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Clinicopathological Characteristics of Collision tumors of Thyroid in the clinical setting of Medullary Thyroid Carcinoma

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ABSTRACT:

OBJECTIVES: To study the clinicopathological features of collision tumours of the thyroid and to develop a logical management regimen in view of the rarity of these tumours.

METHODS: A retrospective study on collision tumours of the thyroid, diagnosed over the previous 15 years in a tertiary cancer care centre. The inclusion criteria were proven cases of collision tumours of medullary thyroid carcinoma (MTC) with papillary thyroid carcinoma (PTC) and/or follicular thyroid carcinoma (FTC).

RESULTS: Among the 470 patients with MTC, 24 were found to harbour collision tumours (5.1%). Amongst 18 patients (75%) with lymph node metastases, 88.8% originated from MTC and 22.2% from the PTC component. Two patients (8.3%) presented with distant metastases. Eight patients underwent radioactive iodine (RAI) scan, of whom 7 demonstrated neck uptake and received RAI therapy. Fifteen patients (62.5%) were disease-free, 8 patients (33.33%) harboured biochemical and/or structural residual disease; and one patient died due to unrelated etiology.

CONCLUSION: Thyroid collision tumour is a relatively rare entity, and is usually undiagnosed preoperatively. The prognosis of the disease primarily depends on the tumour aggressiveness of MTC component. A combined follow-up with tumour markers and imaging, including PET-CT molecular imaging approaches should be adopted.

Keywords: Collision tumour of the thyroid; Medullary thyroid carcinoma; papillary thyroid carcinoma; radioiodine scan; Calcitonin; Thyroglobulin.

INTRODUCTION

Collision tumours are rare entities defined as neoplastic lesions with two or more histologically and morphologically different tumours that maintain distinct borders within the same organ. It can be of two benign tumours, two malignant tumours, or a benign and malignant tumour. (1) Collision tumours are more frequently found in female patients, with the majority of cases occurring in the fifth to seventh decades of life. Often, only one component of these collision tumours is recognised during pre-surgical evaluation, and the other component is an incidental histopathological finding. (2)

Collision tumours are found in various organs, such as the kidneys, colon, stomach, ovaries, colon, and lungs; they are extremely rare in the thyroid and represent only <1% of thyroid cancers. (2-7) Among thyroid collision tumours, co-occurrence of medullary and papillary carcinoma is the most frequent. (8) There are several theories to explain the pathogenesis of collision tumours, including stem cell theory, random effect theory, tumour predisposition, and hypotheses about common genetic behaviour, such as germline mutation of RET (Rearranged during Transfection) proto-oncogene in medullary thyroid carcinoma and papillary thyroid carcinoma co-existence. (8)

The medullary component of thyroid collision tumours is more aggressive than the papillary component, and the MTCs in collision tumors entity is more aggressive than medullary thyroid carcinoma-only tumours. (2) The management of collision tumours is more challenging than that of individual tumours because of different biological aggressiveness, prognosis, and treatment options; therefore, it needs to evolve through critical evaluation of the disease course (8), and there is uncertainty regarding the clinicopathological features of these tumours.

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Management guidelines for these tumours are poorly defined. Therefore, in the present study, we aimed to understand the characteristics of these tumours. We retrospectively analyzed thyroid collision tumours with components of MTC and PTC, confirmed by histopathology, and analyzed the demographic features, clinical characteristics, management, and clinical outcomes of these patients.

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MATERIALS AND METHODS

This was a retrospective study of collision tumours of the thyroid in 24 patients conducted in a tertiary care centre between 2009 and 2022. The inclusion criteria were proven cases of collision tumours of MTC with PTC and/or FTC by histopathological diagnosis and those who had already undergone total thyroidectomy. The exclusion criteria were patients with insufficient histological material, those who received preoperative radiotherapy, and those aged less than 18 years. The clinical details of the patients were obtained from their electronic medical records (EMR).

All patients were evaluated by multidisciplinary team, and therapy decision making was performed based on available guidelines updated at that time and expert individualised remarks from the team. Preoperative and postoperative USG (Ultrasonography) was performed by a senior radiologist using high-frequency linear probes. Serum Tg (Thyroglobulin) and calcitonin assays were performed in standard institutional laboratories using RIA/CLIA (Radioimmunoassay/Chemiluminescence immunoassay). The normal range for S.Tg post-operative patients with TSH stimulation was considered to be less than 1 ng/ml, while post-treatment on thyroid suppression therapy was given as per their ATA (American Thyroid Association) risk category and checked at follow-up visits. Pre-surgery FNAC and post-surgery histopathology samples were handled by a team of senior pathologists specialized in head-neck oncology. Post-surgery RAI scans were performed after administration of 1mCi/37MBq of RAI 24 h prior on planar double-headed gamma camera, and SPECT/CT was performed if needed. For post-RAI therapy, follow-up scans were acquired in a similar manner using 3-5 mCi/111-185MBq RAI activity 2-3 days prior to administration. For SSTR PET 1-2mCi/37-74 MBq of ^{68}Ga-DOTATATE/NOC was administered 1 hour prior to acquisition, and all patients were scanned

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vertex to mid-thigh on PET/CT scanner. RAI therapy (from 30 to 250 mCi) was administered in the high-dose therapy ward after the decision of the team to abide by all national radiation regulatory parameters. Additionally, the team consisted of a head neck oncology specialist surgical oncologist, medical oncologist, and radiation oncologist for each therapy plan and follow-up.

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RESULTS:

As per our retrospective study conducted in patients with medullary thyroid carcinoma over a period of 15 years, 24 of the 470 patients were found to have collision tumours, which is implies a rate of occurrence of 5.1% in all MTC cases.

Demography:

Among 24 patients diagnosed with thyroid collision tumours, the mean age of the patient was 50.7 years (range 26-90 years) with a male-to-female ratio of 1:1.4 (10 males and 14 females).

Clinical characteristics:

All the patients presented with swelling in the front or on either side of the neck.

Investigations:

Preoperative ultrasound data were available for 10 patients. Of these, 5 collision tumours were detected by ultrasonography. Three PTC (two of which were micro-PTCs) and two were missed by ultrasonography.

Preoperative serum calcitonin was available for nine patients, and the mean value was 6476 pg/ml (range, 1080 pg/ml to 20842 pg/ml).

According to histopathology, among 28 lesions of MTC, 16 were in the right lobe (57.14%), 11 in the left lobe (39.28%), and one in the isthmus of the thyroid. (3.57%).

Among 24 patients with collision tumours of the thyroid, in most cases (20/24;83.3%), the average size of the MTC component was higher than that of the PTC component. Among the 25

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PTC, 14 lesions were in the right lobe (56%), 10 were in the left lobe (40%), and 1 lesion was in the isthmus (4%).

Eighteen patients (75%) presented with lymph node metastases. Among them, 16 patients had the MTC component (88.8%); and 4 the PTC component (22.2%). Two patients (11.1 %) had metastases from MTC and PTC.

Thirteen patients had extrathyroidal extension (ETE) (54.16%). Among the 24 patients, only two presented with distant metastasis to the lung and skeletal system (8.3%).

Treatment and follow-up

All patients underwent total thyroidectomy, with or without lymph node dissection. Eight patients underwent RAI scan. Thyroid hormone was withdrawn and a low-iodine diet for 3-4 weeks to attain a TSH value of >30mIU/ml before the RAI scan according to the institutional protocol. One mCi (37 MBq) of radioactive iodine (I-131) was administered orally, and imaging was acquired 24 hours after administration. A wide field-of-view gamma camera with a high-energy parallel-hole collimator was used for imaging, employing a 20% window centred at 364 keV. Images were acquired for 15 min, and the percentage of radioactive iodine uptake was calculated using a thyroid uptake probe. Among them, seven patients showed neck uptake. Four of these seven patients had lymph node metastasis (three from MTC and one from PTC). Patients without lymph nodal metastasis from PTC received around 50-100 mCi (1.85 GBq-3.7 GBq) of radioactive iodine and patient with lymph node metastasis from PTC received around 150 mCi (5550 MBq) of radioactive iodine as per institutional protocol. A negative iodine scan was found in one patient with papillary microcarcinoma (mPTC) component. An RAI scan performed after

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6 months revealed no obvious RAI uptake in all patients who received therapy, suggestive of complete ablation to iodine therapy. Three patients with advanced-stage MTC underwent EBRT of the neck. Irrespective of the treatment received, all patients received a suppression dose of thyroxine and were followed up with tumour markers (Serum Thyroglobulin and Serum Calcitonin).

Follow-up was for a mean period of 7.7 years (range: 5 months to 15 years). Of the 24 patients, 15 patients were disease-free (62.5%) at the time of analysis, 8 patients were living with the disease (33.33%) and one patient died due to aetiology other than primary disease. One patient had locoregional recurrence of MTC 3 years after primary diagnosis and treatment.

Eight patients underwent ^{68}Ga-DOTATATE PET/CT on follow-up, based on the attending oncologists' referral. Whole-body SSTR-based PET/CT scan was performed after IV injection of 2-3mCi (74-111MBq) of ^{68}Ga-DOTATATE, using a whole-body full-ring dedicated PET/CT camera. A whole-body low-dose (50mA, 120 kVp) CT scan was acquired for attenuation correction and anatomical colocalisation. Images were reconstructed using an iterative algorithm. The patient with a calcitonin level of 20,862 pg/ml showed SSTR-expressing regional nodal disease. Another two patients showed non-SSTR expressing regional lymph nodes and non-SSTR expressing bilateral lung nodules at the level of calcitonin 370 pg/ml and 1333 pg/ml respectively, whereas 5 patients whose mean calcitonin level was 32.81pg/ml (ranging from 0.4 pg/ml to 84.19 pg/ml) did not show any residual disease.

DISCUSSION

Collision tumours of the thyroid gland, although rare, are being increasingly reported on histopathological examination. Among MTCs, simultaneous MTC/DTC increased from 2.7 % (in 1988-1997) to 12.3 % (in 2003-2008) (9). Several hypotheses have been postulated to explain the development of collision tumours. Lax et al proposed that they originate from a single pluripotent precursor cell. In the case of medullary and papillary thyroid cancers, the ultimobranchial body represents the embryological nest for such stem cells. (10) Another is 'chance theory', which postulates independent origin of two tumours and mere chance occurrence of one tumour next to an unrelated primary tumour. (11, 12) The third theory proposes that, the presence of first tumour alter the tumour microenvironment, which facilitates the development of second primary tumours. (13)

Our study showed that females were slightly more predominantly affected (58.3%). Other studies conducted by Ryan et al. (9) and Abdullah et al. (14) also showed a female predominance (77.77% and 75%, respectively). The study conducted by Ryan et al. showed that the mean age of occurrence was 53.4 years, a finding quite similar to that obtained in our study, which is 50.7 years. Age ranged from 27 to 84 years in their study, whereas in our study, age ranged from 26 to 90 years (9). All patients in our study presented with anterior neck swelling, which is the usual finding in other reported studies (9).

A retrospective analysis conducted by Kim et al. showed that 19% of the patients with MTC presented with concomitant PTC. They also showed that the incidence of concurrent PTC in patients with medullary carcinoma thyroid, Graves' disease, and follicular carcinoma thyroid are

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similar; they concluded that the concurrent occurrence of MTC and PTC might be a simple coincidence (15).

A retrospective study conducted by Thomas et al. in a tertiary care centre showed that collision tumours comprised 4.7% of all medullary carcinoma thyroid cases diagnosed over 10 years. Co-occurrence of MTC and PTC was the most common type of collision tumour. They also mentioned that the PTC component of collision tumours, which is most frequently a microcarcinoma and unifocal, is usually undiagnosed preoperatively. Therefore, they stressed the importance of adequate sampling, especially for grossly normal lobes, for accurate diagnosis. Their findings also supported the chance theory of co-occurrence. (13)

A study conducted by Biscolla et al. investigated 196 cases of MTC, of which 27 were collision tumours of MTC and PTC (13.8%). Our study showed that 24 of 470 patients were diagnosed with collision tumours of MTC with PTC (5.1%). The authors also stated that the presence of PTC with MTC did not change the outcome of MTC, which is similar to our findings. Additionally, RET gene assessment did not show any common mutations in these collision tumours. (16) Our study showed that 75% of the patients with collision tumours presented with lymph node metastasis. Among these, 80% were from the MTC component, which is comparable to the incidence of lymph node metastasis in sporadic MTC with palpable neck nodules (70%). In our study, the incidence of distant metastasis was 8.3%, which was comparable to the incidence of distant metastasis in sporadic MTC with a palpable neck nodule (10%). (17)

Owing to the rarity of collision tumours, treatment guidelines are poorly defined, and these tumours are generally managed as two separate entities. (13) If an MTC diagnosis is made on FNAC, baseline measurements of serum calcitonin and carcinoembryonic antigen are indicated. If

calcitonin levels are greater than 500 pg/ml or there is clinical suspicion of metastatic disease, further imaging studies are needed. Total thyroidectomy is the definitive treatment for primary disease. Therapeutic central and lateral compartment neck dissection was performed for lymph node metastasis. Prophylactic neck dissection should be performed based on the T stage of the primary disease and serum calcitonin levels. Adjuvant treatment by RAI therapy should be performed if the PTC component falls in the intermediate- and high-risk categories according to ATA risk stratification. RAI plays no role in MTC management.

Adjuvant radiotherapy for differentiated thyroid cancer is recommended in patients with gross residual or unresectable locoregional disease. EBRT has not been routinely considered as an adjuvant therapy after complete resection of the gross disease. Cervical lymph node involvement alone is not an indication for adjuvant EBRT (18). For MTC component, adjuvant EBRT was carried out for unresectable gross residual disease, gross extra-thyroidal extension, macroscopic multifocal disease, macroscopic tumour invasion, microscopic residual disease and multiple lymph nodal metastasis and extra-nodal spread in nodal metastasis (19)

Measurement of Serum Calcitonin and carcinoembryonic antigen (CEA) should be performed approximately 3 months after surgery. If the values are normal, they should be repeated 6-12 months later. (20). Persistently elevated serum calcitonin and carcinoembryonic antigen levels were suggestive of residual/recurrent disease. In such cases, anatomical and functional imaging like SSTR-based PET/CT is recommended, the advantage of functional imaging being that it is a whole-body imaging, and enables better diagnosis of tumour recurrence/metastasis that could be missed by anatomical imaging. Although Kim et al. (15) suggested that collision tumours behave more aggressively than singleton tumours, we found that metastatic incidence and survival rates are similar to those of singleton pathology.

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We consider our sample size to be limited considering the rarity of the disease, and fewer data are available from the MTC-predominant group that showed metastatic disease. The retrospective nature of this study is another limitation.

Based on the current available literature (2,13,17,20) and our experience, we have put up a simplified scheme for managing these tumours (Fig 1), which suggests that decision making should start at the pre-operative stage in the form of suspicious multisite FNAC and tumour marker values (both serum Tg and serum Calcitonin). However, in most cases, the diagnosis of a collision tumour is established by cautious postoperative histopathological examination. Here, treatment protocols for both tumour types are considered, primarily based on the disease extent and pathological risk category. Imaging evaluation of both tumour components, that is, with RAI scan and SSTR-based PET, should be performed meticulously to improve sensitivity for the detection of any residual/metastatic disease. Further treatment plans based on multiple available treatment options for both tumour types should be decided using a multidisciplinary approach. Considering the aggressive nature of MTC compared to its differentiated/PTC counterparts, when residual/metastatic lesions are positive on SSTR-PET, aggressive options such as re-exploration/TKI/adjuvant chemotherapy/PRRT should be considered based on the disease stage; simultaneously, RAI scan should be performed to look for another counterpart. Implementing and sequencing both therapies should not always follow the individual guidelines of one counterpart; in fact, it should be decided on a case-to-case basis by a multidisciplinary team. On commencement of these therapies, follow-up (both imaging and tumour markers) of both counterparts should be performed irrespective of their risk category and baseline staging. Here, we recommend follow-up USG, RAI scan, S.Tg level along with SSTR-PET, and serum calcitonin level based on the risk category. The standard thyroid hormone suppression therapy will go along hand in hand with this

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surveillance; no test is a replacement of other components; for example, the USG neck should not replace the tumour markers or PET examination. The duration and frequency of these follow-up visits should also be decided on a case-to-case basis by the team.

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CONCLUSION:

In summary, thyroid collision tumours in the setting of MTC are a relatively uncommon entity, in which the most common combination is MTC with PTC. This is more commonly observed in women than in males. This tumour is usually undiagnosed preoperatively, and histopathology is the main modality for accurate diagnosis. Lymph node metastasis is common in cases of collision tumours; among these, metastasis from the MTC component is the most common. Although lymph node metastasis from both MTC and PTC in the same patient has been documented, its incidence is rare. The prognosis of the disease depends on the aggressiveness of the MTC. Follow-up of both components should be performed biochemically using serum tumour markers (i.e. Tg and Calcitonin) and imaging, especially molecular imaging approaches in addition to neck USG (i.e. RAI scan and Somatostatin Receptor Imaging PET-CT scan) according to the risk category, tumour marker profile, and staging of the disease.

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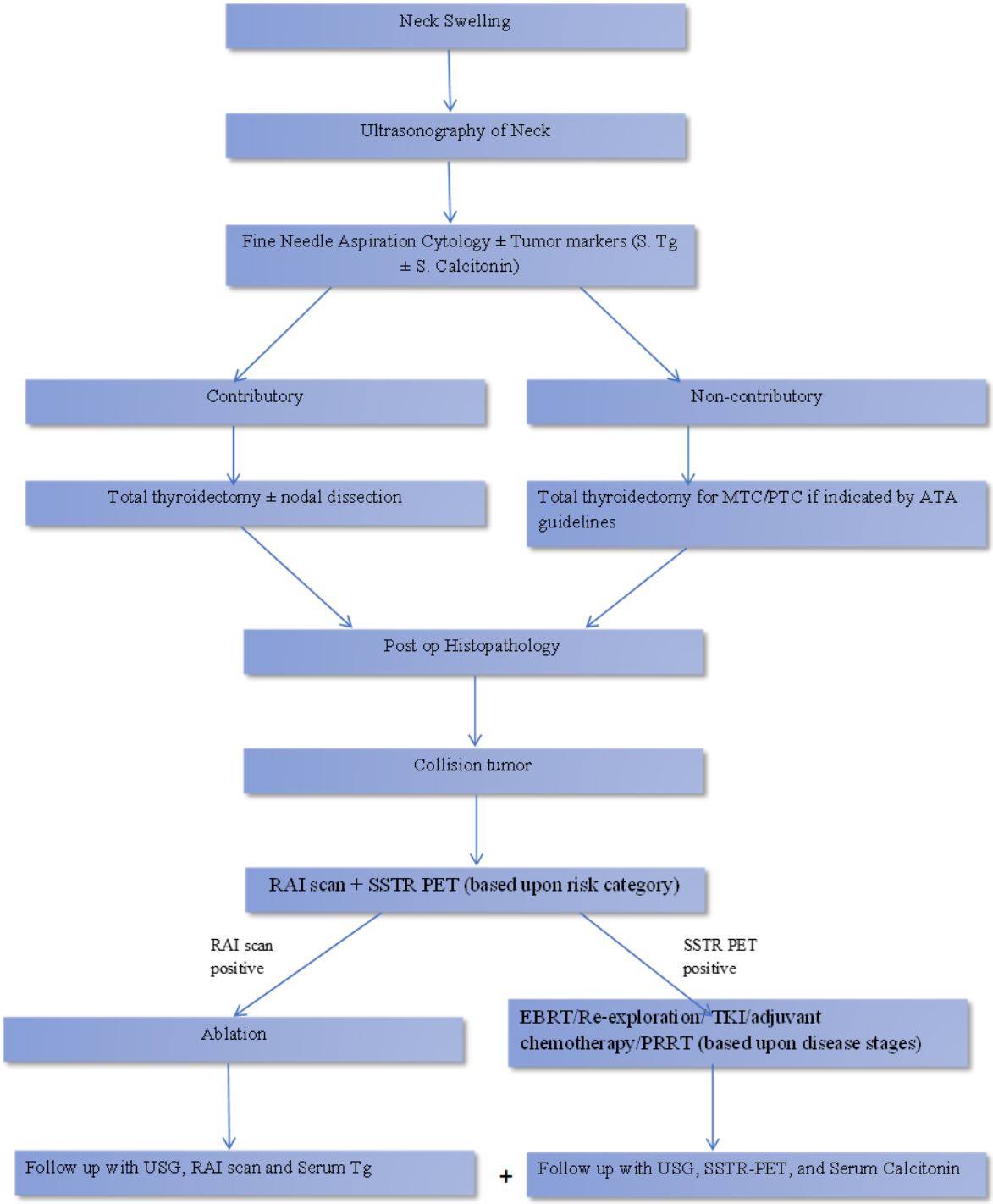

Fig 1. A proposed simplified schema for managing Collision Tumours of Thyroid

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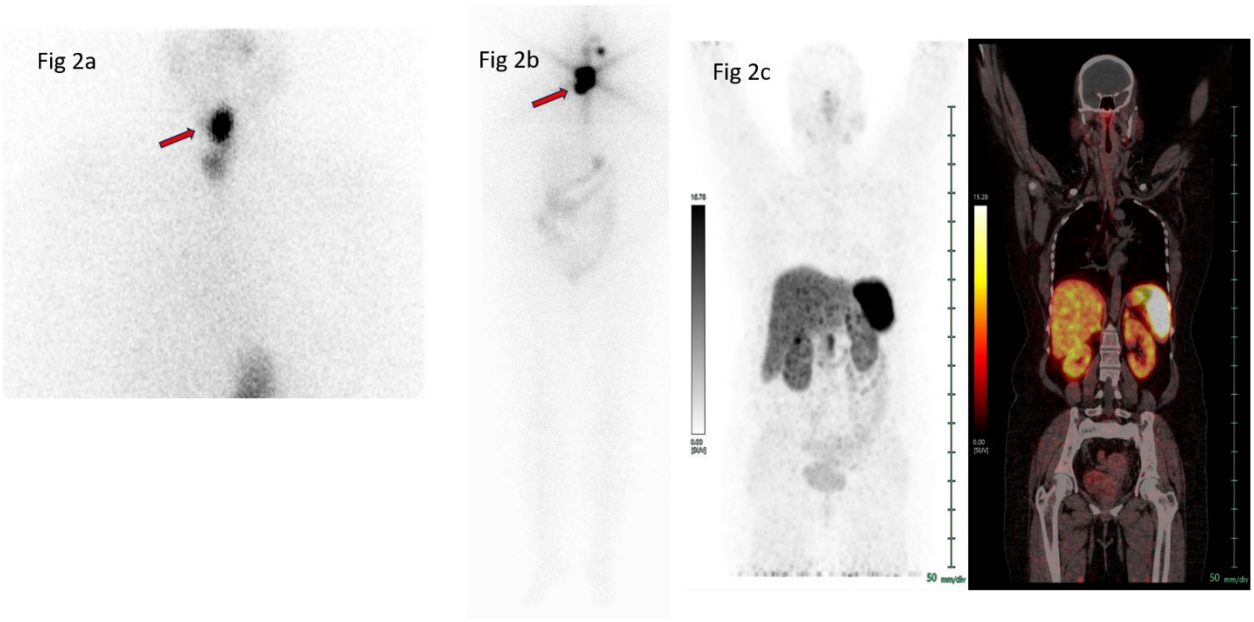

Fig 2. A 43-year-old female patient with histopathology report of right lobe of thyroid lesion 1 showing invasive encapsulated follicular variant of papillary thyroid carcinoma, right lobe of thyroid lesion 2 showing medullary thyroid carcinoma with capsular invasion, and left lobe lesion showing differentiated papillary thyroid carcinoma, classical type. The patient underwent a total thyroidectomy. Postoperative stimulated Tg was 8.23 ng/ml and S. Calcitonin was 0.53 pg/ml. 1mCi (37MBq) radioactive iodine scan showed a bifocal abnormally increased tracer uptake noted in the neck **(Fig 2a)**, whereas the ^{68}Ga-DOTATATE PET/CT scan showed no abnormal SSTR-expressing lesion noted in the whole-body scan **(Fig 2c)**. Patient received 146mCi (5402MBq) of high-dose radioactive iodine therapy, and a post-therapy scan showed abnormal bifocal tracer uptake in the neck region **(Fig 2b).**

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Fig : 3a

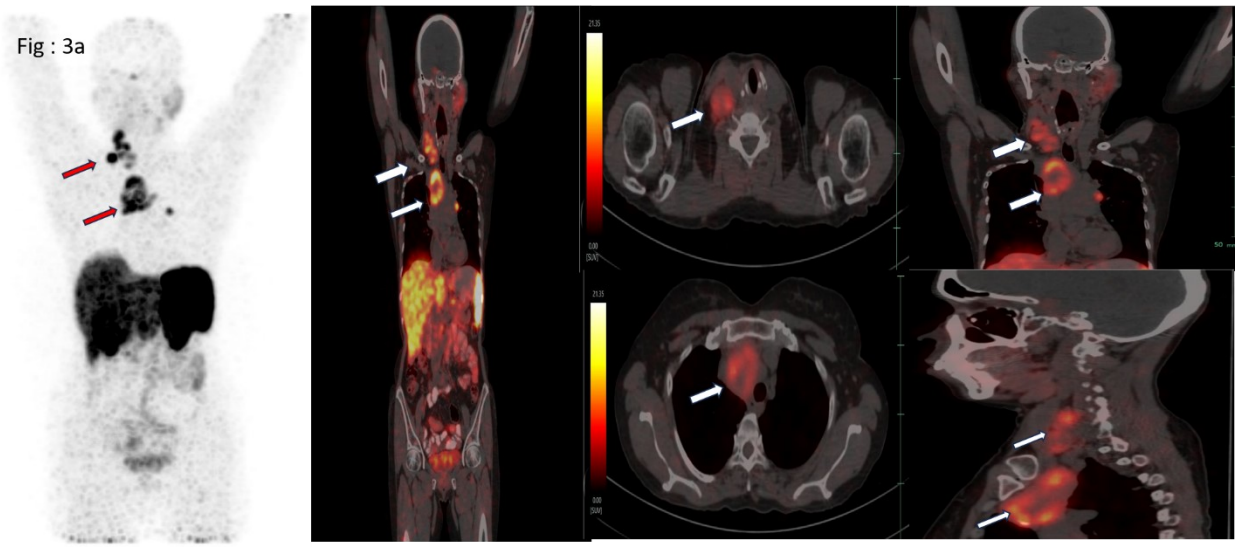

Fig : 3b

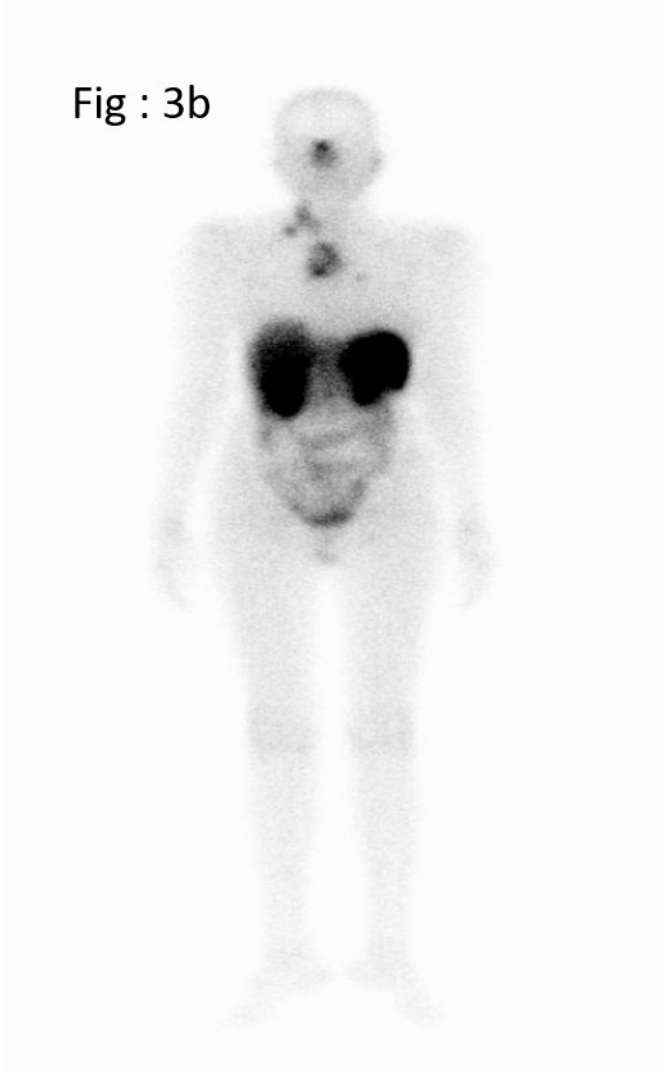

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Fig 3. A 46-year-old female patient presented with a histopathological report of medullary carcinoma of the right lobe and papillary carcinoma of the thyroid of the left lobe. She had received radioactive iodine at 104mCi (3848MBq) 10 years previously. A post-therapy scan revealed focal abnormal tracer uptake in the neck region. Subsequently, the patient had defaulted for 10 years. The patient presented with neck swelling for last 1 year. USG the of neck outside, with FNAC of the right supraclavicular swelling showing medullary carcinoma thyroid. Serum calcitonin level was 20,842 pg/mL.^{68}Ga DOTATATE PET/ CT scan showed SSTR expressing right cervical level III/ IV lymph nodes and mediastinal lymph nodes; Krenning's score: 3 **(Fig 3a)**. The patient received 188mCi (6956MBq) of ^{177}Lu-DOTATATE IV in view of Krenning's score of three lesions. A post-therapy scan showed tracer uptake in the cervical and mediastinal lymph nodes**. (Fig 3b).**

SUMMARY STATEMENT/BULLET POINTS:

- Thyroid collision tumor is a relatively rare entity, in which the most common combination is medullary thyroid carcinoma (MTC) with papillary thyroid carcinoma (PTC). The management guidelines for these tumours are poorly defined in view of its rarity.

- Lymph node metastasis is common in collision tumors; among them, metastasis from MTC component is more common. The prognosis of the disease primarily depended upon the tumor aggressiveness of MTC.

- For residual/metastatic lesions positive on SSTR-PET, definitive treatment options like re-exploration/TKI/adjuvant chemotherapy/PRRT should be considered.

    - Follow-up with biochemical and imaging markers for both tumors should be carried out (i.e., serum thyroglobulin and serum calcitonin with Neck USG, RAI scan and Somatostatin Receptor based PET-CT according to risk category).

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