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Causes of False-Positive Radioactive Iodine Uptake in Patients with Differentiated Thyroid Cancer

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Abstract
Purpose Radioactive iodine (RAI) whole-body scan is a sensitive imaging modality routinely used in patients with differentiated thyroid cancer to detect persistent and recurrent disease. However, there can be false-positive RAI uptake that can lead to misdiagnosis and misclassification of a patient's cancer stage. Recognizing the causes of false positivity can avoid unnecessary testing and treatment as well as emotional stress. In this review, we discuss causes and summarize various mechanisms for false-positive uptake.
Recent Findings We report a patient with differentiated thyroid cancer who was found to have Mycobacterium avium complex infection as the cause of false-positive RAI uptake in the lungs. Using this case example, we discuss and summarize findings from the literature on etiologies of false-positive RAI uptake. We also supplement additional original images illustrating other examples of false RAI uptake.
Summary False-positive RAI uptake may arise from different causes and RAI scans need to be interpreted in the context of the patient's history and corresponding cross-sectional imaging findings on workup. Understanding the potential pitfalls of the RAI scan and the mechanisms underlying false uptake are vital in the care of patients with differentiated thyroid cancer.

Keywords Radioiodine · I-131 · Whole-body scintigraphy · False-positive uptake · Differentiated thyroid cancer

Introduction

Differentiated thyroid cancer (DTC), which includes papillary and follicular histologic types, constitutes greater than 90% of all thyroid cancer cases [1]. Although there is an uptrend in the diagnosis of DTC over the past several decades, with an average annual increase in the incidence of 3.6% in the USA [2], the mortality rate has remained quite low. The good prognosis can be attributed to the less aggressive behavior of most DTC and the effective use of radioactive iodine (RAI) after total thyroidectomy in patients who are at risk for persistent or recurrent disease [3].

The use of RAI has been an integral component of care for patients with DTC after thyroidectomy since the 1940s [4]. RAI whole-body scan (WBS) informs clinicians of disease staging and documents RAI avidity of any structural disease, and it is a critical tool in the treatment and surveillance of DTC [5, 6]. The principle of RAI use in diagnosing and treating DTC is that some thyroid cancer cells retain expression of the sodium-iodide symporter (NIS), which is a plasma membrane glycoprotein that facilitates active iodide transport [7••]. However, NIS expression is not specific to thyroid cells. The false-positive findings on RAI-WBS can be due to ectopic thyroid tissue, physiologic NIS expression in other tissues, RAI retention in body cavities or ducts, direct RAI bonding to

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metallic foreign bodies, contamination, inflammation and infection, and non-thyroid neoplasms. Therefore, the finding of unexpected iodine uptake should be interpreted in the context of the patient's medical history, thyroglobulin levels, and histopathologic features of the tumor, corroborated with cross-sectional imaging studies. Tissue sampling may be needed in some situations.

This review will focus on the various causes of false-positive WBS results and discuss a case example illustrating the potential pitfalls of making clinical judgments solely based on the WBS.

Case

A 51-year-old woman with a history of stage 1B breast cancer treated with mastectomy and adjuvant radiotherapy, was found on breast cancer staging computed tomography (CT) scan to have several hypodense thyroid nodules with dystrophic calcifications. Subsequent workup followed by total thyroidectomy with paratracheal lymph node dissection showed a 1.8-cm follicular variant papillary thyroid carcinoma (PTC) with lymphovascular invasion and positive margins without extrathyroidal extension. There were 4/4 positive lymph nodes with perinodal soft tissue extension, amounting to a pathological stage of pT1bN1aM0 and clinical stage I [8]. Three months later, the patient was treated with 73.6 mCi (2723 MBq) of I-131, and the post-treatment WBS showed accumulation in the thyroid remnant and thoracic outlet lymph nodes without lung or bone uptake (Fig. 1). Stimulated thyroglobulin was 92.8 ng/mL [reference: no evidence of disease defined as < 1 ng/mL] with thyrotropin (TSH) level of 205 mIU/L [reference: 0.4–4.5 mIU/L]. In the next year, the patient had two additional paratracheal lymph node dissections.

Despite repeated surgeries, the patient continued to have a biochemically incomplete response. Her thyroglobulin levels remained stably elevated around 4–5 ng/mL on TSH suppression and around 20 ng/mL on recombinant human thyrotropin (rhTSH) stimulation. Six months after the most recent lymph node dissection and about two years after the initial diagnosis, she underwent a second RAI treatment with 100 mCi (3700 MBq) of I-131. Her stimulated thyroglobulin level was 21.6 ng/mL. This time, the post-treatment RAI-WBS exhibited intense uptake within the lung fields and the thyroid bed (Fig. 2). The new finding of pulmonary uptake prompted a [18F] fluorodeoxyglucose positron emission tomography/computed tomography (FDG PET/CT) scan, which showed findings consistent with right cervical nodal metastases and suggestive of lung metastases (Fig. 3a–d). Four months later, the suppressed thyroglobulin level remained unchanged at 4 ng/mL.

Since it was presumed that the patient had local and distant metastatic disease, she was referred to our institution for further management. The distribution of the lung nodules on FDG PET/CT was found to be atypical for metastatic disease; Chest CT was obtained to further characterize the lung nodules, and it revealed probable chronic airway infection given mucus impaction, bronchiectasis, and clustered subcentimeter nodular opacities throughout the lingula, right middle, and left lower lobes, suggestive of atypical mycobacterial infection (Fig. 3e and f). The area of these abnormalities corresponded to the uptake on the WBS. The patient underwent bronchoscopy with bronchoalveolar lavage and was diagnosed with Mycobacterium avian complex (MAC) infection. Thus, the RAI uptake in the lungs was favored to be a spurious finding due to underlying pulmonary infection rather than metastatic thyroid cancer. The source of her elevated thyroglobulin was attributed to cervical and mediastinal nodal disease that was found on subsequent neck ultrasound and magnetic resonance imaging.

Discussion

Despite the high diagnostic sensitivity and specificity of RAI-WBS for persistent or recurrent DTC, false-positive results can occur and be due to a variety of causes (Table 1). The major diagnostic dilemma is when false-positive uptake occurs in areas where DTC frequently metastasizes to, such as the bones and lungs, as shown in our case. We review additional causes of RAI-WBS false positivity, which are summarized below.

Physiologic Causes

Extraglandular Thyroid Tissue

Embryologically, thyroid tissues originate at the base of the tongue and migrate caudally along the midline via the thyroglossal duct. Failure of embryologic decent leads to ectopic thyroid tissues; the most common locations are at the base of the tongue and along the thyroglossal duct [9]. Other rarer sites can be mediastinal, intrathoracic, and in distant subdiaphragmatic locations [10]. Ectopic thyroid tissues are functional, and therefore, can take up RAI similarly to a normal thyroid gland [11]. The prevalence of ectopic thyroid is low, about 1 in 100,000 to 300,000 people, which is likely an underestimation since the clinical prevalence of those with thyroid disease is about 1 in 4000 to 8000 people [10].

Struma ovarii is a rare variant of an ovarian teratoma composed of more than 50% of mature thyroid tissues. It comprises roughly 1% of all ovarian tumors and 2.7% of all

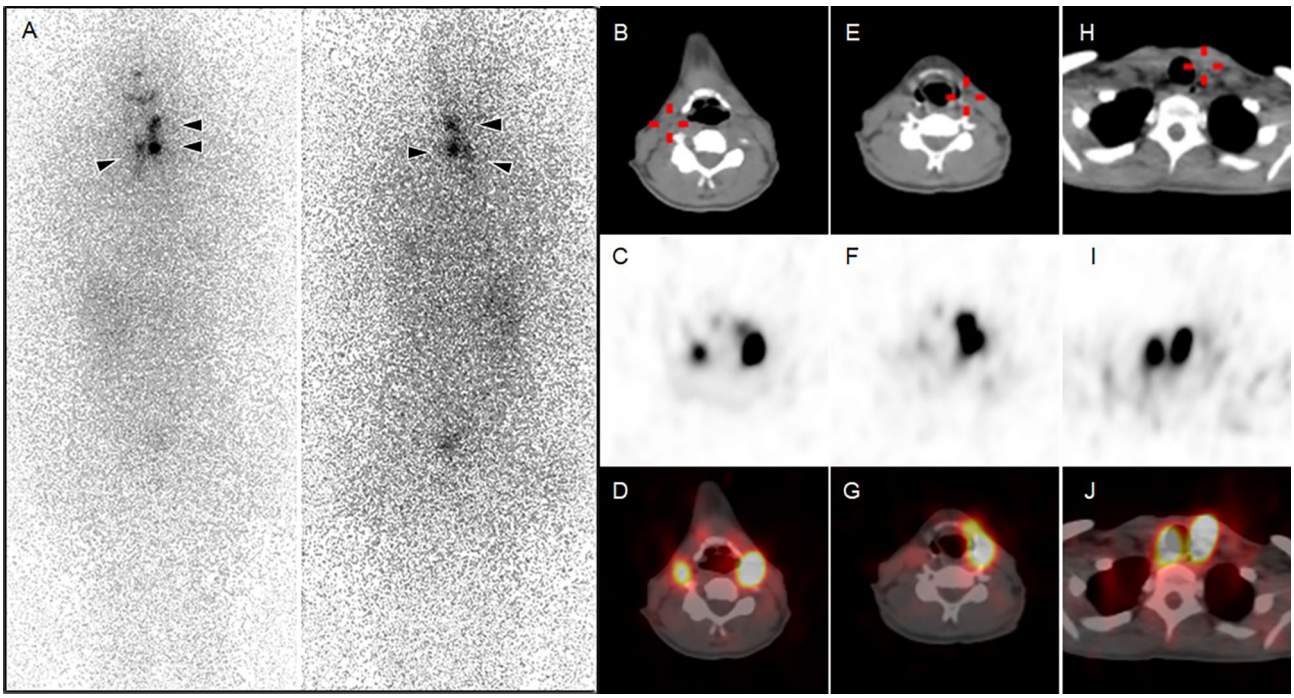

Fig. 1 Imaging after initial I-131 treatment. **a** RAI-WBS with accumulation in the thyroid remnant and neck lymph nodes. **b**, **e** CT, **c**, **f** single-photon emission computed tomography (SPECT), and **d**, **g** transaxial fused SPECT/CT identified bilateral upper and middle cervical lymph nodes with uptake of the radiopharmaceutical. **h** CT, **i** SPECT and **j** transaxial fused SPECT/CT identified left thyroid remnant and right upper paratracheal nodal uptake of the radiopharmaceutical

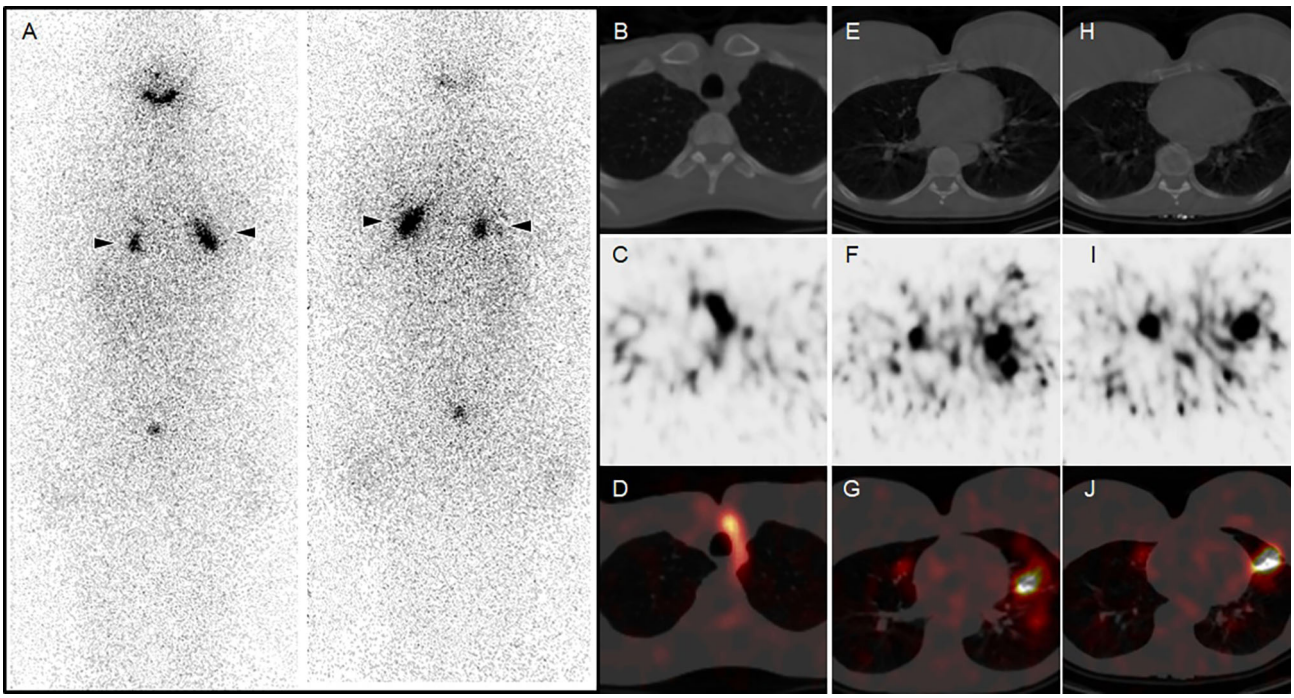

Fig. 2 Imaging after repeat I-131 treatment. **a** RAI-WBS with accumulation in the lungs and the thyroid bed. **b** CT, **c** SPECT, and **d** transaxial fused SPECT/CT identified thyroid bed radiopharmaceutical uptake. **e**, **h** CT, **f**, **i** SPECT and **g**, **j** transaxial fused SPECT/CT identified bilateral pulmonary radiopharmaceutical uptake

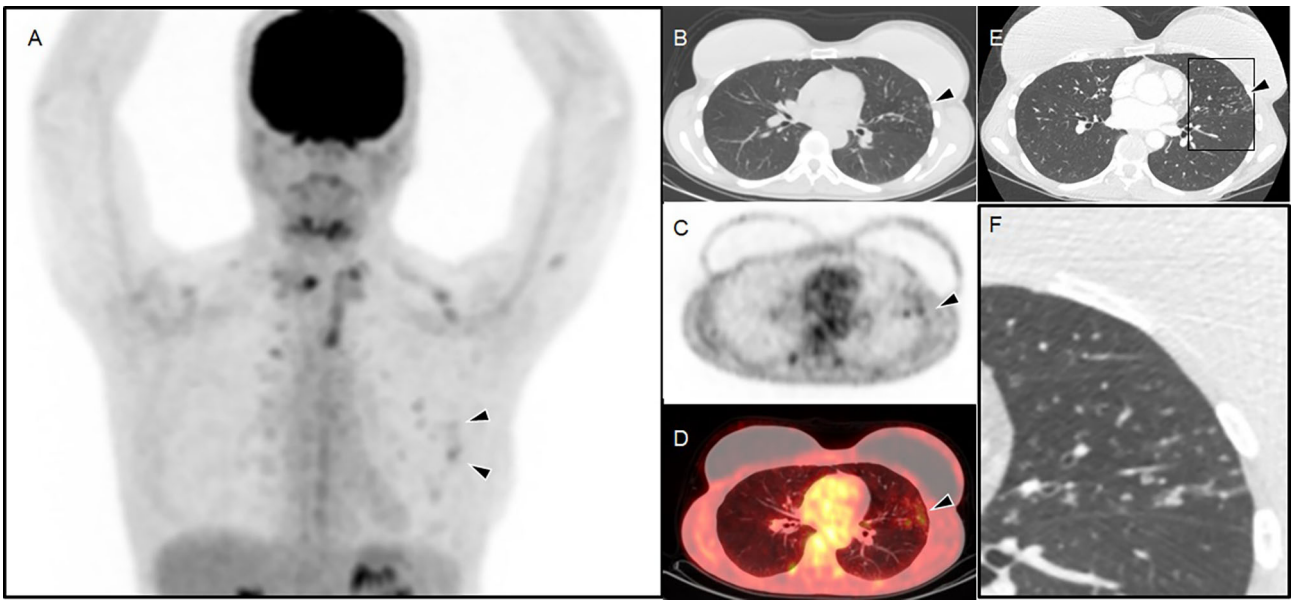

Fig. 3 Imaging to characterize extent of structural disease. PET/CT images including **a** maximum intensity projection (MIP), **b** CT, **c** PET, **d** transaxial fused PET/CT. CT scan performed following patient referral to our institution and two months after the second I-131 treatment showed pulmonary nodules and linear thickening **e** at 1 × and **f** at 2.71 × inset magnification

ovarian teratomas [12]. False-positive RAI uptake has been described in struma ovarii [13–15] and in ovarian teratomas with or without thyroid parenchyma [16, 17].

Uptake in Non-thyroidal Tissue

Expression of NIS in other tissues constitutes an important mechanism of RAI uptake outside of the thyroid. Clinically relevant sites include the salivary glands, lacrimal glands, gastrointestinal tract, choroid plexus, and mammary glands [7••]. The physiologic uptake of RAI in salivary glands is a reason for the commonly observed salivary gland-related side effects such as dry mouth, sialadenitis, and altered taste in up to 40% of patients receiving RAI doses of 75 mCi (2775 MBq) or greater [18]. A more recent systemic review found that salivary gland dysfunction can be found in 16–54% of patients receiving RAI [19]. Mammary glands have been shown to be a major organ expressing NIS with a marked upregulation during lactation by oxytocin and prolactin [20, 21]. Uptake has been described not only in lactating breasts [22] but also in non-lactating breasts [23]. In addition, there was a reported case of bilateral breast RAI concentration in the setting of a pituitary macroprolactinoma [24], which is caused by hyperprolactinemia leading to upregulation of NIS expression. It is essential to correlate the potential uptake in breast tissues clinically and radiographically with single-photon emission computed tomography (SPECT-CT) as the uptake can be misinterpreted as pulmonary metastases given its location.

A frequent finding on RAI-WBS is diffuse uptake of RAI in the liver, reported in 40–94% of patients [25–28]. The exact mechanism is unknown. One proposed mechanism is hepatic metabolism of organified RAI released from the thyroid tissue. This mechanism and the clinical significance are debated, with some reporting an association with residual thyroid tissue [25, 26], while others suggest no correlation with serum thyroglobulin levels or presence of thyroid tissue or disease [27, 29]. Intrahepatic bile duct NIS expression is another postulated mechanism since some report correlation between hepatic uptake and dose of RAI [25–27]. Hepatic steatosis and elevated hepatic enzymes have also been shown to be associated with RAI uptake, both may cause delayed de-iodination and iodine excretion leading to higher liver retention [27, 30••].

RAI uptake by the thymus has been described most often in young patients (< 50 years of age) [31–33]. The mechanism has been attributed to the expression of NIS and thyroid-related proteins in human thymic tissues [34]. However, a study found no difference in NIS and TSH receptor expression with age [35]. An alternate explanation is that older patients have lower thymic tissue volume since the thymus involutes with age, so thymic RAI uptake is less frequently seen in this population.

Radioiodinated Bodily Fluid Retention

An oral dose of RAI is quickly and almost entirely absorbed via the gastrointestinal tract. In fact, more than 90% of the dose is then excreted through the kidney over

Table 1 Summary of the causes of false-positive RAI uptake

Physiologic		Pathologic	
Mechanism	Causes	Mechanism	Causes
Sodium-iodide symporter (NIS) expression	Thyroid tissue	Sodium-iodide symporter (NIS) expression	Benign tumors with NIS expression (non-thyroid)
	Ectopic thyroid along embryonic migration tract (base of tongue and along thyroglossal duct)		Salivary gland tumors, breast fibroadenomas, ovarian tumors (cystadenomas)
	Struma ovarii		Malignant tumors with NIS expression (non-thyroid)
	Non-thyroid tissue		Breast, prostate, ovary, lung, colon, endometrium
	Salivary glands, lacrimal glands, gastrointestinal tract, choroid plexus, mammary glands, thymus		
Retention of radioiodinated bodily fluids	Normal anatomy	Hyperemia/vascularity	Inflammation
	Bladder (urine), colon (feces), esophagus (saliva)		Bronchiectasis, asthma exacerbation, rheumatoid arthritis, folliculitis, sialadenitis, sinusitis, cholecystitis
	Anatomical anomaly		Infections
	Cystic structures (nasolacrimal sac, pleuropericardial, bronchogenic, thymic, breast, hepatic, renal, retroperitoneal, ovarian, Nabothian, sebaceous, epithelial)		Respiratory (fungal infection such as aspergilloma, bacterial infection such as tuberculosis and Mycobacterium avium complex)
	Diverticula (esophageal, epiphrenic, Meckel's, appendix, pelvicalyceal)		Liver abscess
	Vascular aneurysms/ectasia		Trauma
	Ductal/tubal dilation and obstruction (nasolacrimal, parotid gland, salivary gland, achalasia, hiatal hernia, gastric volvulus, biliary tract)		*Benign tumors with high vascularity* (non-thyroid)
	Ectopic kidney		Meningiomas, angiomas/hemangiomas
			Malignant tumors, primary or metastasis (non-thyroid)
Direct RAI bonding to metallic foreign bodies	Copper intrauterine devices		
	Surgical clips, metallic sutures		
Contamination by radioiodinated bodily fluids	Skin, hair, clothes, prosthesis, tracheostomy or gastrostomy site		

5 days, with the majority disposed in the first 24 h [36]. The remaining RAI is excreted via sweat, gut, and other bodily secretions. Any retention of body fluids with or without anatomical abnormalities can be seen on WBS. Commonly, radioiodinated urinary retention in the bladder, salivary retention in the esophagus, and large intestinal retention of feces are noted, especially if the WBS is obtained soon after RAI administration.

Many cases of anatomical variation have been reported to lead to false-positive RAI retention. Examples include cystic and pouch-like structures such as nasolacrimal sac cyst [37], esophageal diverticulum [38–40], epiphrenic diverticulum [41], pleuropericardial cyst [42], bronchogenic cyst [43, 44], thymic cyst (Fig. 4) [45, 46], breast cyst [47], Meckel's diverticulum [48], appendix [49], pelvocalyceal diverticulum [50, 51], hepatic cyst [52–54], renal cyst [55–58], retroperitoneal cyst [59•], ovarian cyst [60, 61], nabothian cyst [62], sebaceous cyst [63, 64], and epithelial cyst [65]. Arterial dilation leading to stasis of blood flow has also been shown to have false-positive RAI retention [66, 67]. The mechanism is the passive diffusion of iodine into cysts and diverticula that becomes trapped due to the slow drainage. Ovarian cysts are commonly found in women of reproductive age, with the vast majority being physiologically functional [68]. Functional ovarian cysts have been described to retain RAI uptake [61]. Thus, a potential clinical implication is to time the RAI administration with the menstrual cycle in premenopausal women to minimize this potential uptake in these cysts.

Ductal and tubular pathology or dilation can cause retention of RAI as well. Cases in the head and neck include nasolacrimal duct obstruction [69], parotid gland

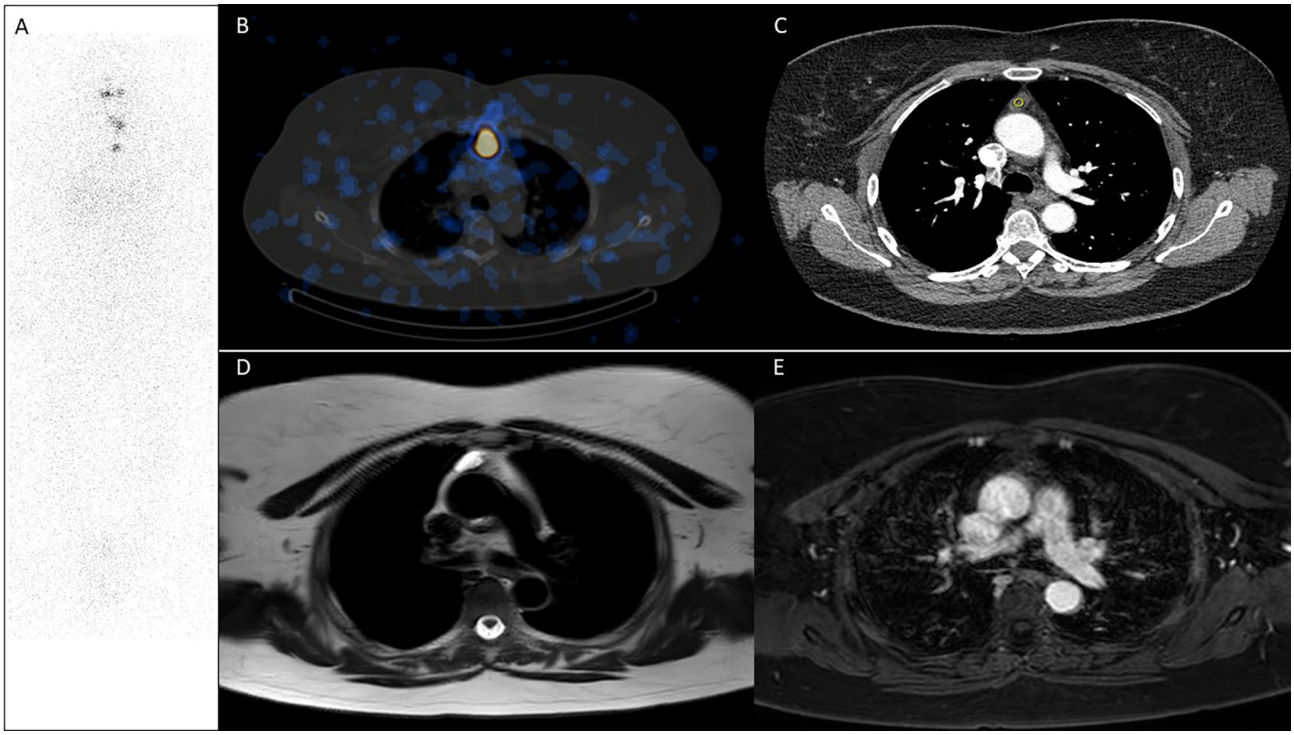

Fig. 4 I-131 retention in thymic cyst. **a** RAI-WBS, **b** SPECT/CT, **c** CT with contract, **d** MRI T2-weighted, **e** MRI post-gadolinium identified I-131 retention in thymic cyst

duct ectasia [70], and xerostomia [71]. In the gastrointestinal tract, cases of false positivity have been demonstrated in achalasia [72], esophageal scarring [73], hiatal hernia [74, 75], gastric volvulus [76], and biliary tract obstruction [77]. Other reported anomalies with RAI uptake are seen in ectopic kidney [78] and hematocolpos [79]. The patient's medical history and cross-sectional images can aid in identifying these structural anomalies.

Direct RAI Bonding to Metallic Foreign Bodies

Various metallic foreign bodies have been shown to have apparent increased RAI uptake on RAI-WBS. One of the most commonly seen materials with this behavior is dental amalgam (Fig. 5a) [80, 81]. The proposed mechanism of tracer retention demonstrated by in-vitro binding assays is the interaction and chemical binding of negatively charged iodide ions to the positively charged metal ions [80, 81]. Through a similar mechanism, copper intrauterine devices (Fig. 5b), metallic surgical clips, and metallic sutures also retain RAI tracer [82–85•]. Moreover, these foreign bodies induce a local inflammatory response that subsequently may lead to RAI uptake in surrounding tissues (see Section on Inflammation and infection).

Contamination by Radioiodinated Bodily Fluid

Contamination artifacts are caused by radioiodinated bodily fluids on the skin, hair, and clothes. Perspiration can result in skin and hair contamination in the axilla [86] and scalp [87]. Contamination has also been described in artificial eyeballs by tear contamination [88•, 89•] and within tracheostomy sites [90]. Uptake from contamination tends to be superficial and/or in odd locations, and this can be easily corrected by washing the suspected area or by removing contaminated clothing.

Pathologic Causes

Inflammation and Infection

Inflammation and infection can cause spurious RAI uptake. The proposed mechanisms are inflammation-mediated hyperemia, increased vessel permeability, and possibly, increased retention of organified I-131 by leukocytes during bactericidal activity [91]. Diseases of the lungs are particularly important clinical entities because of their mimicry of pulmonary metastases. Conditions of the lungs such as bronchiectasis [92–94], acute respiratory infection [95], asthma exacerbation [96•], and fungal infections like aspergilloma [97] and tuberculosis [98, 99] have been known to cause spurious RAI uptake. Case reports of

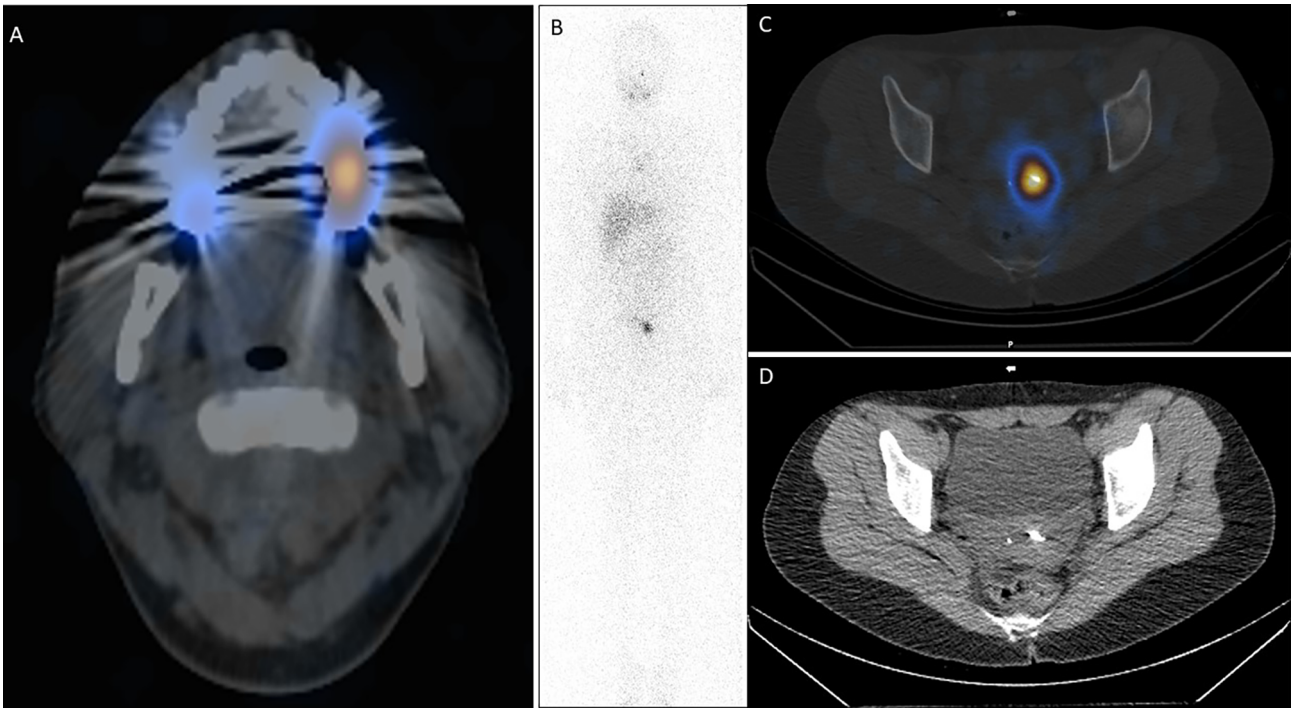

Fig. 5 I-131 retention by direct chemical binding to metallic foreign bodies. **a** SPECT/CT with RAI retention in dental amalgam. **b** RAI-WBC, **c** SPECT/CT, and **d** CT showing uptake by copper intrauterine device

inflammatory and infectious conditions causing RAI uptake in other body sites include rheumatoid arthritis [100], folliculitis [101•, 102], sialadenitis [103], sinusitis [104], cholecystitis [64], and liver abscess [105].

Our case describes a case of Mycobacterium avium complex infection causing chronic inflammation and bronchiectasis in the lungs. The distribution of pulmonary nodules on cross-sectional imaging with its corresponding RAI uptake on RAI-WBS was unusual for metastatic thyroid cancer, which typically spreads with hematogenous dissemination producing a miliary or randomly distributed pattern of nodules throughout all lobes, often with a basilar predominance [106]. The discordant findings prompted further evaluation and eventual diagnosis of the underlying cause. Our case highlights the importance of a multi-disciplinary team approach to explain questionable findings.

Trauma

Similarly, trauma also leads to increased perfusion and vasodilation and can be a cause of false positivity. In fact, there has been a reported case of chest wall uptake after needle biopsy [107] and uptake in post-traumatic superficial scabs [108].

Benign and Malignant Tumors

Benign tumors may have unexpected uptake of RAI. The mechanism can be due to functional physiologic NIS expression in the parenchymal tissues, such as in salivary gland tumors [109–112], breast fibroadenomas [113], and ovarian tumors [114–117]. Tumors with high vascularity, such as meningiomas [118, 119] and angiomas/hemangiomas [120–124] can cause RAI pooling and uptake on RAI-WBS.

Pathologic RAI accumulation in non-thyroid malignancies and metastases occurs in various body sites. The underlying mechanisms are similar to benign tumors, including NIS expression, high vascularity, and in addition, local tumoral inflammatory response in these malignancies. NIS expression is shown in carcinomas of the prostate, ovary, lung, colon, endometrium, and breast [125, 126]. Since NIS expression has been found by immunohistochemistry in about 80% of human breast cancers, there is a growing interest in employing RAI in the treatment of breast cancer [127].

Conclusion

RAI-WBS is an essential diagnostic tool for detecting normal thyroid tissue and metastatic differentiated thyroid cancer based on iodine avidity of the follicular thyroid cells

mediated by the sodium-iodide symporter. It can detect persistent or recurrent disease after thyroidectomy. However, a variety of other disease and physiologic processes can cause false-positive results, and without careful evaluation, misinterpretation can lead to a cascade of diagnostic procedures and unnecessary treatment, which are not only expensive, but can be emotionally taxing on the patient.

Our case is a salient demonstration of how false-positive RAI uptake can be encountered in clinical practice. It highlights the potential pitfalls of the RAI-WBS and the importance of interpreting the results in the context of clinical presentation and corroborating cross-section imaging studies, especially when the uptake patterns are unusual. It is important for clinicians taking care of patients with DTC to recognize the potential causes and mechanisms for false-positive RAI uptake.

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Declarations

Conflict of interest The authors have nothing to disclose.

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Recently published papers of particular interest have been highlighted as:
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- •• Of major importance

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