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(CASE REPORT)

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Subclinical hypothyroidism and dilated cardiomyopathy (DCM) in children

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Abstract

Introduction: Subclinical hypothyroidism is defined by the combination of an elevated thyroid-stimulating hormone (TSH) level and a normal free thyroxine (T4L) level.

Dilated cardiomyopathy is characterized by both dilation of the ventricles and hypokinesia of these ventricles. It predominantly involves the left ventricle, but the right ventricle is frequently involved.We present two cases of idiopathic dilated cardiomyopathy associated with a subclinical picture of hypothyroidism.

Case report 1: An 11-year-old female patient was admitted to cardiology for congestive heart failure on CMD; she presented with New York Heart Association (NYHA) stage IV dyspnea and generalized edema. Tran's thoracic echocardiography showed left ventricular (LV) dilatation with global hypokinesia, LV ejection fraction at 15%. The biological workup showed a subclinical hypothyroidism profile with TSH at 11.2 mIU/l, T4: 21.2 pmol/l, T3: 4.2pmol/l.

Case report 2: A 9-year-old patient, without any notable history, was referred to the cardiology department for global cardiac decompensation with NYHA stage IV dyspnea and lower limb edema despite optimal medical treatment. Tran's thoracic echocardiography showed dilated cardiomyopathy with severe impairment of LV systolic function (10%). The biological workup showed a subclinical hypothyroidism profile with TSH: 7.7 mIU/l, T4: 19.2 pmol/l.

Conclusion: Dilated cardiomyopathies (DCM) represent the most common cardiomyopathies in children either of constitutional or acquired origin. Subclinical hypothyroidism is frequently observed in patients with DCM and increases the risk of mortality.

Keywords: Subclinical hypothyroidism; Dilated cardiomyopathy; Heart failure

1. Introduction

Subclinical hypothyroidism is defined by the combination of an elevated thyroid stimulating hormone (TSH) level and a normal free thyroxine (T4L) level.

Dilated cardiomyopathy is characterized by both dilation of the ventricles and hypokinesia of these ventricles. It predominantly involves the left ventricle, but the right ventricle is frequently involved. The positive diagnosis presupposes the elimination of other causes of dilated and hypokinetic heart, such as myocardial infarction or heart valve disease. Many of the cardiovascular alterations result from genomic and non-genomic actions of thyroid hormone on the heart and blood vessels.

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1.1. Case report 1

An 11-year-old female patient was hospitalized in cardiology for congestive heart failure. She presented with New York Heart Association (NYHA) stage IV dyspnea and generalized edema. On physical examination, the blood pressure was 105/59mm Hg, thyroid was not palpable, hepatomegaly, impetiginous detachment opposite both knees, chest auscultation found crackling rales in both lung fields. The electrocardiogram (ECG) showed sinus tachycardia. The biological workup showed a subclinical hypothyroidism picture with a TSH of 11.2 mIU/l, T4: 21.2 pmol/l, T3: 4.2pmol/l, a natraemia of 139 mmol/L, a kalemia of 4.3 mmol/L, a corrected calcemia of 89.6 mg/L, a Vitamin D deficiency of 13.7ng/ml, a correct lipid balance and anti TPO antibodies: negative less than 40 IU/ml.

Cervical ultrasound: the 2 thyroid lobes are of normal size, regular contours and homogeneous echostructure.

Tran's thoracic echocardiography showed dilatation of the left ventricle (LV), global hypokinesia with an ejection fraction at 15%.

We retained the diagnosis of CMD associated with a subclinical hypothyroidism picture. The initial treatment was based on dobutamine, furosemide with administration of ACE inhibitor and beta-blockers three days later and substitution of vitamin D deficiency.The evolution after 3 months of treatment was favorable. Echocardiography showed a slight improvement in ejection fraction (25%).

1.2. Case report 2

A 9-year-old patient was admitted to the cardiology department for global cardiac decompensation. He presented with New York Heart Association (NYHA) stage III dyspnea with orthopnea. On physical examination, his blood pressure was 83/42 mm Hg, his thyroid was not palpable, he had a large amount of ascites, multiple ulcerations on his knees and ankles, and his chest auscultation revealed crackling rales in both lung fields. The biological work-up showed a picture of frust hypothyroidism with a TSH of 7.7 mIU/l, T4: 19.2 pmol/l, a natraemia of 124mmol/L, a kalaemia of 4mmol/L, a corrected calcaemia of 94mg/L, a Vit D deficiency: 9ng/ml, a correct lipid balance and anti TPO antibodies: negative less than 40 IU/ml.

Cervical echography: no abnormalities. Transthoracic echocardiography showed dilatation of the left ventricle (LV), global hypokinesia with a LV ejection fraction of 10%.

We retained the diagnosis of CMD associated with a subclinical hypothyroidism picture. The initial treatment was based on dobutamine, furosemide with administration of ACE inhibitor and beta-blockers three days later and substitution of vitamin D deficiency. The evolution after 3 months of treatment was unfavorable.

2. Discussion

We presented two cases of dilated cardiomyopathy associated with a picture of subclinical hypothyroidism. The thyroid workup was performed as part of the etiologic workup.

Thyroid hormones exert positive chronotropic, dromotropic, inotropic and lusitropic effects on cardiac function. This results in an acceleration of the cardiac rhythm, an improvement of the conductivity, contractility and diastolic relaxation of the myocardium. In addition, they decrease peripheral vascular resistance [1].

These interactions can be direct on the cardiomyocyte by binding the hormones to their specific nuclear receptor or indirect, either through a circulatory effect (effect on total blood volume and peripheral vascular resistance) or through cardiac sympathetic innervation [2-3].

Numerous observational studies have shown a potential association of subclinical hypothyroidism and dilated cardiomyopathy.

Karolina Zawadzka published a review in 2021, Radosław Dziedzic [4] showed that not only the local bioavailability of myocardial thyroid hormones could be decreased due to altered expression of desiodase, but practically all genes involved in thyroid hormone biosynthesis are expressed in the myocardium.

In the healthy heart, deiodase 2 activates T4 to the bioactive hormone T3. Expression of deiodase 3 is undetectable in the healthy heart, except during embryonic development.

In a transgenic mouse model of CMD described by Wassner et al [6], abnormal activation of deiodase 3 leads to subclinical hypothyroidism by inactivating T3 in T2.

Gil-Cayuela et al [5] showed the role of deiodase in human cardiomyocytes with alteration of genes involved in thyroid hormone biosynthesis in patients with DCM.

A Cohort Study published in 2015 by Wenyao Wang, Haixia Guan [7] which included 458 patients with idiopathic dilated cardiomyopathy, whose objective was the evaluation of impact of dysthyroidism on the survival rate of these patients.

The most common thyroid dysfunction was subclinical hypothyroidism, followed by subclinical hyperthyroidism, low T3 syndrome, and hypothyroidism.

During the follow-up of these patients, hypothyroidism was the predictor of mortality, followed by low T3 syndrome and subclinical hypothyroidism.

Fruhwald F.M and Ramschak-Schwarzer [8] showed that subclinical hypothyroidism is frequently observed in patients with DCM when they live in an area of chronic iodine deficiency. This may be explained by chronic salt restriction as a basic treatment for congestive heart failure.

3. Conclusion

Dilated cardiomyopathies (DCM) represent the most common cardiomyopathies in children either of constitutional or acquired origin. Subclinical hypothyroidism is frequently observed in patients with DCM and increases the risk of mortality.

Compliance with ethical standards

Acknowledgments

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Disclosure of conflict of interest

The authors declare no conflict of interests.

Statement of ethical approval

The present research work does not contain any studies performed on animals/humans subjects by any of the authors.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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