Case Report

When Palpitations Unmask Crista Terminalis Hypertrophy: A Case Report and Review of Current Literature

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When Palpitations Unmask Crista Terminalis Hypertrophy: A Case Report and Review of Current Literature

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Abstract

Background: The crista terminalis (CT) is a fibromuscular ridge forming the embryonic boundary between the sinus venosus and the primitive right atrium. While a physiological structure, rare cases of CT hypertrophy present a diagnostic challenge, often appearing as a right atrial pseudo-mass on imaging. Given its arrhythmogenic potential, known to be associated with two-thirds of right atrial arrhythmias, distinguishing hypertrophy from pathological masses is clinically vital. **Case Presentation:** We present a case of a 58-year-old female who was referred to our hospital for rapid, irregular palpitations, accompanied by hypertension. Transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) revealed an isoechoic right atrial mass, attached to the posterolateral wall of the right atrium, with a broad base of implantation and no intrinsic mobility. To exclude high-risk diagnoses, such as thrombi and myxomas, as well as other common right atrial mass mimics like the Chiari network, cardiac computed tomography angiography (CCTA) was performed. CCTA provided high-resolution tissue characterization, confirming the mass as a hypertrophied CT by its lack of contrast enhancement and its precise anatomical orientation. **Discussion:** A literature review of 12 cases confirms a clinical phenotype characterized by a female predominance (66.67%) and a mean diagnostic age of 58 years, which our case reinforces. While a hypertrophied CT is a benign anatomical variant, it serves as a significant arrhythmogenic substrate through conduction anisotropy, enhanced cellular automaticity, and its anatomical link to the sino-atrial (SA) node. Morphologically, it poses a diagnostic challenge, since it appears on TTE as a right atrial mass. However, we demonstrate that TEE provides essential visualisation of the right atrium, while CCTA and cardiac magnetic resonance imagery (CMR) offer definitive tissue characterization to exclude neoplastic or thrombotic processes. This report bridges the gap between imaging and electrophysiology, highlighting that CT thickness is not merely an incidental finding but a possible predictor of electrical instability and arrhythmia recurrence. **Conclusions:** This case reinforces the epidemiological profile of CT hypertrophy, which predominantly affects females in their sixth decade. It highlights the advantages of a multi-modal imaging approach, transitioning from TEE to CCTA or CMR, to prevent unnecessary invasive interventions or anticoagulation. Meanwhile, it also seeks to reinforce the need to establish clear characteristics of CT hypertrophy on TEE, making 3D TEE the sole necessary imagery tool for diagnosis in the future. Furthermore, this report supports the hypothesis that structural hypertrophy may exacerbate the CT's underlying arrhythmogenic

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potential, possibly through enhanced conduction anisotropy. Further research is needed to establish the correlation between CT thickness and the severity of atrial tachyarrhythmias

Keywords: crista terminalis hypertrophy; right atrial mass; atrial tachyarrhythmia; diagnostic imaging; differential diagnosis; anatomical variant

1. Introduction

The CT is a C-shaped fibromuscular ridge located in the right atrium, arising during embryogenesis and forming the boundary between the primitive right atrium and the sinus venosus [1]. Clinically, the CT is important due to its ability to mimic a right atrial mass and to its arrhythmogenic potential.

In most patients, this structure regresses to an approximate size of 3-6 mm by adulthood [2]. In rare cases, the CT is hypertrophied and leads to diagnostic confusion with life-threatening conditions such as thrombi or myxomas [3]. Proper identification of this benign structure is therefore essential in avoiding unnecessary anticoagulation, invasive biopsies, or cardiac surgery.

Due to its unique electrophysiological properties, the CT is a major site of origin for right atrial tachyarrhythmias, accounting for two-thirds of cases in patients without structural cardiac abnormalities [2,4]. Mechanisms responsible for these arrhythmias include focal automaticity and conduction anisotropy, both of which are thought to be aggravated by age-related remodelling [4]. Since CT arrhythmias have been documented in patients with and without concomitant CT hypertrophy, it is uncertain if this increase in tissue contributes to the worsening of atrial fibrillation.

In this case, a patient presenting with palpitations prompted imaging that revealed a pseudo-mass in the right atrium, eventually confirmed as being a hypertrophied CT. The aim of this report is to add to the bank of currently available literature on the topic, in the hopes of streamlining diagnosis in future patients, and adding to the data identifying the CT as an important location of origin of atrial tachyarrhythmias.

2. Detailed Case Description

A 58-year-old female patient presented to another medical service for palpitations with a rapid, irregular rhythm that had begun approximately one month earlier, accompanied by elevated blood pressure values. The patient had no cardiovascular risk factors and no previously known cardiac pathology.

12-lead electrocardiography (ECG) showed sinus rhythm, heart rate of 60 bpm, no ST-segment modifications and no T-wave modifications. 24-hour Holter ECG showed self-limited episodes of atrial tachycardia, reaching a maximum heart rate of 170 bpm.

Following the incidental finding on TTE of a right atrial mass, TEE was performed. TEE revealed a 12 x 9 mm echogenic and relatively homogeneous structure at the level of the lateral right atrial wall, near the junction of the inferior vena cava (IVC) (Figure 1). The lesion a presented a broad base of implantation, with regular margins and no intrinsic mobility. Color Doppler imaging showed no internal flow, suggesting a non-vascularized structure. While the appearance was non-specific, an initial differential diagnosis of an atrial lipoma was considered.

Additional TEE findings included a non-dilated left atrium and a thrombus-free left atrial appendage with preserved Doppler flow. The left ventricle was not dilated, with a preserved ejection fraction (LVEF > 55%) and no regional wall motion abnormalities. The mitral valve leaflets were supple, showing only trace/mild regurgitation. Dimensions of the aortic root and ascending aorta were within normal limits.

CCTA was subsequently performed for better tissue characterization, results are shown in Figure 2. The calcium score was 76.3, indicating low cardiovascular risk. Analysis of the coronary arteries revealed right-dominant circulation with mild, non-obstructive atherosclerotic disease

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(CAD-RADS 1/P1). Specifically, the left anterior descending artery (LAD) and the second diagonal branch (D2) exhibited partially calcified and calcified plaques, resulting in minimal luminal stenosis (10-20%). The left main, circumflex, and right coronary arteries were unremarkable. Evaluation of the cardiac morphology revealed non-dilated chambers and no evidence of septal defects.

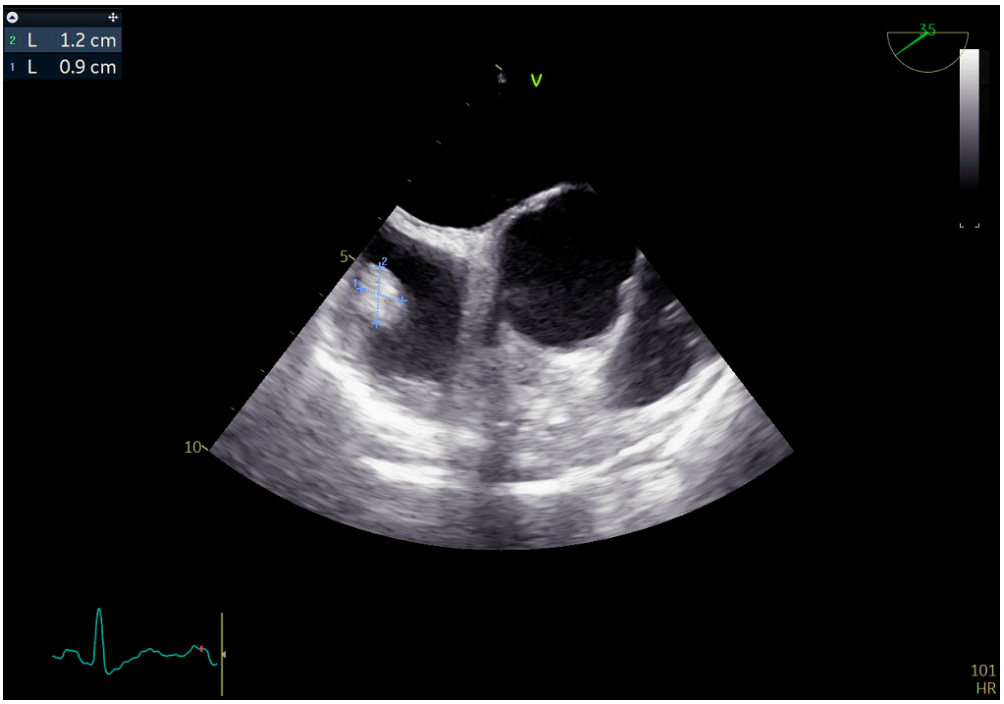

Figure 1. Transesophageal Echocardiography demonstrating a right atrial mass.

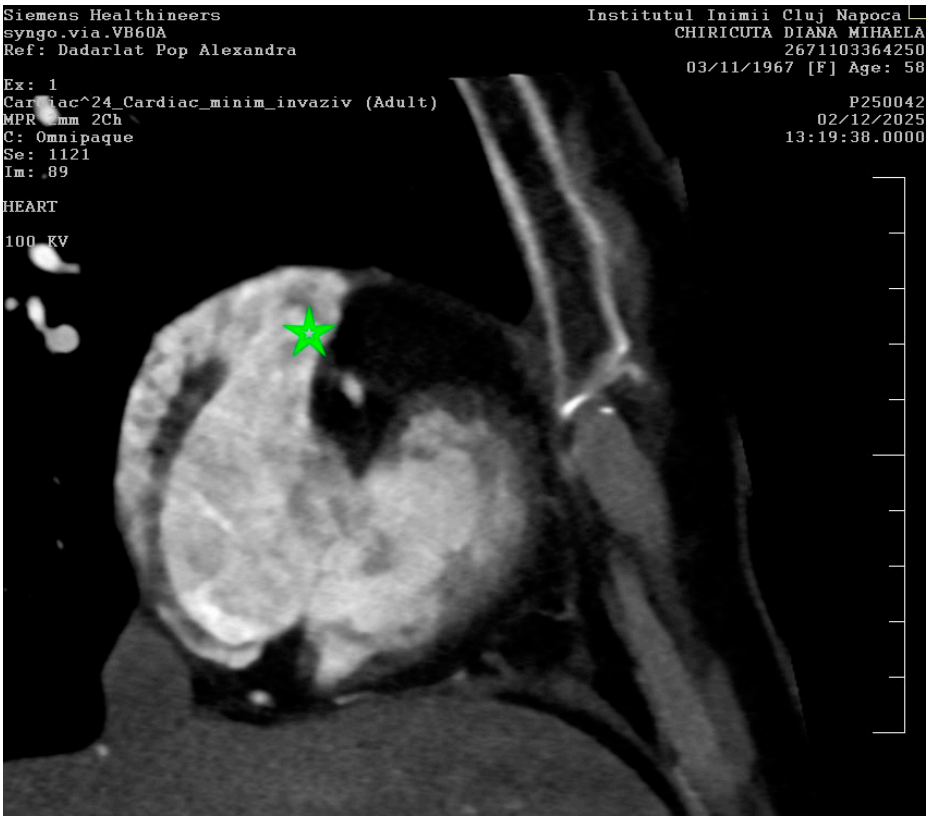

Figure 2. Contrast-enhanced CT demonstrates a non-enhancing intracavitary mass within the right atrium, presenting as a well-circumscribed filling defect, highlighted by the green marker.

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Notably, the examination confirmed a prominent, hypertrophic crista terminalis with a characteristic congenital appearance. There was no evidence of abnormal tissue enhancement or calcification within the right atrium, effectively ruling out a pathological mass. Extracardiac findings were limited to mild paraseptal emphysema in the pulmonary apices and minimal non-calcified atheromatosis of the thoracic aorta.

3. Discussion

3.1. Crista Terminalis Hypertrophy in the Current Literature

A literature review was conducted to identify cases of CT hypertrophy, using the PubMed database and keywords "crista terminalis" (Figure 3). An iterative search strategy was employed to ensure maximum capture of relevant cases. To ensure the reproducibility of our literature review and prioritize the most accessible clinical data for the general practitioner, inclusion was limited to free full-text articles. Database results were screened and records that did not present a case report and case reports detailing pathologies other than a hypertrophied CT were excluded. 12 freely available case reports examining this pathology remained [1–3,5–13].

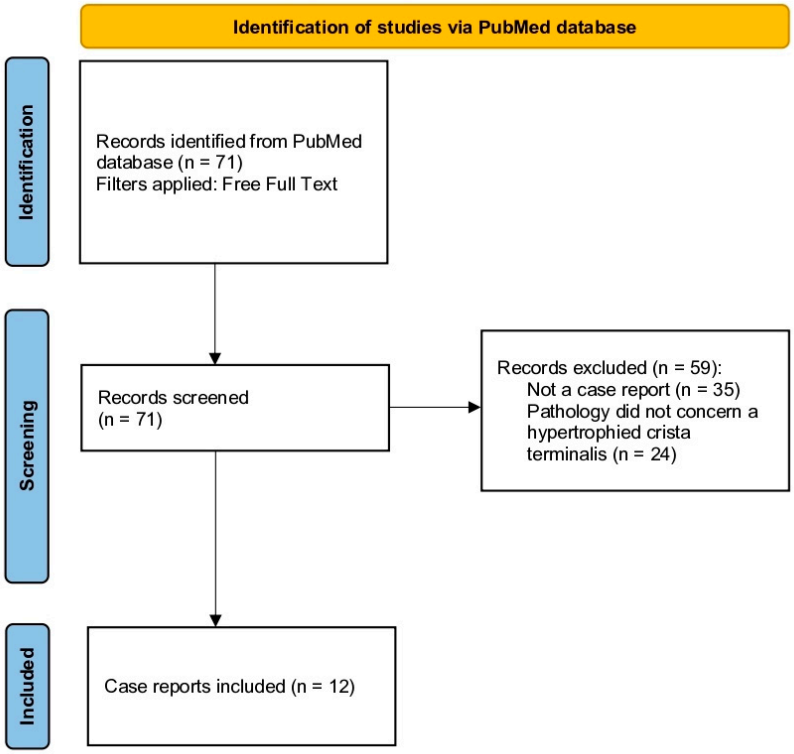

Figure 3. PRISMA flow diagram showing article selection.

Table 1 summarizes the sex and age of the patients, if concomitant arrhythmia was present, and the key imaging techniques used to establish a diagnosis. In the available literature, 66.67% (6/9) of adult patients with crista terminalis hypertrophy were female, 22.22% (2/9) presented a concomitant atrial tachyarrhythmia, and ages ranged from 26 to 79 [1,2,5–10,13]. Mean age of diagnosis for the adults included in these case reports was 58.11 ± 17.74 [1,2,5–10,13].

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Table 1. Epidemiological data and presence of concurrent arrhythmia in published case reports concerning crista terminalis hypertrophy.

References	Sex (F/M)	Age	Concomitant Arrhythmia	Key Imaging
Pati et al. (2025) [12]	NR[1]	Fetus (32w)	NR	Prenatal ultrasound
Nalawade et al. (2024) [6]	F	65	NR	Real-time 3D TEE
Shoji *et al*. (2024) [13]	M	57	NR	Real-time 3D TEE
Ghesani et al. (2023) [8]	F	79	NR	18F-FDG PET-CT[2], TEE
Bhatia et al. (2021) [11]	NR	Fetus (23w)	NR	Prenatal ultrasound
Lakhani *et al*. (2021) [7]	M	78	NR	CMR
Ahmed et al. (2020) [5]	F	59	Atrial fibrillation	CCTA
Evong et al. (2018) [3]	NR	Fetus (20w)	NR	Prenatal ultrasound
Wang et al. (2018) [9]	M	54	NR	Cardiac PET/MRI
Salim et al. (2016) [1]	F	32	Paroxysmal atrial fibrillation	CMR
Na et al. (2011) [10]	F	73	NR	TEE
Salustri et al. (2010) [2]	F	26	NR	TEE

[1] NR = not reported; [2]18F-fluorodeoxyglucose positron emission tomography.

Of the 9 cases present in adults, only 3 were recorded in male patients and all 3 of these patients did not present a concomitant arrhythmia [7,9,13]. This tendency to present silently in males could explain certain outliers among female patients, notably the case report concerning a 32-year-old female with paroxysmal atrial fibrillation [1]. If CT hypertrophy is mainly silent in males, it follows that age at diagnosis is closer to the age at which they would first undergo a full cardiovascular assessment. However, if there does exist a link between a hypertrophied CT and atrial tachyarrhythmias, patients would seek medical advice earlier in life, at the onset of symptoms. 3 of the 12 cases concerned fetuses [3,11,12]. These cases highlight the need to distinguish between the mechanisms by which a prominent CT may arise. The CT could present a failure of complete regression during embryogenesis, or it could present over-development. Cases in fetuses provide an interesting avenue for following the development of this prominent CT over time. Evong et al. (2018) and Pati et al. (2025) detail follow-up in infancy showing no progression in the size of the CT [3,12]. In the case described by Bhatia et al. (2021), however, the structure showed regression in infancy [11]. Hypertrophied CT is considered to be a benign anatomical variant, therefore close postnatal monitoring is not currently indicated [12]. Nevertheless, knowing a patient presents a congenital right atrial formation can help with the differential diagnosis of an intracardiac mass upon reaching adulthood.

3.1.1. Association with Atrial Tachyarrhythmia

A well-documented origin for atrial arrhythmias, the CT accounts for two-thirds of right atrial arrhythmias in patients without structural cardiac abnormalities [1]. Clinical presentation of atrial tachyarrhythmias can, such as was the case here, lead to the discovery of a crista terminalis hypertrophy. Epidemiologically, the literature shows that patients are more commonly over 50 and female, with Morris et al. (2019) placing mean age at presentation at 56.9 ± 1.5 [4]. Our case report adds to this available data, as our patient was also over 50 and female, and presented concomitant atrial tachyarrhythmia. The CT being a structure that arises during embryonic development, its discovery in young patients is not rare. Salim et al. (2016) presented the case of a 32-year-old woman with paroxysmal atrial fibrillation, for whom a prominent crista terminalis was discovered during her pre-operative cardiology assessment [1]. Pati et al. (2025), Bhatia et al. (2021), and Evong et al. (2018) detail the discovery of right atrial masses in fetuses that were later confirmed to be prominent crista terminalis [3,11,12]. In the fetal and neonatal context, the differential diagnosis of right atrial masses is critical, as other pathologies such as cardiac rhabdomyoma can present with supraventricular extrasystoles, requiring surgical resection for symptom resolution [14]. The case of other right atrial masses leading to symptoms also favours the notion that CT hypertrophy, when it reaches a certain thickness or a precise anatomical region, may cause arrhythmias. In older patients,

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the association between the CT and atrial tachyarrhythmia is thought to be linked to age-related remodeling leading to slowed conduction [4]. This indicates that a prominent crista terminalis may be a risk factor for developing arrhythmias later in life.

The CT is an arrhythmogenic substrate for both micro- and macro-reentrant arrhythmias [15]. Physio-pathological mechanisms behind the arrhythmogenic potential of the CT include its relation to the SA node, conduction anisotropy, and cellular automaticity [4,16,17].

The CT corresponds internally to the sulcus terminalis, structure that houses the SA node [17]. Conduction velocity in the CT is high when compared to other regions of the right atrium and the SA node [18]. Abnormal thickening of the CT may therefore alter the way electrical impulses spread from the SA node. When measuring atrial wall thickness, the CT is found to be significantly thicker than the surrounding tissue [19]. Furthermore, the most common origin of atrial tachycardia is located at the mid-third of the structure, with superior and mid-sites being significantly more frequent origins than the inferior third [4]. Critically, these areas, the superior and mid-third of the CT, overlap with main SA node exits and earliest atrial activation sites [15]. In fact, the study of ex-vivo human hearts showed that most superior, mid-lateral, and inferior SA node exits were often located along the CT [15]. Mapping by Zhao et al. (2023) has identified that right atrial drivers of atrial fibrillation are consistently anchored on the CT at both superior and inferior sections of the right atrium [19].

Conduction anisotropy across the CT means that conduction velocity is fast longitudinally but slow transversely [16]. Sánchez-Quintana et al. (2002) describe how the CT acts as a natural barrier to transverse conduction, favorizing the appearance of macro-reentrant circuits as seen in atrial flutter [16]. A more recent study by Morris et al. (2019) suggests that this same anisotropy leads to micro re-entrant circuits [4]. Slow transverse conduction due to poor transverse cell-to-cell coupling leads to *both* micro- and macro-reentrant circuits [15]. The prototype of macro-reentrant atrial tachycardia is atrial flutter [20]. Typical atrial flutter arises from a circuit that links the tricuspid annulus anteriorly and the CT and eustachian ridge posteriorly [20]. Bhargav et al. (2025) describe a case of counterclockwise macro-reentry around the IVC with breakthrough at the lower end of the CT causing atypical atrial flutter [21]. A possible complication of lateral tunnel Fontan surgery is a macro-reentrant circuit around the baffle-CT suture line [22]. Age-related remodelling further contributes to this anisotropy due to further loss of gap junction proteins, already poorly expressed in the CT [4]. Thus, modification of the tissue of the CT can disrupt local electrical transmission.

Cellular automaticity in the CT is due to greater Hyperpolarization-activated Cyclic Nucleotide-gated (HCN) channel expression than in the surrounding myocardium [4]. While macro-reentrant tachycardia is often associated with structural heart disease, heart failure or ischemic cardiomyopathy, focal atrial tachycardia frequently occurs in healthy individuals without structural abnormalities [20]. Focal atrial tachycardia within this region is driven by three main mechanisms : enhanced automaticity, triggered activity, or micro-reentry [20]. When HCN channels are overactive, or if surrounding tissue is remodeled, focal automaticity can be triggered, leading to atrial tachycardias [4]. Specifically, enhanced automaticity results from an accelerated spontaneous phase 4 depolarization upslope [20]. When the cell is hyperpolarized, the activation of HCN channels triggers an influx of sodium and potassium that allows the cell to reach threshold [20]. Alternatively, triggered activity involves calcium channel dysfunction, while micro-reentry occurs between adjacent substrates with varying conduction velocities and repolarization rates, much like is the case for conduction anisotropy across the CT [20].

Catheter ablation was shown to be successful in treating these tachyarrhythmias [4,15]. With the addition of 3D mapping, ablation success rate is as high as 98.5% [4]. However, a retrospective study showed that CT thickness is an independent predictor of atrial flutter recurrence after radiofrequency ablation [23]. Specifically, a median CT thickness of 3.70 mm (IQR 3.30, 4.40) was associated with recurrence, compared to 3.40 mm (IQR 3.10, 3.90) in non-recurrent cases [23]. Furthermore, ultra-high-resolution 3D mapping has recently demonstrated the complexity of this barrier, revealing that unusual posterior transverse conduction can persist across the CT even after standard ablation procedures [24]. Possible risks of catheter ablation include proximity of the phrenic nerve to the right

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atrium [25]. However, this risk can be reduced by performing the ablation during inspiratory hold and using concomitant phrenic nerve monitoring [25].

3.1.2. Diagnostic Imaging and Differential Diagnosis

On transthoracic echocardiography (TTE), the CT is not always clearly visible [3]. When hypertrophied, it can mimic an abnormal right atrial mass, such as a thrombus or a tumour [3]. Other important right atrial mass mimics include the Eustachian valve, Thebesian valve, persistent sinus venosus, atrial septal aneurysms, and the Chiari network [1]. An embryological structure similar to the CT, the Chiari network presents as a mobile, fenestrated, and thread-like structure [26]. Phuyal et al. (2025) recently highlighted how its high mobility and web-like appearance often lead to its initial misidentification as a thrombus or infective vegetation, mirroring the diagnostic challenges posed by a prominent CT [26].

Following identification of a suspicious mass on TTE, TEE provides superior visualisation. Clearly defining the echocardiographic characteristics of a hypertrophied CT reduces diagnostic uncertainty and may preclude the need for additional imaging in straightforward cases, particularly when 3D and 4D TEE are utilized. A hypertrophied CT typically appears as a C-shaped muscular band or well-defined fibromuscular ridge with a broad base of insertion, located on the posterolateral wall of the right atrium [2]. The CT originates near the medial border of the superior vena cava (SVC) and extends along the posterolateral wall towards the IVC [27]. It is typically isoechoic to the adjacent myocardium [6]. The CT separates two visually distinct areas : the trabeculated and non-trabeculated regions of the right atrium [28]. 3D TEE can further clarify its role as a boundary between the smooth venous component and the rough pectinate muscles of the right atrial appendage [29]. There are currently no formal diagnostic criteria for what constitutes a prominent CT, however, a normal CT in adults has a mean length of 51 ± 9 mm and thickness of 5.5 mm [27]. The mass does not enhance with contrast, and tissue Doppler imaging shows no aberrant flux [27]. The CT exhibits phasic changes in shape during atrial systole but lacks independent mobility, moving in synchronicity with the cardiac cycle [6].

Further imaging using CCTA or CMR can provide definitive tissue characterisation [7]. CCTA provides high special resolution and rapid image acquisition [7]. It is particularly useful for identifying the relationship between the CT and the SVC, as well as its role as a line of conduction block [7,30]. While it involves ionizing radiation and iodinated contrast, it is often more accessible and faster than MRI [7]. CMR is considered a preferred diagnostic tool due to its excellent contrast resolution and lack of ionizing radiation [7,31]. It can differentiate the CT from neoplasms or thrombi by confirming that signal intensity is identical to that of normal myocardium and showing no abnormal gadolinium enhancement [9]. However, its use is limited by higher costs, longer scan times, and incompatibility with certain intracardiac devices [7]. In the case of our patient, initial TTE suggested a potential intra-atrial mass while TEE raised suspicion for a lipoma due to its echogenicity and sessile attachment. Subsequent CCTA provided definitive characterization. The mass was identified as a prominent, hypertrophic crista terminalis, a well-documented pseudotumor of the right atrium. The definitive diagnosis was supported by the CCTA's ability to demonstrate the structure's characteristic anatomical course along the posterolateral wall and its lack of abnormal contrast enhancement or fatty infiltration, which effectively ruled out neoplastic or thrombotic processes.

While sometimes confirmation by CCTA or CMR is necessary, the importance of TEE as a diagnostic tool should not be overlooked. Real-time 3D TEE is recognized for its precision when measuring anatomical structures of the right atrium [32]. Hassan et al. (2023) discovered that annular dimensions measured with 4D TEE matched strongly with those measured using multi-detector computer tomography (MDCT) [33].

In complex cases, 18-FDG PET/CT may be utilized. A hypertrophied CT typically shows no abnormal FDG uptake, helping rule out malignant tumours or inflammatory masses [6,9]. However,

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some cases have noted focal uptake that can masquerade as a tumour thrombus, requiring further verification by TEE or angiography [8].

3.2. Significant Contributions

Our case significantly contributes to the sparse literature on symptomatic hypertrophic CT by reinforcing a specific clinical and demographic phenotype. The patient's profile, a 58-year-old female, aligns precisely with the demographic data shown in the above literature review. The mean age of diagnosis at 58 and the female predominance noted in the aforementioned review support that a prominent CT should be considered a primary differential in women in their sixth decade presenting with unexplained palpitations and a right atrial finding.

Furthermore, while existing literature focuses on either the importance of imaging or the electrophysiology of the CT, our report bridges these two domains. We demonstrate the diagnostic dilemma, how a benign anatomical structure can lead to the suspicion of a high-risk mass on initial imagery, and highlight the possibility that the hypertrophy of this structure could lead to electrical instability.

Our diagnostic pathway underscores the central role of TEE as the first-line modality for evaluating right atrial structures, while validating CCTA as a helpful confirmatory tool in cases of diagnostic uncertainty. By providing superior spatial resolution and tissue characterization, CCTA allows clinicians to definitively rule out malignancy or benign pseudo-tumours, such as lipomas, through the absence of contrast enhancement or fatty infiltration. Crucially, CCTA offers the unique advantage of simultaneously assessing coronary anatomy, thereby excluding ischemic triggers as a potential cause for the patient's palpitations. Finally, our findings advocate for the broader clinical adoption of 3D and 4D TEE, which facilitate the definitive anatomical confirmation of a prominent crista terminalis by providing a photorealistic, volumetric perspective that traditional 2D imaging lacks.

4. Conclusions

The CT is a benign, physiological structure located in the right atrium. However, hypertrophy and the arrhythmogenic potential of this structure can lead to clinical repercussions.

A hypertrophied CT mimics a right atrial mass, requiring a mandatory differential diagnosis with life-threatening aetiologies such as thrombi and tumours [3]. First-line TTE is often insufficient to differentiate CT from high-risk masses [3]. TEE is a powerful tool that provides superior tissue visualisation, however clear diagnostic criteria for identifying hypertrophied CT need to be established. Currently, advanced cross-sectional imaging by CMR or CCTA is valuable, allowing for greater tissue characterization and confirming the diagnosis of CT hypertrophy [7]. In the future, hypertrophic CT identification will likely necessitate only 3D or 4D TEE, further accelerating the time it takes to establish a diagnosis.

The CT is frequently the origin of atrial tachyarrhythmias, through mechanisms such as anisotropy and cellular automaticity, as well as through its anatomical proximity to the SA node [4,16,17]. Steps have been taken towards better understanding the link between CT thickness and arrhythmogenic potential, showing that recurrence post-ablation is more frequent once CT thickness surpasses 3.70 mm [23]. Further research is needed to properly understand the relationship between CT hypertrophy and the induction of arrhythmogenic events.

In conclusion, our case highlights that a hypertrophied CT should be considered in the differential diagnosis of a right atrial mass, especially in female patients in the sixth decade with a history of atrial tachyarrhythmia. Use of multi-modal imaging to confirm this benign anatomical variant can spare the patient from unnecessary and potentially invasive interventions.

Supplementary Materials: The following supporting information can be downloaded at the website of this paper posted on Preprints.org. Video S1, Video S2.

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Informed Consent Statement: Written informed consent for publication was obtained from the patient.

Data Availability Statement: The original contributions presented in this study are included in the article/supplementary material. Further inquiries can be directed to the corresponding author(s).

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

CT	Crista terminalis
TEE	Transesophageal echocardiography
CCTA	Cardiac computed tomography angiography
CMR	Cardiac magnetic resonance imagery
ECG	Electrocardiography
SA	Sino-atrial
HCN	Hyperpolarization-activated Cyclic Nucleotide-gated
SVC	Superior vena cava
IVC	Inferior vena cava

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