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Paraneoplastic Hypertrophic Osteoarthropathy in the Setting of Dual Primary Malignancies: Follicular Thyroid Carcinoma and Metastatic Colon Adenocarcinoma

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Abstract

Various malignancies have the potential to secrete differing cytokines and hormones, as well as cause a variety of unique pathologies. While observations have been made regarding diseases that cancers tend to produce, there are rare instances when malignancies can cause other associated illnesses. Hypertrophic osteoarthropathy (HOA) is one ailment that has a number of clinical symptoms, albeit the mechanism as to how some cancers cause this syndrome and not others is still in question. While it is more commonly seen as a paraneoplastic syndrome in lung cancers, there are very rare instances where it can be seen in other malignancies as well. In this report, we would like to present a 42-year-old patient with a past medical history of autoimmune disease who developed follicular thyroid carcinoma and metastatic adenocarcinoma of the colon, resulting in secondary HOA. Although thyroid malignancies have been seen with this paraneoplastic syndrome, this case is one of the few in which the possibility of metastatic adenocarcinoma of the colon could be associated with HOA as well.

Categories: Oncology, Pulmonology, Endocrinology/Diabetes/Metabolism
Keywords: colon adenocarcinoma, follicular thyroid carcinoma, hypertrophic pulmonary osteoarthropathy (hoa), metastatic colon adenocarcinoma, multiple primary malignancies, paraneoplastic syndrome

Introduction

Hypertrophic osteoarthropathy (HOA) is a syndrome that presents with digital clubbing, overgrowth of the periosteum of the tubular bones, arthralgias, joint effusion, and a variety of other symptoms. The condition exists in two forms, primary and secondary, with 95% of cases being the latter, and are caused by malignancies via paraneoplastic syndrome [1]. In fact, pulmonary malignancies are more commonly associated with this disease, with approximately 5% of lung cancer patients being affected [2]. However, there are instances where associations have been documented with other malignancies. Thyroid cancer, although a rare cause of secondary HOA, is one such malignancy. However, what is interesting is that very few, if any, cases have been documented regarding any link between adenocarcinoma of the colon and secondary HOA. In fact, only two studies have noted connections between juvenile and adenomatous polyposis and secondary HOA, making the occurrence of adenocarcinoma of the colon and secondary HOA an unlikely association [3,4].

As such, we would like to present an exceedingly rare presentation of a patient with two separate primary malignancies who developed secondary HOA via paraneoplastic syndrome. Although it is most likely that the patient has developed secondary HOA from his follicular thyroid carcinoma, since there has been a documented case of it previously [5]. The possibility that the patient could have developed the paraneoplastic syndrome from his adenocarcinoma of the colon, although unlikely, could not be ruled out since some studies have noted other related gastrointestinal tumors' association with secondary HOA [3,4]. However, it should be noted that there have been few, if any, documented cases of adenocarcinoma of the colon specifically causing secondary HOA. In short, the scant coexistence of the diseases is described, with the results of intervention assessed in this report and how the patient himself had improvement after proper treatment. An analysis regarding the presumptive mechanism of how these malignancies cause secondary HOA will also be discussed.

Case Presentation

A 42-year-old male with a past medical history significant for Hashimoto's thyroiditis, hyperparathyroidism, and primary hypogonadism on testosterone replacement therapy presented with progressive generalized fatigue, polyarthralgia, and exertional dyspnea persisting for four years. The patient reported that his symptoms began following an episode of acute respiratory distress syndrome (ARDS) secondary to COVID-

19 infection in 2020. Following hospital discharge in 2020, he required home oxygen therapy for several months, which was later discontinued after improvement in oxygen saturation; however, his dyspnea persisted. Physical examinations between 2020 and 2024 were done by various specialists and primary care physicians.

Rheumatologic and endocrine evaluation

Given his persistent polyarthralgia involving bilateral knees, ankles, and shoulders, he was referred to rheumatology and endocrinology clinics. Hydroxychloroquine and ibuprofen provided minimal relief. Examination in 2024 revealed digital clubbing with distal interphalangeal (DIP) joint swelling (Figure *1*), with no other significant physical exam findings noted.

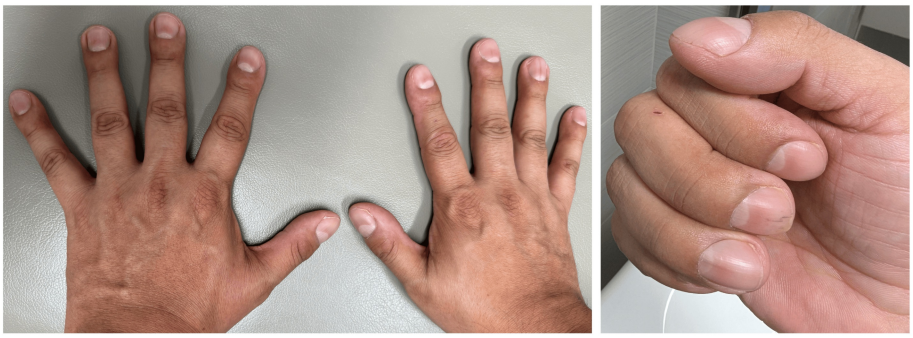


FIGURE 1: Clubbing of Hands

Endocrine workup (Table *1*) revealed biochemical euthyroidism with elevated thyroid peroxidase (TPO) antibodies, consistent with Hashimoto's thyroiditis, and elevated parathyroid hormone levels. Given the patient's symptoms suggestive of hypothyroidism and high TPO antibody titers, levothyroxine 50 µg daily was initiated.

Parameter	Value	Reference
Sodium	139 mmol/L	136 mmol/L - 145 mmol/L
Potassium	4.0 mmol/L	3.5 mmol/L - 5.1 mmol/L
Chloride	108 mmol/L	98 mmol/L - 107 mmol/L
Calcium (corrected)	9.2 mg/dL	8.5 mg/dL - 10.1 mg/dL
Magnesium	1.9 mg/dL	1.8 mg/dL - 2.4 mg/dL
Phosphorus	3.2 mg/dL	2.5 mg/dL - 4.9 mg/dL
Alanine transaminase	38 units/L	13 units/L - 61 units/L
Aspartate transaminase	21 units/L	15 units/L - 37 units/L
Total bilirubin	0.3 mg/dL	0 mg/dL - 1 mg/dL
Albumin	3.7 g/dL	3.4 g/dL - 5 g/dL
White blood cell count (WBC)	6.8 × 10^3/mcL	4.5 × 10^3/mcL - 11 × 10^3/mcL
Hemoglobin (Hgb)	14.2 × 10^3/mcL	13.2 g/dL - 17.4 g/dL
Mean corpuscular volume (MCV)	87.6 HI	80 HI - 98 HI
Platelet	362 × 10^3/mcL	150 x 10^3/mcL - 450 x 10^3/mcL
Neutrophil %	68.9%	50% - 75%
Lymphocyte %	20.7%	20% - 45%
Monocyte %	7.3%	2% - 12%
Eosinophil %	2.6%	< 6%

Absolute neutrophil	4.7 × 10^3/mcL	1.8 × 10^3/mcL - 7.7 × 10^3/mcL
Absolute lymphocyte	1.4 × 10^3/mcL	1.2 × 10^3/mcL - 4.5 × 10^3/mcL
Absolute monocyte	0.5 × 10^3/mcL	0.1 × 10^3/mcL - 1 × 10^3/mcL
Absolute eosinophil	0.2 × 10^3/mcL	<0.7 × 10^3/mcL
Cortisol a.m.	11.2 mcg/dL	4.3 mcg/dL - 22.4 mcg/dL
Prolactin	5 ng/mL	2 ng/mL - 18 ng/mL
Parathyroid	100 pg/mL	18 pg/mL - 88 pg/mL
T4 free	1 ng/dL	0.9 ng/dL - 1.8 ng/dL
Thyroid-stimulating hormone (TSH)	2.783 mIntUnit/mL	0.550 mIntUnit/mL - 4.780 mIntUnit/mL
Hemoglobin A1c	5%	4.2% - 6.3%
Cholesterol total	190 mg/dL	< 200 mg/dL
High-density lipoprotein (HDL)	45 mg/dL	> 40 mg/dL
Triglyceride	53 mg/dL	< 150 mg/dL
Low-density lipoprotein (LDL)	134 mg/dL	< 100 mg/dL
Alpha-fetoprotein (AFP)	3 ng/mL	< 8.0 ng/mL
Carcinoembryonic antigen (CEA)	0.9	N/A
Follicle-stimulating hormone (FSH)	3.5 mIntUnit/mL	1.0 mIntUnit/mL - 18 mIntUnit/mL
Luteinizing hormone (LH)	2 mIntUnit/mL	1.0 mIntUnit/mL - 9 mIntUnit/mL
Testosterone total	239 ng/dL	250 ng/dL - 1100 ng/dL
Testosterone free	36 pg/mL	46 pg/mL - 224 pg/mL
Beta-human chorionic gonadotropin (Beta-hCG)	< 1 IU/L	N/A
Testosterone bioavailable	66.2 ng/dL	110 ng/dL - 575 ng/dL
Thyroperoxidase antibody	> 900 IntlUnit/mL	< 9 IntlUnit/mL
Prostate-specific antigen (PSA) level total	1.0 ng/mL	N/A
PSA free	0.4 ng/mL	N/A

TABLE 1: Initial Blood Work

Laboratory results also demonstrated low testosterone with low-normal gonadotropins, consistent with primary hypogonadism, for which testosterone cypionate 50 mg intramuscular injection every two weeks was started, and the patient was compliant with treatment. Prostate-specific antigen and complete blood count were within normal limits.

Rheumatologic evaluation included radiographs of the upper extremities and an autoimmune panel (Table *2*). Imaging demonstrated bilateral HOA (Figure *2*), while autoimmune serologies were unremarkable. Hydroxychloroquine was discontinued, and conservative management with acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs) was advised.

Parameter	Value	Reference
Adrenocorticotropic hormone (ACTH)	12 pg/mL	6-50 pg/mL
Antinuclear antibody (ANA)	Negative	N/A
Antineutrophil cytoplasmic antibody (ANCA)	Negative	N/A
Cytoplasmic antineutrophil cytoplasmic antibody (C-ANCA)	Negative	N/A
Perinuclear antineutrophil cytoplasmic antibody (P-ANCA)	Negative	N/A
Carbohydrate antigen 19-9 (CA 19-9)	9 units/mL	<34 units/mL
Complement C3	161 mg/dL	82-185 mg/dL
Complement C4	34 mg/dL	15-53 mg/dL
CCP Ab IgG	<16 Units	N/A
DNA (DS) antibody Crithidia IFA	Negative	N/A
Sjogren's antibody A	<1.0	Negative
Sjogren's antibody B	<1.0	Negative
Smith antibody	<1.0	Negative

TABLE 2: Autoimmune Panel

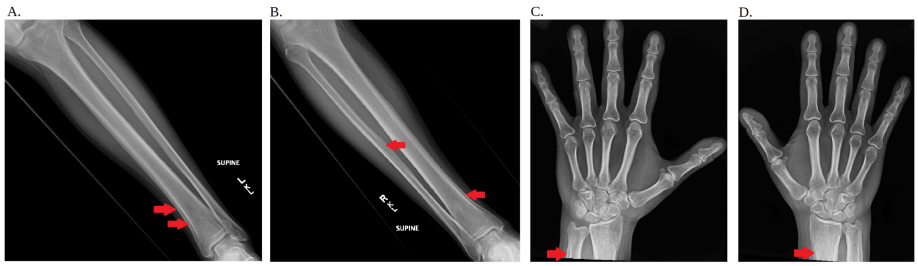


FIGURE 2: X-ray of Bilateral Hands and Arms

(A) X-ray imaging of the left arm shows tibial and fibular periosteal thickening which is indicated by the red arrows.

(B) X-ray imaging of the right arm shows tibial and fibular periosteal thickening which is indicated by the red arrows.

(C) X-ray imaging of the left hand shows distal left radial and ulnar periosteal thickening which is indicated by the red arrows.

(D) X-ray imaging of the right hand shows distal right radial and ulnar periosteal thickening which is indicated by the red arrows.

Pulmonary evaluation

Persistent dyspnea on exertion prompted a referral to pulmonology. A six-minute walk test, full pulmonary function testing, particularly spirometry (Table *3*), chest X-ray (Figure *3*), FeNO measurement, serum IgE, and an allergy panel were obtained. Results were largely unremarkable except for FeNO of 44 ppb, suggesting possible undiagnosed asthma. He was started on budesonide-formoterol 100/4.5 µg, two puffs twice daily, and albuterol as needed.

	Units	Predicted (Favor Mean)	Pre-drug Reported	Pre-drug % Predicted	Post-drug Reported	Post-drug % Predicted	% Change
FVC	L, btps	5.49	4.79	87	4.62	84	-4

FEV1	L, btps	4.51	3.76	83	3.64	81	-3
FEV1/FVC	%	82.59	78.39	95	78.93	96	1
FEF 25-75%	L/s	4.66	3.22	69	3.16	68	-2
FEF 75%	L/s	2.29	1.55	68	1.53	67	-2
FEF 50%	L/s	5.54	3.47	63	3.81	69	10
FEF 25%	L/s	9.11	7.91	87	7.89	87	0
FEF max	L/s	9.83	8.4	85	8.29	84	-1
MVV	L/min, btps	144.03	148.3	103	131.56	91	-11
Lung Volumes							
	Units	**Predicted (Favor Mean)**	**Pre-drug Reported**	**Pre-drug % Predicted**		**Units**	**Predicted (Favor Mean)**
VTG	L, btps	-	2.79	-	VTG	L, btps	-
VC	L, btps	5.49	4.44	81	VC	L, btps	5.49
TLC	L, btps	7.44	6.07	82	TLC	L, btps	7.44
RV	L, btps	2.15	1.63	76	RV	L, btps	2.15
FRC N2	L, btps	3.8	-	-	FRC N2	L, btps	3.80
FRC PL	L, btps	3.8	2.14	56	FRC PL	L, btps	3.80
ERV	L, btps	1.65	0.51	31	ERV	L, btps	1.65
IC	L, btps	3.64	3.93	108	IC	L, btps	3.64
Diffusion							
	Units	**Predicted (Favor Mean)**	**Pre-drug Reported**	**Pre-drug % Predicted**		**Units**	**Predicted (Favor Mean)**
Dsb	ml/min/mmHg, stpd	31.02	22.96	74	Dsb	ml/min/mmHg, stpd	31.02
D/VAsb	ml/min/mmHg/L, stpd	4.17	3.47	83	D/VAsb	ml/min/mmHg/L, stpd	4.17
Vinsp	L	-	5.06	-	Vinsp	L	-
DsbHb	ml/min/mmHg, stpd	31.02	22.98	74	DsbHb	ml/min/mmHg, stpd	31.02
SBO2 DN2	%	-	-	-	SBO2 DN2	%	-
SBO2 CV /VC	%	-	-	-	SBO2 CV /VC	%	-
SBO2 Vds	L, btps	-	-	-	SBO2 Vds	L, btps	-
Resistance							
	Units	**Predicted (Favor Mean)**	**Pre-drug Reported**			**Units**	**Predicted (Favor Mean)**
RAW	cmH2O/L/s	0.20-2.50	3.84	RAW	RAW	cmH2O/L/s	0.20-2.50
SGaw	L/s/cmH2O	-	0.1	SGaw	SGaw	L/s/cmH2O	-

TABLE 3: Spirometry

FVC: Forced vital capacity; FEV1: Forced expiratory volume in 1 second; FEF: Forced expiratory flow; MVV: Maximum voluntary ventilation; VTG: Thoracic gas volume; VC: Vital capacity; TLC: Total lung capacity; RV: Residual volume; FRC: Functional residual capacity; ERV: Expiratory reserve volume; IC: Inspiratory capacity; Dsb: Diffusing capacity for carbon monoxide; D/VAsb: Diffusing capacity for carbon monoxide per unit of alveolar volume;

Vinsp: Inspired volume; DsbHb: Diffusing capacity for carbon monoxide corrected for hemoglobin; RAW: Airway resistance; SGaw: Specific airway conductance; STPD: Standard temperature and pressure, dry; FeNO: Fractional exhaled nitric oxide

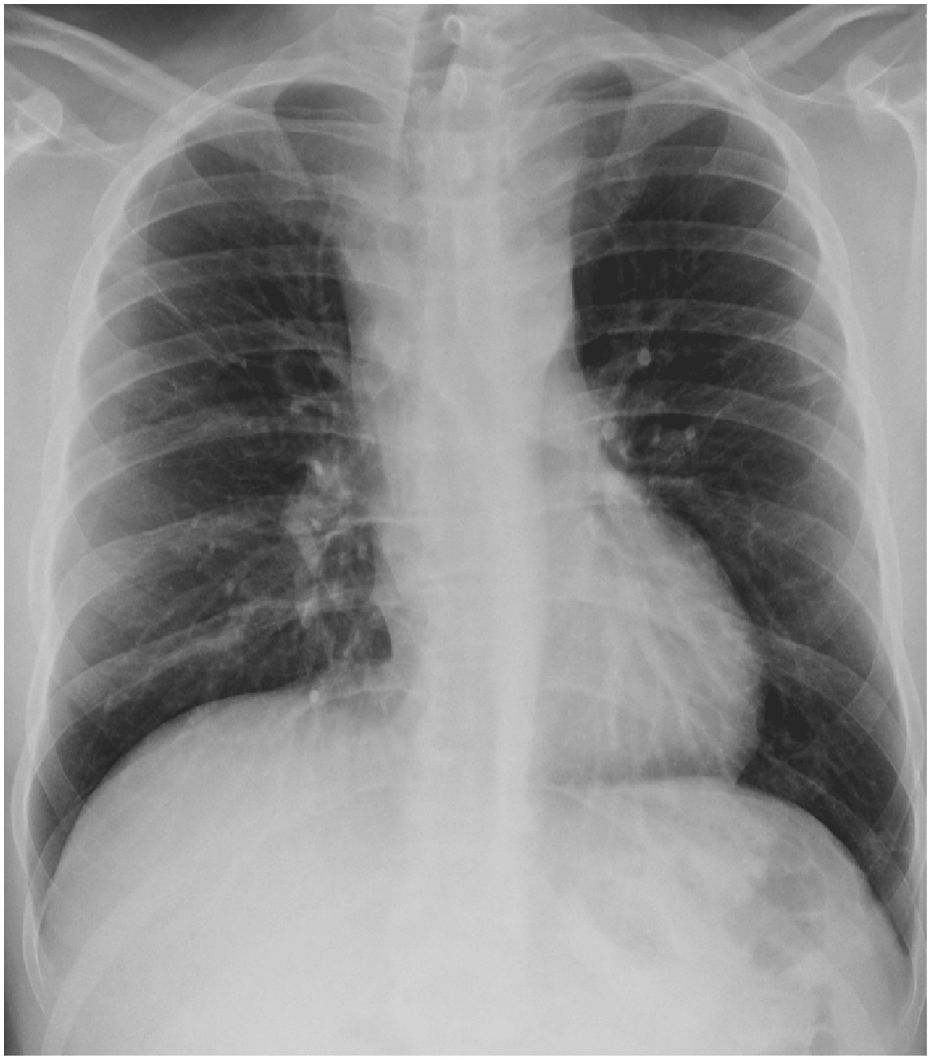


FIGURE 3: Chest X-ray

Mild atelectasis or scarring in the bilateral mid lungs but no new focal consolidation.

Although his dyspnea improved modestly, symptoms persisted, prompting a CT of the chest without contrast, which revealed a heterogeneous oval mass at the left thoracic inlet.

Subsequent imaging and diagnostic workup

Given the patient's known Hashimoto's thyroiditis, a neck CT was obtained, confirming a mass at the thoracic inlet (Figure *4*). A CT abdomen and pelvis revealed a hypoattenuating right hepatic lesion and focal wall thickening of the distal sigmoid colon, concerning for adenocarcinoma (Figures *5A*-*5B*). An MRI of the liver showed a heterogeneous T1 hypointense, T2 slightly hyperintense lesion (Figure *6*). Colonoscopy demonstrated a fungating mass in the distal sigmoid colon. A liver biopsy was done to ensure the patient didn't have another underlying primary hepatic malignancy. The results of the liver biopsy confirmed metastatic adenocarcinoma of the colon.

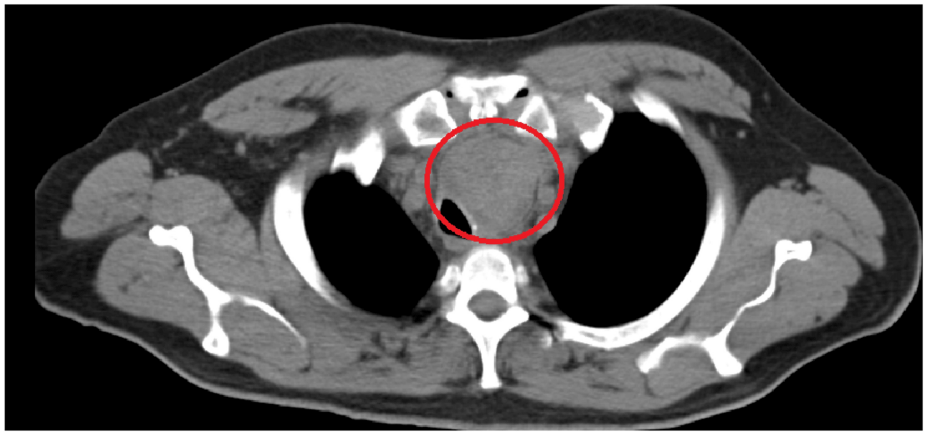


FIGURE 4: CT Neck and Chest With Contrast

The image indicates the CT of the chest and neck, where the red circle highlights a mildly heterogeneous, oval-shaped mass at the left thoracic inlet, measuring about 5 cm × 5 cm × 5 cm, with surrounding fat stranding.

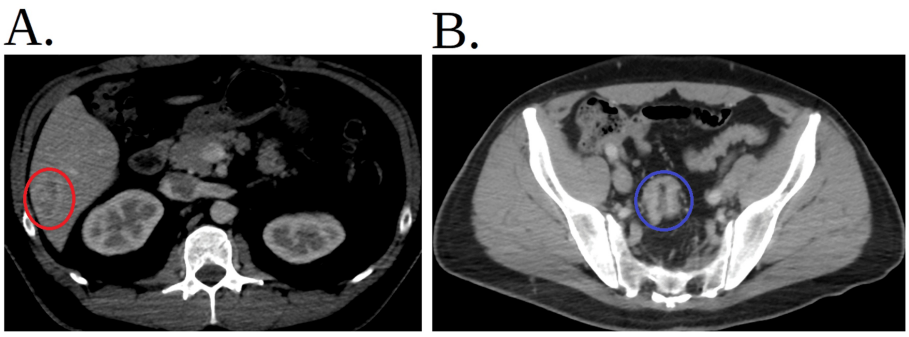


FIGURE 5: CT Abdomen and Pelvis

(A) The image shows a faint, ill-defined mass in red involving the lateral right liver, measuring up to 3.2 cm. This mass is minimally hypoattenuating/hypoenhancing relative to the surrounding liver parenchyma and is delineated by irregular, mild peripheral hyperenhancement.

(B) The image indicates the CT of the abdomen, with the blue circle highlighting abrupt, shelf-like, enhancing wall thickening of the colon, raising concern for colon cancer.

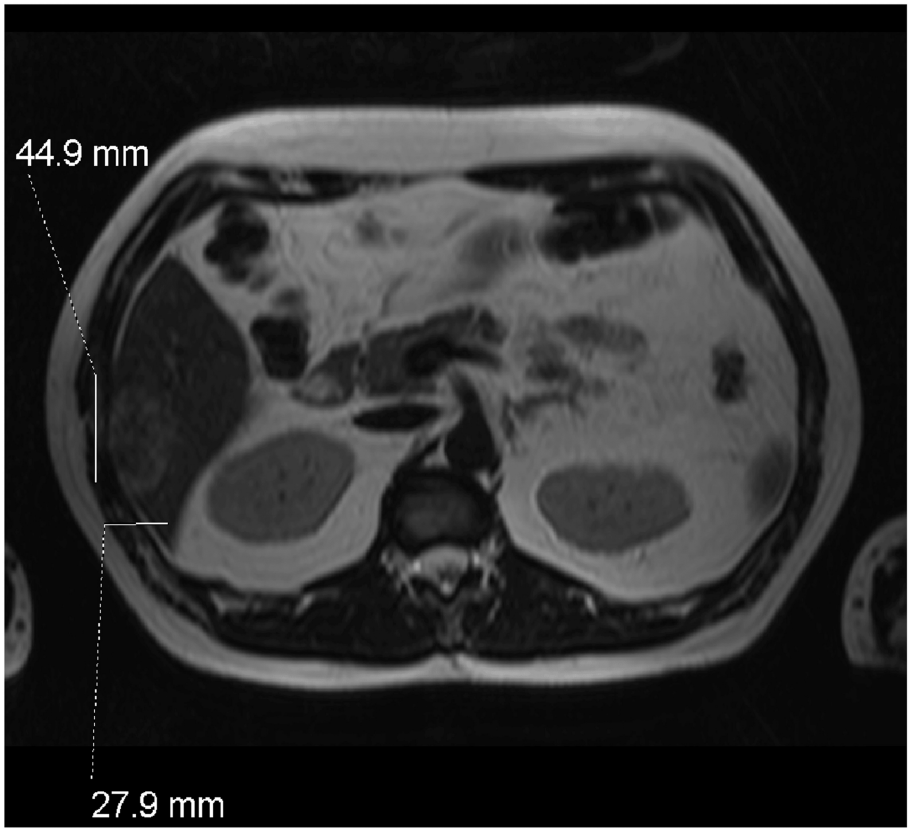


FIGURE 6: MRI With Liver Protocol

The figure shows a heterogeneous T1 hypointense and slightly hyperintense T2 signal mass in the right lobe of the liver measuring approximately 4.5 x 2.8 cm.

A thyroid mass biopsy revealed lymphocytic infiltration consistent with Hashimoto's thyroiditis with microfollicular proliferation, suggestive of a follicular neoplasm (Figures *7A*-*7B*). A liver biopsy confirmed metastatic adenocarcinoma (Figure *7C*), with immunohistochemical staining of cytokeratin 7, cytokeratin 20, SATB2, CA19.9, CDX2, napsin A, PAX8, and TTF1 which the results of the staining supporting colorectal origin (Table *4*).

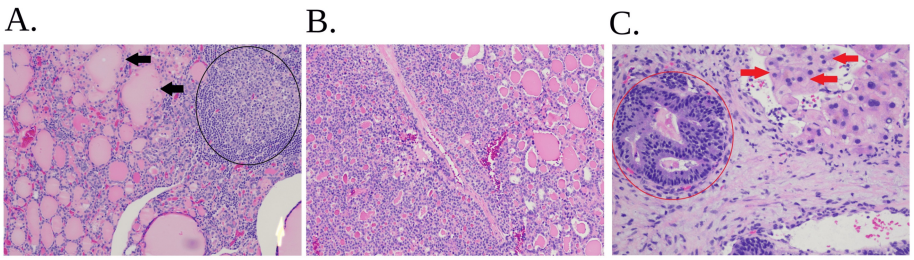


FIGURE 7: Pathology Results of the Thyroid, Mediastinal Mass and Liver Mass

(A) The pathology image shows a biopsy of the thyroid, with the black arrows indicating the follicles and the black circle showing lymphocytic infiltration, indicative of Hashimoto's thyroiditis.

(B) The biopsy of the mediastinal mass shows microfollicles, indicating a neoplasm, particularly an adenoma or carcinoma.

(C) The pathology image shows a biopsy of the liver mass, with the red arrows indicating liver cells and the red circle indicating glandular adenocarcinoma.

Parameter	Value
Cytokeratin 7	Negative
Cytokeratin 20	Positive
SATB2	Positive
CA19.9	Focally positive
CDX2	Positive
Napsin A	Negative
PAX8	Negative
TTF1	Negative

TABLE 4: Immunohistological Staining

Specialist management

Otolaryngology concluded that the thyroid mass represented a follicular neoplasm with a 20%-30% risk of malignancy. Due to progressive dysphagia and positional discomfort, a left hemithyroidectomy with mediastinal mass resection was scheduled and performed.

Oncology subsequently reviewed all pathology and imaging and confirmed follicular thyroid carcinoma. Given the presence of colorectal adenocarcinoma with hepatic metastasis, the patient was further diagnosed with metastatic colorectal cancer. However, the question remains whether the patient had developed HOA secondary to either metastatic colorectal cancer or follicular thyroid carcinoma.

Regardless, a port-a-cath was placed, and neoadjuvant chemotherapy with the FOLFOX regimen (folinic acid, oxaliplatin, fluorouracil) was initiated for three to four cycles prior to surgical resection.

The patient then received neoadjuvant chemotherapy along with surgical resection for his colon adenocarcinoma. At his most recent follow-up, he reported notable improvement in joint pain and fatigue, with a physical examination confirming partial but significant regression of HOA findings.

Discussion

HOA is a syndrome characterized by the triad of particular symptoms, particularly digital clubbing, synovial effusions, and periostosis of the tubular bones [6]. Secondary HOA is most frequently associated with various intrathoracic malignancies, particularly non-small cell lung carcinoma [7]. Although there has been a reported association with various pathologies such as infections, inflammatory bowel disease, chronic pulmonary conditions, and, rarely, extrathoracic malignancies such as gastrointestinal tumors and thyroid cancers [3,4,8,9], the occurrence of HOA as a paraneoplastic manifestation of follicular thyroid carcinoma is exceptionally rare, with only a limited number of cases documented in the literature [5]. With regard to colonic adenocarcinoma, very few, if any, cases of such malignancy have been shown to cause secondary HOA. The most recent research has only depicted generalized juvenile and adenomatous polyposis being associated with HOA [3,4].

This raises the question of the pathophysiology of HOA. Unfortunately, it remains to still be incompletely understood, albeit some proposed mechanisms include increased levels of circulating vascular endothelial growth factor (VEGF) and platelet-derived growth factor (PDGF), which may promote periosteal new bone formation and connective tissue proliferation [9,10]. It has also been theorized that prostaglandins, particularly E2, stimulate VEGF, which activates osteoblasts, resulting in HOA [9].

In thyroid malignancies, few cases have been documented, all with differing types of thyroid cancers, including but not limited to follicular thyroid cancer, anaplastic thyroid carcinoma, undifferentiated fusiform cell thyroid cancer, etc. [5,9,11]. Such thyroid malignancies have been hypothesized to cause secondary HOA via PDGF, which is released in response to platelet aggregates or megakaryocyte fragments that are noted in the pulmonary capillary system, resulting in mesenchymal cell growth and new bone development, resulting in digital clubbing rather than the VEGF pathway [9,12]. In fact, such prior thyroid carcinoma case reports with secondary HOA have noted unremarkable VEGF [5,9,11].

However, this brings into question another interesting factor, since the patient had also developed colon adenocarcinoma, which had metastasized to the liver. To date, there hasn't been much literature regarding

colon adenocarcinoma causing secondary HOA. Yet some gastrointestinal generalized juvenile and adenomatous polyposis have been associated with secondary HOA [3,4]. Unfortunately, in both cases, the patients had pulmonary arteriovenous malformations, which could also have played a role in the development of secondary HOA rather than the polyposis itself.

In summary, this case portrays the patient presenting with symptoms of periostosis of the tubular bones and digital clubbing, which could have been due to either his follicular thyroid carcinoma or his colon adenocarcinoma as the primary cause, or even possibly both. This temporal relationship supports a paraneoplastic mechanism, as resection of the primary tumor and treatment of his colon cancer improved his secondary HOA by decreasing the source of circulating mediators responsible for the disease. As such, the key feature of this case revolves around surgical removal of the mediastinal mass as well as treatment of the patient's colonic adenocarcinoma, with subsequent improvement in digital clubbing. While it is most likely that the patient's follicular thyroid carcinoma is the cause, since it has been reported in the literature previously [5], the possibility of colonic adenocarcinoma playing a role shouldn't be excluded. This is particularly true since the inherent mechanism by which malignancies cause HOA is still not clearly understood.

Conclusions

Overall, despite the limitations of establishing a causal relationship, this case highlights an association between certain unknown malignancies' abilities to cause secondary HOA. Thus, clinicians should consider a broad differential diagnosis when encountering severe digital clubbing. Early recognition of clubbing as a possible paraneoplastic sign can facilitate prompt investigation and diagnosis of an occult malignancy. Such a resolution also underscores the reversibility of certain paraneoplastic manifestations when the underlying malignancy is treated in a timely manner. Furthermore, this case adds to the limited body of literature describing follicular thyroid carcinoma and potentially colon adenocarcinoma as potential causes of HOA, reinforcing the concept that paraneoplastic syndromes may manifest in association with less common malignancies.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Kevin T. Dao, Konni Granma, Lambert Diep, Ayham Aboeed

Acquisition, analysis, or interpretation of data: Kevin T. Dao, Konni Granma, Reshma Mikky Shaji, Mia Yasonova, Wilbur Montana, Ayham Aboeed

Drafting of the manuscript: Kevin T. Dao, Konni Granma, Lambert Diep, Reshma Mikky Shaji, Wilbur Montana, Ayham Aboeed

Critical review of the manuscript for important intellectual content: Kevin T. Dao, Konni Granma, Reshma Mikky Shaji, Mia Yasonova, Ayham Aboeed

Supervision: Wilbur Montana, Ayham Aboeed

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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