate comparative outcomes.

RESULTS

The initial search identified 243 potentially relevant articles. After title and abstract screening, 48 studies were selected for full-text evaluation. Following application of the inclusion and exclusion criteria, 35 studies were included in the final analysis (Figure 1).

Table I summarizes the characteristics of the studies included in this scoping review, including author, year of publication, country of origin, study design, number of patients, age, and sex distribution. Of the included articles, 29 were case reports[2,3,5-8,10-12,15-34], five were retrospective cohort studies[1,4,35-37], and one was a case series[13]. 13 studies originated from the United States[1,3-5,11,12,17,19-21,26,29,32], three from Italy[18,31,35], two from France[22,36], two from India[7,16], two from Australia[13,30], two from Spain[28,34], and one each from Canada[2], Sri Lanka[6], Japan[23], China[8], Romania[24], Tunisia[25], Saudi Arabia[10], Portugal[37], Denmark[15], Germany[27], and South Korea[33] (Table I).

A total of 154 patients were reported across the included studies. The mean age at diagnosis was 30 years, ranging from 11 to 71 years. A slight female predominance was observed, with 79 females and 74 males; one study did not report patient age[12], and another did not report sex distribution[34].

Reported clinical manifestations were heterogeneous and included facial swelling and asymmetry, trismus, dysphagia, respiratory symptoms, fatigue, gastrointestinal complaints, neurocognitive symptoms, polyuria, bone pain, pathological fractures, and gynecological symptoms such as menorrhagia.

Table II presents the characteristics reported in each study, including the number of patients, sex, laboratory findings, and clinical manifestations. The reported laboratory parameters included serum calcium (Ca) and parathyroid hormone (PTH). Detailed data are provided regarding serum calcium and PTH levels, the presence of PHPT, and associated parathyroid, maxillofacial, gynecological, and renal findings. This table highlights the clinical heterogeneity of HPT-JT syndrome and the variability of organ involvement described in the literature.

The percentage distribution of the main findings associated with HPT-JT syndrome is shown in Table III. At the parathyroid level, adenomas were the most frequently reported finding, followed by parathyroid carcinomas. Regarding maxillofacial involvement, ossifying fibroma

was the most commonly reported lesion. Uterine pathologies were the most frequent gynecological abnormalities. With respect to renal involvement, nephrolithiasis was the most prevalent finding.

Biochemical evaluation revealed elevated serum calcium levels in 95.4 % of patients in whom this parameter was reported, and elevated parathyroid hormone levels in 98.9 %. PHPT was present in 90.2 % of patients, with parathyroid involvement being the most frequent manifestation. Maxillofacial involvement was detected in 24.2 % of patients, while gynecological and renal abnormalities were observed in 18.5 % and 18.4 % of patients, respectively. All of these findings are detailed in Table IV.

Imaging modalities included 99mTc-sestamibi scintigraphy, cervical ultrasound, computed tomography, magnetic resonance imaging, panoramic radiography, renal and pelvic ultrasound, and bone densitometry. Genetic testing for *CDC73* mutations was performed in 31 studies, while four studies[3,10,16,24] did not include genetic confirmation.

DISCUSSION

HPT-JT syndrome represents a rare form of multiple endocrine neoplasia characterized by PHPT caused by single or multiple parathyroid tumors, fibro-osseous jaw lesions, renal abnormalities, and uterine tumors[5,15,26]. The syndrome follows an autosomal dominant inheritance pattern with variable and incomplete penetrance, with up to 10% of mutation carriers remaining asymptomatic[15,28]. This variability is consistent with the findings of the present review, in which a subset of patients did not exhibit overt clinical manifestations at diagnosis. The findings of this scoping review provide a framework for contextualizing the clinical variability of the syndrome based on the available evidence.

The syndrome typically manifests during late adolescence or early adulthood. No definitive sex predilection has been established. While some studies report a male predominance[2,26,28], others suggest a higher prevalence in females[9,37] or an equal sex distribution[36]. In the present review, a slight female predominance was observed; however, no definitive conclusion can be drawn due to the heterogeneity of the available evidence and the predominance of case reports and small series.

PHPT represents the main manifestation of the syndrome[10,28,35] and has been reported in more than 95% of mutation carriers[10,35], predominantly caused by solitary parathyroid

adenomas[10], thereby conferring a significant risk of morbidity and mortality due to severe hypercalcemia[2]. In the present review, 90.2 % of patients affected by the syndrome were found to have PHPT.

The pathogenesis of HPT-JT syndrome is linked to germline mutations in *CDC73*, a gene composed of 17 exons[5,16,26,38] that encodes the tumor suppressor protein parafibromin[5,16,26]. Most pathogenic variants result in truncated or nonfunctional parafibromin, leading to dysregulated cellular proliferation[26]. Loss of parafibromin expression has been consistently demonstrated in tumors associated with the syndrome[33,37,38].

The initial mutation in HPT-JT is usually inherited from one of the parents or, in rare cases, arises *de novo* during embryonic development[38]. Genetic counselors estimate that each child of an affected individual has a 50 % likelihood of inheriting a clinically significant mutation[11].

Maxillofacial manifestations, although eponymous, are present in only approximately one-third of affected individuals[22,24,38]. Ossifying fibromas of the mandible and maxilla are the most characteristic lesions and were first described and associated with familial hyperparathyroidism by Kennett and Pollick in 1971[13,25]. Often appear earlier in life compared with sporadic jaw tumors[25] and fewer than 0.5 % carry a risk of malignant transformation[11,22]. These lesions may be multifocal or bilateral and can cause significant functional impairment, often requiring surgical resection[11]. Recurrence following surgery has been reported, highlighting the need for long-term follow-up[25]. Radiographically, non-syndromic ossifying fibromas are commonly described as mixed radiolucent–radiopaque lesions; however, in syndromic cases, they typically appear as radiolucent lesions[25].

The main differential diagnoses include osteolytic brown tumors associated with hyperparathyroidism[3]; however, these entities are histologically distinct[22,28]. Furthermore, management of HPT-JT–associated ossifying fibromas involves surgical resection of the affected mandibular region[10,11].

Renal involvement occurs in approximately 15-20 % of patients and includes kidney stones, cystic kidney disease, chronic kidney disease and, less commonly, malignant stromal neoplasms[7,15,17,28,38]. This proportion of renal involvement is consistent with the findings of the present review, which identified renal manifestations in 16.8% of patients with HPT-JT syndrome.

Beyond the manifestations described above, uterine tumors have been consistently reported in association with HPT-JT syndrome and constitute the most frequent extracervical clinical feature after PHPT, affecting more than 50 % of patients[19,21,22,38]. Several studies, including those by Mathews et al. and Bellido et al., have reported frequencies as high as 75 % among affected females[26,28]. In the present review, uterine involvement was observed in 62.02 % of women with HPT-JT, falling within the range previously described in the literature. Clinically, menorrhagia represents the most common presenting symptom, and affected women may also exhibit a history of spontaneous abortions[21,38] and reduced fertility[38], underscoring the significant reproductive impact of the syndrome. Reported uterine lesions include a broad spectrum of benign and malignant entities, such as adenomyosis, adenofibromas, leiomyomas, endometrial hyperplasia, adenosarcomas, and tumors of Müllerian duct origin[22,38]. From a clinical management perspective, women carrying HPT-JT should undergo regular gynecological assessment beginning at menarche when clinically indicated, as early detection may influence therapeutic decision-making and reproductive counseling[4,21]. However, the absence of standardized surveillance and treatment guidelines represents a major clinical gap[21,38]. Given the high prevalence and clinical relevance of uterine involvement, its inclusion among the diagnostic criteria for HPT-JT, as suggested by Garrigues et al., warrants serious consideration[22].

Physiologically, the parathyroid glands tightly regulate extracellular calcium concentrations through circulating parathyroid hormone (PTH), which stimulates osteoclast-mediated bone resorption and mobilization of calcium from the skeleton[17]. Consequently, an elevated plasma concentration of intact PTH in the presence of hypercalcemia is indicative of PHPT[17,24]. Following clinical suspicion, laboratory evaluation is mandatory for the diagnosis of PHPT, with serum calcium and PTH levels representing the most relevant biochemical tests[11]. In addition, imaging studies play a crucial role in identifying both parathyroid pathology and other syndrome-associated manifestations. These include technetium-99m sestamibi scintigraphy, which demonstrates a diagnostic accuracy exceeding 90 % for the detection of ectopic parathyroid adenomas, single-photon emission computed tomography (SPECT)[25], parathyroid ultrasonography[38], panoramic radiography of the mandible, and renal and uterine ultrasonography and computed tomography with and without contrast[31].

In HPT associated with HPT-JT syndrome, the recommended therapeutic approach is parathyroidectomy, involving resection of macroscopically enlarged parathyroid glands, unless parathyroid carcinoma is suspected[25]. However, Guarnieri et al. have reported that subtotal parathyroidectomy should be considered due to multiglandular involvement, recurrence and persistence rates, surgical complexity, and the associated risk of parathyroid carcinoma[31]. Furthermore, Mehta et al. and Rekik et al. have advocated bilateral neck exploration as the initial surgical strategy in all patients[4,25]. Surgical success can be assessed intraoperatively by a decrease of more than 50 % in PTH levels measured 5 to 10 minutes after gland excision, confirming adequate removal of hyperfunctioning parathyroid tissue[24,25]. Finally, timely referral to appropriate medical and surgical specialists is essential to evaluate and manage potential multiorgan involvement associated with the syndrome.

It should be emphasized that genetic testing remains the gold standard for definitive diagnosis and enables early identification of affected family members[3,23,38]. Early diagnosis facilitates appropriate surgical intervention and coordinated multidisciplinary surveillance for both affected patients and at-risk family members. In this context, the oral and maxillofacial surgeon plays a pivotal role in the early recognition of the syndrome, as mandibular ossifying fibromas may represent the first clinical manifestation prompting diagnostic suspicion. Awareness of this association allows the health care professional to initiate timely referral for genetic testing, biochemical evaluation, and multidisciplinary assessment, thereby facilitating early diagnosis and coordinated management. Systematic follow-up is essential to monitor for the development of mandibular tumors, as well as associated renal and uterine lesions, underscoring the importance of close collaboration between maxillofacial surgeons, endocrinologists, geneticists, and gynecologists.

Several limitations should be considered when interpreting the findings of this review. Most of the available evidence consisted of case reports and small case series, with only a limited number of retrospective cohort studies. Consequently, the reported frequencies of clinical manifestations should be interpreted as descriptive findings derived from the published literature rather than as accurate estimates of their prevalence in patients with HPT-JT syndrome. In addition, as this was a scoping review intended to map the available evidence, no formal risk-of-bias assessment or quantitative synthesis was performed. Therefore, the results should be interpreted with caution and considered an overview of the current

evidence rather than definitive epidemiological estimates.

CONCLUSION

HPT-JT syndrome is a clinically heterogeneous condition that requires a high level of diagnostic awareness. Genetic testing is essential for definitive diagnosis and should be pursued early. Moreover, when a pathogenic result is identified in an affected patient, genetic testing should be extended to family members. Early recognition allows timely and effective treatment and facilitates surveillance for the multiple manifestations associated with the syndrome. Management should be multidisciplinary, involving endocrinologists, surgeons, geneticists, and oral and maxillofacial specialists to optimize patient outcomes.

FUNDING

No funding to declare.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

AUTHORS' CONTRIBUTIONS

Study Design: Gustavo Matus-Miranda, Benjamín Puente, Pedro Tapia.
Literature Review: Gustavo Matus-Miranda, Benjamín Puente.
Article Selection and Data Analysis: Gustavo Matus-Miranda, María Padilla-Ruiz, Juan Berríos-Bugueño,
Manuscript Writing: Gustavo Matus-Miranda, María Padilla-Ruiz, Juan Berríos-Bugueño.
Manuscript Supervision: Gustavo Matus-Miranda, Pedro Tapia.
All authors participated in the final approval of the manuscript

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Figure 1. PRISMA flow diagram for scoping reviews.

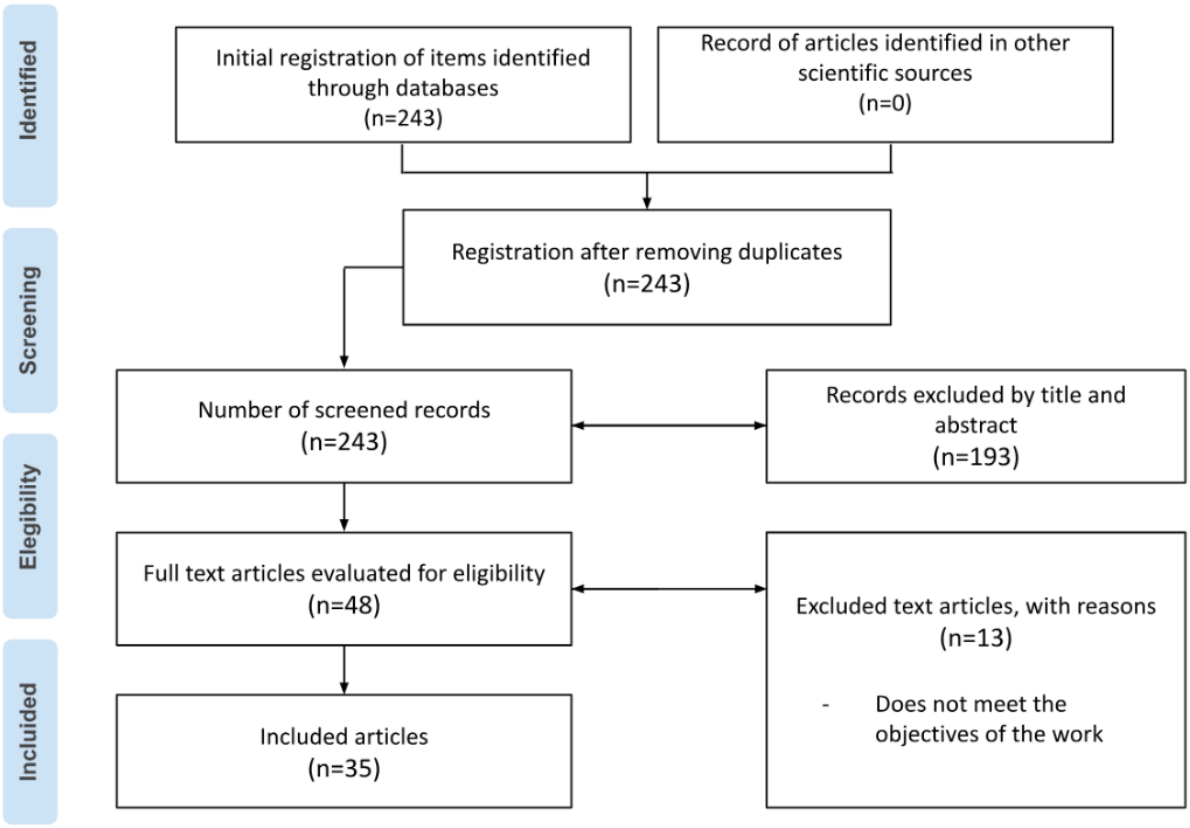

Table I. Demographic and methodological characteristics of the included studies.

	Author	Year	Country	Type of Study	Number of Patients	Age/Average Age*	Sex	
							Male	Female
1	Danda *et al.*	2023	India	C.R	1	21	-	1
2	Kang *et al.*	2023	U.S.A.	C.R	1	60	1	-
3	Tora *et al.*	2023	U.S.A.	R.C	61	39*	34	27
4	Yang *et al.*	2022	China	C.R	1	32	-	1
5	Garrigues *et al.*	2022	France	C.R	1	28	-	1
6	Khan *et al.*	2022	U.S.A.	C.R	1	42	-	1
7	Le Collen *et al.*	2021	France	R.C	13	37*	7	6
8	Weaver *et al.*	2021	U.S.A.	C.R	1	23	-	1
9	Wijewickrama *et al.*	2020	Sri Lanka	C.R	1	22	-	1
10	Grigorie *et al.*	2019	Italy	C.R	1	35	-	1
11	Sirbiladze *et al.*	2019	U.S.A.	C.R	1	31	-	1
12	Iacobone *et al.*	2017	Italy	R.C	20	37.5*	7	13
13	Guarnieri *et al.*	2017	Italy	C.R	1	48	-	1
14	Satpathy *et al.*	2017	India	C.R	1	15	-	1
15	Dhas *et al.*	2017	Saudi Arabia	C.R	1	23	-	1
16	Koikawa *et al.*	2017	Japan	C.R	1	20	-	1
17	Rubinstein *et al.*	2016	U.S.A.	C.R.	1	32	-	1
18	Bellido *et al.*	2016	Spain	C.R	1	23	-	1
19	Mele *et al.*	2016	Denmark	C.R	1	41	-	1
20	Mathews *et al.*	2016	U.S.A	C.R	1	19	-	1
21	Piciu *et al.*	2016	Romania	C.R	1	33	-	1
22	Mehta *et al.*	2014	U.S.A.	R.C	16	30.7*	10	6
23	Parfitt *et al.*	2014	U.S.A.	C.R	1	NR	1	-
24	Wofl *et al.*	2012	U.S.A.	C.R	1	26	-	1
25	Kutcher *et al.*	2011	Canada	C.R	1	22	1	-
26	Rekik *et al.*	2010	Tunisia	C.R	1	23	-	1
27	Veiguela *et al.*	2010	Spain	C.R	1	27	1	-
28	Panicker *et al.*	2010	U.S.A	C.R	1	25	N.R	N.R
29	Schmidt *et al.*	2009	U.S.A.	C.R	1	37	1	-
30	Carlson *et al.*	2009	U.S.A.	C.R	1	22	1	-
31	Raue *et al.*	2007	Germany	C.R	1	29	1	-
32	Moon *et al.*	2005	South Korea	C.R	2	34*	2	-
33	Aldred *et al.*	2005	Australia	C.S	3	35*	2	1
34	Howell *et al.*	2004	Australia	C.R	1	16	1	-
35	Cavaco *et al.*	2004	Portugal	R.C	11	37.8*	4	7
TOTAL					154	30*	74	79

(C.R: Case Report: R.C: Retrospective Cohort; C.S: Case Series; NR: Not Reported)

Table II. Clinical manifestations, laboratory findings and associated findings of HPT-JT syndrome by study.

	Author/Year	n	Sex		Laboratory Tests		PHPT	Findings Associated with HPT-JT			
			M	F	Ca (mg/dL) (8.5-10.5 mg/dL)	PTH (pg/mL) (10-65 pg/mL)		Parathyroid	Jaw	Gynecological	Renal
1	Danda *et al. 2023*	1	-	1	15.1	1697	Yes	P.A	N.F	N.F	. Nephrolithiasis . Bilateral cysts . Bilateral medullary nephrocalcinosis . Ectopic kidney
2	Kang *et al. 2023*	1	1	-	8.9	118.6	Yes	P.A	O.F	N.A	N.R
3	Tora *et al. 2023*	61	34	27	N.R	N.R	Yes (55)	P.A (36), P.C (17), A.P.A (2)	O.F (7)	· Myoma (5) · Uterine fibroids (5) · Cervical polyps (4) · Uterine polyps (2) · Uterine fibroids (1)	· N.S Kidney Tu (4)
4	Yang *et al. 2022*	1	-	1	8.94	25	No	N.F	O.F	Menorrhagia with anemia Endometrial hyperplasia	· Kidney cysts
5	Garrigues *et al. 2022*	1	-	1	10.7	662	Yes	P.A	N.F	N.F	N.F
6	Khan *et al. 2022*	1	-	1	14	338	Yes	P.A	N.S Tu.	N.R	· Nephrolithiasis · Cortical cysts
7	Le Collen *et al. 2021*	13	7	6	12.5*	406*	Yes (7)	P.A (7)	O.F (2)	· Uterin polyp (1)	· Nephrolithiasis (3) · Chronic Renal Failure (1)
8	Weaver *et al. 2021*	1	-	1	11.7	192	Yes	P.A	N.F	· Bilateral ovarian cysts · Uterine polyp	N.R
9	Wijewickrama *et al. 2020*	1	-	1	14.3	1025	Yes	P.A	Fibrous dysplasia	N.F.	Nephroblastoma
10	Grigorie *et al. 2019*	1	-	1	13.9	327	Yes	P.C	O.F	· Uterine fibroid	· Nephrolithiasis
11	Sirbiladze *et al. 2019*	1	-	1	10.8	107	Yes	P.A	N.R	· Ovarian granulosa cell Tu	N.R

(*: Average Value, P.A: Parathyroid Adenoma, N.F: No Findings, O.F: Ossifying Fibroma, N.A: Not Applicable, N.R: Not Reported, P.C: Parathyroid Carcinoma, A.P.A: Atypical Parathyroid Adenoma; N.S: Not Specified, Tu: Tumor, C.G.C.G: Central Giant Cell Granuloma, C.O.F: Cemento-Ossifying Fibroma, C.P.A: Cystic Parathyroid Adenoma)

Table III. Findings associated with HPT-JT: percentage distribution.

Findings	n	%
Parathyroid	**136**	**100**
Parathyroid adenomas	97	71.3
Parathyroid carcinomas	31	22.8
Atypical parathyroid adenoma	6	4.4
Cystic parathyroid adenoma	2	1.5
Jaw	**36**	**100**
Ossifying fibroma	23	63.8
Ossifying cement fibroma	2	5.6
Central giant cell granuloma	2	5.6
Mixed tumor	1	2.8
Maxillary Fibrous dysplasia	1	2.8
Brown tumor of hyperparathyroidism	1	2.8
Unspecified maxillary tumor	6	16.6
Gynecological	**46**	**100**
Uterine	39	84.8
Ovarian	4	8.7
Tubal	1	2.2
Unspecified gynecologic tumor	2	4.3
Renal	**31**	**100**
Kidney stones	12	38.7
Cystic	5	16.1
Chronic kidney disease	2	6.5
Ectopic kidney	2	6.5
Neoplastic tumor	2	6.5
Medullary nephrocalcinosis	1	3.2
Unspecified renal tumor	7	22.5

Table IV. Percentage of patients affected.

Findings / Abnormalities	Total Patients Reported	Affected Patients (AP)	Percentage AP	Patients without alterations
Ca levels	87	83	95.4%	4
PTH levels	92	91	98.9%	1
HPTP	153	138	90.2%	15
Parathyroid findings	153	135	90.6%	18
Maxillary findings	149	36	24.2%	113
Gynecological findings	135	25	18.5%	110
Renal findings	136	25	18.4%	111

Ca: calcium. PTH: parathyroid Hormone. HPTP: hyperparathyroidism.