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Case report

Hereditary cardiac amyloidosis: a case report

Amiloidose cardíaca hereditária: relato de caso

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Abstract

Objective: to report a case of hereditary transthyretin cardiac amyloidosis (ATTRv) in order to contribute to awareness, early diagnosis, and therapeutic management of the condition. **Case report:** a 56-year-old male patient presented with fatigue and dyspnea on minimal exertion since July 2024, with a family history and positive genetic testing for cardiac amyloidosis (TTR gene). He has a history of comorbidities including hypertension, prediabetes, and obesity, and is on regular medication. On physical examination, he was hemodynamically stable and had no significant abnormalities on cardiac and pulmonary auscultation. The electrocardiogram showed right bundle branch block and left anterior fascicular block. Echocardiography demonstrated ventricular hypertrophy, enlarged left-sided chambers, and mild diastolic dysfunction with preserved systolic function. The echocardiogram revealed concentric ventricular hypertrophy, increased volume of the left chambers, preserved systolic function (ejection fraction of 66%), and left ventricular diastolic function alteration. Sequencing of the TTR gene for hereditary transthyretin amyloidosis showed a susceptibility result for amyloidosis associated with the TTR gene (OMIM#105210). The patient remained under follow-up at the health unit with a stable clinical condition and was also referred to the cardiology service. **Conclusion:** ATTRv is still an underdiagnosed condition but of significant clinical relevance. Early identification enables the timely initiation of therapies that can modify the course of the disease, delay the progression of cardiomyopathy, and improve patients' quality of life.
Keywords: Amyloidosis. Heart disease. Congestive heart failure.

Resumo

Objetivo: relatar um caso de amiloidose cardíaca hereditária por transtirretina (ATTRv) com intuito de contribuir para a conscientização, o diagnóstico precoce e o manejo terapêutico da condição. **Relato de caso:** paciente masculino, 56 anos, com fadiga e dispneia aos mínimos esforços desde julho de 2024, com histórico familiar e teste genético positivo para Amiloidose Cardíaca (gene TTR). Histórico de comorbidades, como hipertensão, pré-diabetes e obesidade, em uso de medicação regular. Ao exame físico, estável hemodinamicamente e sem alterações significativas na ausculta cardíaca e pulmonar. O eletrocardiograma mostrou bloqueio de ramo direito e hemibloqueio anterosuperior esquerdo. Ecocardiograma evidenciou hipertrofia ventricular, câmaras esquerdas aumentadas e disfunção diastólica leve com função sistólica preservada. O Ecocardiograma (ECO), revelou hipertrofia concêntrica ventricular, câmaras esquerdas com aumento de volume, função sistólica preservada (fração de ejeção de 66%), função diastólica do ventrículo esquerdo. O exame de sequenciamento de gene TTR para Amiloidose Hereditária Ligada à Transtirretina apresentou resultado de susceptibilidade para amiloidose associada ao gene TTR (OMIM#105210). O paciente manteve o acompanhamento na unidade de saúde, com quadro clínico estável e foi encaminhado ao também ao serviço de cardiologia. **Conclusão:** a ATTRv é uma condição ainda pouco diagnosticada, mas de grande relevância clínica, cuja identificação precoce possibilita a introdução oportuna de terapias que podem modificar o curso da doença, retardar a progressão da cardiomiopatia e melhorar a qualidade de vida dos pacientes.
Palavras-chave: Amiloidose. Cardiopatia. Insuficiência cardíaca congestiva.

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Introduction

Amyloidosis is a term that defines a disease with a heterogeneous group of subtypes (hereditary or acquired, localized or systemic) due to alterations in protein metabolism, resulting in the extracellular deposition of certain polypeptides in a characteristic abnormal fibrillar form, altering tissue architecture and inducing functional changes, which can affect different organs[1].

The term cardiac amyloidosis (CA) is the result of amyloid deposition in myocytes, which causes cardiomyopathy with restrictive pathophysiology[2]. Although several types of amyloidosis can affect the heart, two types predominate: immunoglobulin light chain amyloidosis and transthyretin amyloidosis (ATTR). Cardiac involvement is the main cause of morbidity and mortality in systemic amyloidosis, regardless of the underlying pathogenesis of amyloid production[3].

CA is considered a rare and often neglected condition[7]. Transthyretin amyloidosis has an increased incidence in patients over 70 years of age, is more common in males and in the African-American population[5,7]. Light chain amyloidosis has an estimated incidence of 6 to 10/million people/year, being more common in men; and has a worse prognosis due to its aggressive nature[5]. Regarding diagnosis, studies indicate that cardiac amyloidosis may be underdiagnosed, with a late diagnosis rate of more than 2 years after the onset of symptoms, which hinders early treatment and effective management of the disease[5,7].

The initial symptoms of CA can be insidious, with nonspecific complaints. However, as the disease progresses, these symptoms become more evident. Congestive heart failure is often the predominant picture[1]. CA is usually confused with other more common heart diseases, resulting in late or even incorrect diagnoses[3]. Arrhythmias are also common due to impaired electrical conduction of the heart[1]. Cardiac changes associated with involvement of other systems, such as nephrotic syndrome, peripheral neuropathy, and plasma cell dyscrasia, may be present4. The diversity and overlap of these often nonspecific clinical signs make early diagnosis difficult, and adequate laboratory and imaging investigations are essential for the confirmation of CA[6].

Since it is a rare condition, it is frequently underdiagnosed as its symptoms are similar to other cardiological conditions, leading the patient to develop serious complications such as congestive heart failure, arrhythmias, and involvement of other systems[4-6].

Documenting clinical cases is crucial for raising awareness, improving early diagnosis and therapeutic management, and contributing to the development of more effective diagnostic and treatment protocols, considering the clinical variability of the disease and recent advances[3,5]. The aim was to report a case of CA in order to contribute to awareness, early diagnosis and therapeutic

management of the condition, as well as to collaborate in the development of more effective treatment protocols.

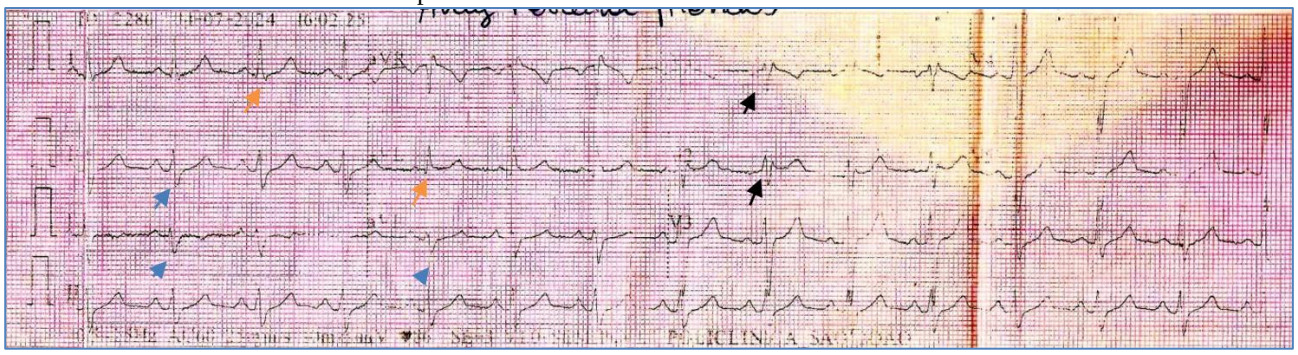
Case report

This work was approved by a Research Ethics Committee under opinion number 89543625.2.0000.5141.

A 56-year-old male patient from a municipality in northern Minas Gerais came to his primary health care unit with the main complaint of fatigue and dyspnea on minimal exertion, symptoms that began in July 2024, at the time with a two-month history. He had a family history and positive genetic test for acne. His past medical history included systemic arterial hypertension, pre-diabetes, and obesity, with medication use of hydrochlorothiazide, spironolactone, amlodipine, and losartan.

On physical examination, the patient was in good general condition, acyanotic, anicteric, afebrile, hydrated, normochromic, with a systemic blood pressure of 140 x 95 mmHg; heart rate of 89 beats per minute and respiratory rate of 17 breaths per minute. Cardiac auscultation revealed a regular heart rhythm, in two beats, normal heart sounds, without murmurs. Pulmonary auscultation was physiological, with absence of crackles and wheezes. The electrocardiogram showed right bundle branch block and left anterior superior hemiblock (Figure 1).

Figure 1. Electrocardiogram: the black arrows indicate a widening of the QRS complex (greater than 120ms), with a typical pattern in leads V1 and V2 that may resemble "rabbit ears" (rsR' or rSR') representing right bundle branch block. The orange arrows indicated in leads DI and aVL show a positive QRS complex with a qR pattern (a small q wave followed by a tall R wave), and the blue arrows indicate a predominantly negative QRS complex with an rS pattern (a small r wave followed by a deep S wave), which demonstrates a left axis deviation (-30º and -90º), these being the most characteristic criteria of left anterior superior hemiblock.



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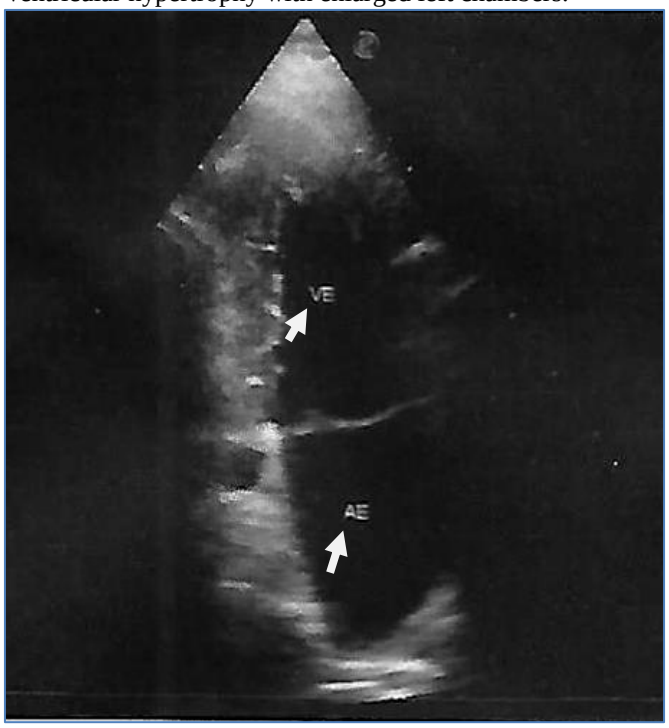
The echocardiogram, performed on January 21, 2025 (Figure 2), revealed concentric ventricular hypertrophy, left chambers with increased volume, preserved systolic function (ejection fraction of 66%), left ventricular diastolic function on inlet Doppler, and ventricular wall tissue Doppler shows an abnormal relaxation pattern.

Figure 2. Echocardiogram: the white arrows indicate the left ventricle (LV) and left atrium (LA), showing concentric ventricular hypertrophy with enlarged left chambers.



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Tissue Doppler ultrasound showed the following results: septal e' (*early diastolic velocity of the myocardium*): 8.5 cm/s. E/e' ratio (*ratio between the diastolic velocity E of mitral flow and the velocity e' of the mitral annulus*): 7.5 (*normal value < 8, compatible with normal left atrial pressure; above 15 – compatible with LAE – left atrial pressure*). Mild tricuspid and mild mitral regurgitation.

The TTR gene sequencing test for Hereditary Transthyretin-Linked Amyloidosis showed susceptibility to amyloidosis associated with the TTR gene (OMIM#105210). Currently, the patient is being monitored at the health unit, with a stable clinical picture, and has been referred to the cardiology service for joint follow-up.

Discussion

Hereditary transthyretin amyloidosis (ATTRv) is an underdiagnosed but clinically relevant condition, especially in patients with insidious cardiac manifestations and a positive family history. This case illustrates a typical presentation of the disease, but with slow progression and suggestive echocardiographic findings in a middle-aged patient with hypertension and multiple cardiovascular risk factors, which may mask or delay diagnosis[7,8]. Among the echocardiographic findings most frequently reported in the literature are concentric ventricular hypertrophy, valvular thickening, right ventricular wall thickening, biatrial dilation, presence of discrete pericardial effusion, progressive

diastolic dysfunction, and apical sparing—the latter considered highly suggestive of amyloid infiltration[7,8,10,12,13]. See Chart 1 for other case reports in the literature.

Chart 1. Case reports found in the literature on Cardiac Amyloidosis.

Author and year	Number of patients	Symptoms	Treatment	Outcome
Dagan *et al*., 2021	01	Asymptomatic from a cardiological standpoint. Progressive peripheral neuropathy with paresthesia in both feet.	Notreported.	Cobalt cardiomyopathy is a rare complication of failed hip replacements. If not identified early, it has a high mortality rate, but is easily reversible with timely treatment.
Panagiotopoulos *et al*., 2023	01	Dyspnea on exertion (NYHA class III)	The patient was initially treated with tafamidis, furosemide 20 mg, eplerenone 25 mg, ramipril 2.5 mg, bisoprolol 1.25 mg, vitamins B1 and B12, and duloxetine 60 mg, and was regularly monitored. Several months later, inotersen was added to his medication due to the progression of peripheral neuropathy, primarily affecting the lower limbs and his ability to walk. Due to side effects and a deterioration in the patient's overall condition, tafamidis was discontinued eight months later.	Currently, the peripheral neuropathy has worsened (Grade II) and an assistive device is required for the patient to ambulate. The latest echocardiographic evaluation showed mild thickening of the mitral membranes with mild mitral valve regurgitation and thickening of the interventricular septum, along with a scintillating appearance.
Oliveira *et al*., 2020	01	Nausea, anorexia, weight loss (3.6 kg in 3 months), asthenia, and dyspnea on minimal exertion.	Not performed.	The patient died 6 months after the onset of symptoms.
Schwarz *et al*., 2022	01	Progressive dyspnea (NYHA grade III–IV) and intermittent chest complaints, weight gain, and edema in the lower extremities.	Diuretic medication and therapy for heart failure (ACE inhibitors, beta-blockers, eplerenone).	After therapy was initiated, the patient's condition improved significantly, including the pleural effusion. A specific neurological examination was also performed to rule out a possible polyneuropathy (a genetic study).
Morgado *et al*., 2021	01	Retrosternal pain on exertion.	Antiplatelet therapy, statins, calcium channel blockers, and transdermal nitrates.	Three years later, he developed complete atrioventricular block and was admitted to the emergency room with sudden hemodynamic collapse. A dual-chamber pacemaker was implanted, and he was discharged asymptomatic. Shortly afterward, the patient developed progressive symmetrical tetraparesis, associated with marked muscle atrophy, numbness in the hands, orthostatic hypotension, and dysphagia.
Lima *et al*., 2023	01	Fatigue, orthopnea, and progressive bilateral edema in the legs.	Treatment with Tafamidis 61 mg	The response to treatment was quite unsatisfactory, with rapid worsening of heart failure, culminating in death 6 months after the initial diagnosis.

Hyer *et al*., 2021	01	Recurrent angina on exertion associated with mild shortness of breath.	Treatment includes aspirin 81 mg once daily, metoprolol succinate 50 mg once daily, lisinopril 10 mg once daily, atorvastatin 80 mg once daily, ezetimibe 5 mg once daily, and apixaban 5 mg twice daily.	Referred to the heart failure clinic for medical optimization of fluid status and modification of risk factors for heart failure.
Lyng *et al*., 2023	01	Dyspnea on exertion, stage II (NYHA), peripheral polyneuropathy, weight loss of 10 kg, and mild peripheral edema.	Treatment with Tafamidis 61 mg	Outpatient follow-up after 6 months, transthoracic echocardiography showed hemodynamic improvement after biventricular CRT, with an estimated ejection fraction >55%. The clinical improvement was substantial and the patient resumed physical activities at NYHA stage I. The patient received genetic counseling and family members are undergoing genetic screening.
Oye *et al*., 2021	02	Patient 1: Worsening dyspnea, fever, hypotension, and tachypnea. Patient 2: Presents with worsening dyspnea, edema in the lower extremities, hypotension, and tachypnea. *Note: Both patients are presenting with cardiogenic shock.	Patient 1: treated with intravenous diuretics and supportive care. Patient 2: treated with supportive care and IV diuretics.	Patient 1: Despite treatment, he became progressively more obtunded. After a week of hospitalization, the patient was transferred to the cardiac intensive care unit. He required intubation for acute hypoxemic respiratory failure and inotropic support with dobutamine. Despite the use of vasoactive medications, he remained hemodynamically unstable. He eventually went into cardiac arrest due to pulseless electrical activity and was not resuscitated. Patient 2: Complicated hospital course by atrial fibrillation with rapid ventricular response. The hypotension worsened, and the patient required three vasopressors at maximum doses. Considering the poor prognosis, the palliative care team was consulted for further care guidance, being eventually transferred to palliative care for comfort care; passed away a week later.
Yuan *et al*., 2023	01	Recurrent vomiting and sudden syncope for several months.	Metoprolol 25 mg twice daily, spironolactone 20 mg tablets once daily, and trimetazidine 20 mg three times daily.	Symptomatic improvement after starting medication.
Matamala *et al*., 2022	01	Numbness and distal paresthesia, associated with neuropathic pain in both lower limbs, without autonomic impairment or loss of strength.	Not reported.	Sequencing of the TTR gene confirmed the pathogenic Val50Met mutation, consistent with polyneuropathic ATTRv.
Awaya *et al*., 2024	01	Symptoms of exacerbated heart failure.	Therapy with small interfering RNA (siRNA) molecules. Two years later, siRNA therapy was switched from LNP-encapsulated siRNA (patisiran, first generation) to GalNAc-conjugated siRNA (vutrisiran, second generation).	There were no hospitalizations related to heart failure following siRNA therapy.

Kitakata *et al*., 2022	02	Patient 1: Progressive dyspnea on exertion and peripheral edema, dizziness and vertigo, and peripheral neuropathy with numbness in the hands. Patient 2: Progressive edema and numbness of both lower extremities with gait disturbances.	Patient 1: patisiran 0.3 mg/kg every three weeks. Patient 2: started treatment with patisiran 0.3 mg/kg every three weeks with diuretics.	Patient 1: After one year, their subjective symptoms of peripheral neuropathy showed some improvement in lower limb numbness, but heart failure symptoms did not show significant improvement. Patient 2: The symptoms remained unchanged for almost six months after administration, but their dyspnea and systemic edema gradually decreased. Eleven months after starting patisiran, the heart failure signs worsened to NYHA class IV with exacerbation. Treatment with patisiran was discontinued and replaced with oral tafamidis meglumine 80 mg daily.

NYHA: New York Heart Association; **ACE inhibitor:** Angiotensin-Converting Enzyme Inhibitors; **HF:** Heart Failure; **TTR gene:** Transthyretin gene; **ATTRv:** Hereditary Transthyretin Amyloidosis; **siRNA:** Small RNA Interfering Molecule Therapy; **LNP:** Lipid Nanoparticles; **GalNAc:** N-acetylgalactosamine.
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The identification of fatigue and dyspnea on minimal exertion as initial complaints in a patient with a family history and a positive genetic test for a mutation in the TTR gene (OMIM#105210) reinforces the importance of a targeted history and early genetic evaluation in suggestive family contexts. In addition to symptoms of cardiovascular origin, the literature highlights frequent extracardiac manifestations in ATTRv, such as bilateral carpal tunnel syndrome, lumbosciatica, sensorimotor peripheral neuropathy, autonomic dysfunctions (including postural hypotension and gastrointestinal disorders), as well as lumbar spinal stenosis and a history of early orthopedic surgery in major joints, especially the hip and shoulder[7,8,12]. The presence of these signs, especially when associated with each other or preceding cardiac manifestations, should increase clinical suspicion and motivate specific complementary investigation.

Although the ECG showed right bundle branch block and left anterior superior hemiblock, the classic low-voltage pattern frequently associated with cardiac amyloidosis was not observed, reinforcing that this sign is not present in all cases and its absence does not exclude the diagnosis[7,10]. The discrepancy between ventricular thickness on echocardiography and electrical voltage on ECG is one of the classic clues to the disease, but it is not always evident, especially in patients with comorbidities such as systemic arterial hypertension. In these cases, a pattern of true concentric myocardial hypertrophy is frequently observed, with partial preservation of voltage on the ECG, in addition to superimposed structural changes resulting from chronic cardiac remodeling induced by pressure overload. This overlapping phenotype can make it difficult to differentiate between hypertrophy due to hemodynamic overload and the infiltrative thickening characteristic of ATTRv, requiring greater accuracy in the correlation between clinical findings and imaging methods[8,11].

From a clinical and imaging standpoint, transthyretin deposition cardiomyopathy (TTRv) can share characteristics with several structural myocardial conditions, making the recognition of the main differential diagnoses essential. Among the diseases that most frequently mimic TTRv are hypertensive cardiomyopathy, hypertrophic cardiomyopathy of sarcomeric origin, infiltrative diseases such as Fabry disease and light chain amyloidosis (LA), as well as aortic stenosis, particularly in elderly patients[7,9,10]. In cases of ventricular hypertrophy, the absence of significant left ventricular dilation, associated with early diastolic dysfunction and diffuse myocardial thickening, may indicate an infiltrative pattern.Additionally, findings such as valvular thickening, disproportionate septal hypertrophy, interatrial septal involvement, and conduction abnormalities such as bundle branch blocks and bradycardia reinforce the diagnostic suspicion of ATTRv, differing from the patterns observed in non-infiltrative cardiomyopathies[8,10,12]. In this context, cardiac magnetic resonance imaging, through the detection of transmural late enhancement and elevated extracellular volume, associated with echocardiography showing a specific pattern of preserved apical longitudinal strain, plays a relevant role in distinguishing between possible etiologies[10,13].

The presence of normal left atrial filling pressure (E/e' = 7.5) and mild valvular insufficiency corroborates the early stage of the disease, which is favorable for early therapeutic management and specialized follow-up[8,13]. The clinical picture, combined with the echocardiogram showing concentric ventricular hypertrophy and altered diastolic function, raises a high suspicion of amyloid infiltration, despite the preserved ejection fraction, which is common in the early stages of amyloid cardiomyopathy[7,10].

The timely recognition and referral to the cardiology service, as performed in this case, are fundamental for risk stratification, diagnostic confirmation, and early initiation of disease-modifying therapies. Among the currently available therapeutic options, tafamidis stands out, a transthyretin stabilizer that acts by preventing the dissociation of the TTR tetramer into unstable monomers, the initial process in the formation of amyloid fibrils. Clinical evidence demonstrates that the use of tafamidis 20 mg or 80 mg orally, once daily, is associated with a reduction in cardiovascular mortality and hospitalization rates for heart failure in patients with ATTRv, especially when initiated in early stages of the disease[9,12].

The adverse effects of tafamidis are generally mild, with the most frequently reported being abdominal pain, urinary tract infections, and nonspecific gastrointestinal symptoms. The drug's good tolerability and favorable safety profile make it a first-line choice for the treatment of symptomatic ATTRv cardiomyopathy. In addition, studies demonstrate that the earlier the start of therapy, the better the clinical outcomes, with improved functional capacity, stabilization of quality of life, and delayed progression of cardiac dysfunction[9,12].

Other therapeutic approaches have been investigated and, in some cases, used in a complementary or alternative manner. These include inhibitors of hepatic transthyretin synthesis, such as patisiran (an interfering RNA) and inotersen (an antisense oligonucleotide), which are more commonly used in cases with neurological involvement, but with promising cardiac effects described in recent studies. Investigational therapies, such as next-generation gene silencing agents and amyloid fibril scavengers, represent emerging strategies with the potential to substantially alter the course of the disease. The therapeutic choice should consider the clinical stage, the predominant phenotypic form (cardiac or mixed), the genetic profile, and the patient's comorbidities, with specialized multidisciplinary follow-up[9,12].

This case reinforces the importance of genetic screening in patients with a positive family history, even when symptoms are nonspecific, and highlights the usefulness of tissue Doppler echocardiography as a sensitive tool for early detection of diastolic dysfunction associated with amyloid cardiomyopathy[10,13].

Conclusion

ATTRv is a condition that is still underdiagnosed, but of great clinical relevance, and early identification is essential for effective treatment and improved prognosis. The case presented highlights the importance of a detailed clinical investigation, including a targeted anamnesis and gathering of family history, in addition to the use of complementary tests, such as echocardiography with tissue Doppler and genetic analysis of the TTR gene.

The absence of classic signs, such as low voltage on the electrocardiogram, demonstrates the variability of the clinical manifestations of ATTRv and reinforces the need for a comprehensive diagnostic approach. Early diagnosis allows for the timely introduction of therapies that can modify the course of the disease, delay the progression of cardiomyopathy, and improve the quality of life of patients.

Authors' contributions

Research conception and design: Isabella Ribeiro Azzi; Emily Duarte Macedo. Analysis, interpretation of the data, and manuscript writing: Isabella Ribeiro Azzi; Emily Duarte Macedo; José da Silva de Magalhães. Critical review of the manuscript regarding intellectual content and final presentation: Marcelo José da Silva de Magalhães. The authors approved the final version of the manuscript and declared themselves responsible for all aspects of the work, including ensuring its accuracy and integrity.

Conflict of interests

None.

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