East African Scholars Journal of Medical Sciences
Abbreviated Key Title: East African Scholars J Med Sci
ISSN 2617-4421 (Print) | ISSN 2617-7188 (Online) |
Published By East African Scholars Publisher, Kenya

OPEN ACCESS

DOI: 10.36349/easms.2019.v02i01.006 Volume-2 | Issue-1 | January-2019 |

Case Reports

Reversible Dilated Cardiomyopathy Complicating Iatrogenic Chronic Hypocalcaemia: About 3 Cases

Fatima Zahra Zaher[1], Loubna Oukit [2], Naima Bouznad [3], Sana Rafi [4], Ghizlane El Mghari [5], Mustapha El Hattaoui [6], Nawal El Ansari [7]

[1,2,3,4,5,7]Department of Endocrinology, Diabetes, Metabolic diseases and Nutrition, Mohammed VI university hospital, Marrakech
[6]Department of cardiology, Mohammed VI university hospital, Marrakech

*Corresponding Author
Fatima Zahra ZAHER

Abstract: Dilated cardiomyopathy (DCMP) is a serious and rare complication of unknown chronic hypocalcaemia. We report three cases of asymptomatic chronic hypoparathyroidism post thyroidectomy complicated of DCMP with heart failure. The first case is 56-year-old patient, asymptomatic for 14 years, admitted for generalized seizures of sudden onset revealing hypoparathyroidism, on examination we found diastolic murmur at mitral focus with signs of hypocalcaemia confirmed by biology: Serum calcium at 40 mg/L, serum phosphate at 98mg/L, her brain scan showed a Fahr syndrome and her transthoracic echocardiography (TTE) showed a DCMP, global hypokinesia and an ejection fraction at 39%. The second is 29-year-old patient admitted for insidious hypoparathyroidism dating back to 8 years, On blood examination she had hypocalcaemia at 32 mg/l with PTH level at 10.5 pg/ml, her TTE showed DCMP, global hypokinesia and an ejection fraction at 28%. The third case is a 62-year-old patient who had thyroidectomy 5 years ago, admitted for etiological assessment of DCMP revealed by dyspnea, his blood examination showed hypocalcemia at 50mg/l, phosphoremia at 109 mg/l, his TTE objectified DCMP with ejection fraction at 20%. DCMP is a very serious complication of hypoparathyroidism after cervical surgery. Hence, the importance of the systematic screening based on calcium levels after any thyroid surgery and adequate supplementation.

Keywords: reversible cardiomyopathy – chronic hypocalcemia – hypoparathyroidism

INTRODUCTION

Hypoparathyroidism, defined as the decrease or absence of parathyroid hormone resulting in hypocalcaemia and hyperphosphoremia, represents a frequent affection mainly caused by cervical surgery. Chronic hypocalcaemia induced by iatrogenic hypoparathyroidism is a rare cause of dilated cardiomyopathy, the particularity of this entity of DCMP is its reversibility at different degrees after the correction of hypocalcaemia, and which depends on the precocity and effectiveness of its management. The incidence of this complication is very rare in clinical practice because of the early and adequate management of any hypocalcaemia. Calcium plays an important role in cardiac contractility, during hypocalcaemia the sarcoplasmic reticulum is unable to maintain sufficient calcium to initiate myocardial contraction (Vlot M *et al*, 2014). DCMP complicating hypocalcaemia has been described especially in children; in adults the number of patients is limited. We report three cases of DCMP complicating chronic iatrogenic hypoparathyroidism; the particuliarity is the rarety of this condition and the insidious caracter of hypocalcemia for several years.

Patients and Observations

Case 1:

A 56-year-old patient having a history of thyroidectomy for toxic multinodular goiter 14 years ago put under levothyroxine 100 micrograms per day, followed for bilateral cataract for 4 years admitted for generalized tonic-clonic seizures in a context of apyrexia.The interrogation found no paresthesia or convulsive seizure immediately postoperatively. On examination we found a conscious patient, positive Chvostek and Trousseau signs and a diastolic murmur of 4/6 at mitral, the neurological examination is without particularity. An emergency brain scan showed calcification of the basal ganglia suggestive of Fahr syndrome. The electrocardiogram (ECG) recorded a regular, sinus rhythm with no repolarization disorder or

Quick Response Code

Journal homepage:
http://www.easpublisher.com/easjms/

Article History
Received: 12.01.2019
Accepted: 22.01.2019
Published: 27.01.2019

prolongation of the QT space. The chest x-ray showed cardiomegaly with bilateral bronchial syndrome. Transthoracic echocardiography (TTE) showed extensive dilated cardiomyopathy in left cavities associated with global hypokinesia with systolic dysfunction, ejection fraction was 39% and portal hypertension was moderate. On blood examination, she had hypocalcemia at 40mg/l, hyperphosphoremia at 98mg/l, serum albumin at 37g/l, sodium concentration at 130mmol/l and serum potassium level of 3.7 mmol/l, hypothyroidism with thyroid stimulating hormone (TSH) at 7.98 µIU/ml. The patient was put on diuretic, calcium inhibitor, converting enzyme inhibitor, beta-blocker calcium supplementation and 1-alpha-hydroxy vitamin D. The evolution was marked by the improvement of the cardiac function with a systolic ejection fraction passed to 60%.

Case 2:

A 29-year-old patient with a history of total thyroidectomy 8 years ago, receiving levothyroxine 75 µg daily, and a bilateral cataract operated one year ago, was admitted for tetanus attacks. The interrogation found the notion of tingling, muscle cramps and paraesthesia occurring in a paroxysmal manner triggered by physical effort for the past 5 years, with no notion of immediate postoperative tetanus seizures. On examination she had tetany crisis with diffuse muscular cramps, a positive Chvostek sign, her blood pressure was 90/60 mmHg, thoracic auscultation found a systolic murmur of 3/6 on the apex and rattles crackling in the two lung fields. Chest X-ray showed cardiomegaly with bilateral interstitial edema. ECG showed sinus tachycardia, negative T waves in anterolateral leads, and an extended QT interval. The biological assessment showed corrected serum calcium at 32 mg/l, albumin at 29 g/l, sodium at 140mmol/l and potassium at 3.5 mmol/l. TSH was at 7mIU/l and the intact parathyroid hormone (PTH) was low at 10.5 µg/ml. TTE showed dilated cardiomyopathy, global hypokinesia, and systolic dysfunction with an ejection fraction at 28%. A cervical ultrasound found an empty thyroid and parathyroid lodge without cervical lymphadenopathy. The patient was treated by intravenous calcium until normalization of the calcemia with an oral relay and supplementation in active vitamin D (Calcitriol) with 2 µg per day, and a load in magnesium. For her heart failure, she was put on ACE inhibitor in combination with a diuretic. The evolution was marked by the improvement of the clinical parameters, namely the signs of neuromuscular hyper excitability (cramps, paresthesia) with an improvement of the cardiac function and a systolic fraction ejection increased to 42%.

Case 3:

A 62-year-old patient with a history of thyroidectomy 5 years ago receiving 100ug per day of levothyroxin, admitted for dyspnea, on interrogation we found notion of tingling and tetany crisis. On examination, his blood pressure was 130/70 mmHg, negative Chvostek and Trousseau sings. His ECG showed regular sinus rhythm, complete left branch block, and an extended QT interval. His TTE showed dilated cardiomyopathy, global hypokinesia, and systolic dysfunction with an ejection fraction at 20%. His blood examination showed hypocalcemia at 50mg/l, hyperphosphoremia at 109 mg/l, serum sodium at 140mmol/l, potassium at 3.9 mmol/l and TSH was at 17mUI/l. The patient was treated by intravenous calcium until normalization of the calcemia with an oral relay and supplementation with active vitamin D. For his heart failure, he was put on ACE inhibitor in combination with a diuretic.

DISCUSSION

Postoperative hypocalcaemia is a serious complication after total thyroidectomy which may cause severe symptoms. The majority of postoperative hypocalcaemia is transient with spontaneous recovery. However, hypocalcaemia is considered permanent when it does not return to normal within 6 months (1.3-3% of cases) especially when it is caused by irreversible damage or devascularisation to the parathyroid glands (Abboud B *et al*, 2002). This permanent hypocalcaemia causes short and long-term complications that can be life-threatening and requires lifelong replacement therapy.

The clinical manifestations of hypocalcaemia are related to the intensity of the disorder, the age of onset and especially its speed of installation. It can be expressed clinically by variable signs, especially neuromuscular and sensory ones, ranging from simple tingling of extremities which was found in two of our three cases, to a real tetanus attack; it can also remain asymptomatic and of biological expression (Bousaadani Soubai R *et al*, 2012).

However, hypocalcaemia can sometimes lead to cardiac complications, mostly in patients with profound hypocalcaemia with total calcium lower than 70 mg/l, such as hypotension, prolongation of the QT interval, arrhythmias, myocardial dysfunction, and sometimes even heart failure (Sung JK *et al*, 2010).

Cardiac contractility is mainly influenced by calcium levels in extracellular fluid to initiate excitation and contraction of cardiac muscle fibers (Vlot M *et al*, 2014). In fact, calcium, which is a central mediator of electrical activation and ion exchange, plays a key role in excitation-contraction coupling; extracellular calcium ions flux into the myocytes, which induces the release of calcium from sarcoplasmic reticulum and initiates myocardial contraction when it binds to the troponin C-tropomyosin complex allowing attachment between the actin and myosin filaments (Benzarouel D *et al*, 2014).

Even if the physiological role of calcium on contraction is well-known, the pathophysiology of

hypocalcemic DCMP is still unclear. More recent evidence suggests that vitamin D and PTH may also have an independent role (Bansal B *et al*, 2014).

Vitamin D deficiency is a common and important cause of DCMP especially in pediatric population (Yazici MU *et al*, 2018, Tamirat M *et al*, 2017) which is due to the autocrine function of vitamin D in cardiomyocytes. This role has been shown experimentally in rats where Vitamin D deficiency causes cardiac hypertrophy which in not prevented by normalization the calcium level (Tishkoff DX *et al*, 2008), also, vitamin D deficiency in mothers has been shown to be responsible of abnormal maturation of cardiomyocytes in the offspring despite a normal calcium level (Gezmish O *et al*, 2010).

Also isolated hypoparathyroidism had been associated with reversible left ventricular dysfunction in several studies (Jung YJ *et al*, 2013, Rhee HS *et al*, 2013) which is due to its role in the maintenance of a normal cardiac function by stimulating protein kinase C which simulates intracellular protein synthesis.

The peculiarity of hypocalcemic heart failure is the often complete recovery of myocardial function after correction of hypocalcaemia; patients with hypocalcaemia recover in 3 to 12 months after calcium, vitamin D replacement therapy and the treatment of heart failure as reported in our cases. Improvements in cardiac morphology and functions had been reported to be observed precociously a week after the initiation of treatment in a 6-month-old male patient with dilated cardiomyopathy secondary hypocalcaemia resulting from vitamin D deficiency (Yilmaz O *et al*, 2014).

CONCLUSION

Hypocalcemic dilated cardiomyopathy is a rare but serious complication which highlights the importance of the systematically monitoring of the phosphocalcic balance and the prescription of a vitaminocalcic substitution if necessary. Its management is based on active vitamin D and calcium substitution with close cardiological monitoring and its evolution is marked by the more or less complete regression of heart failure.

REFERENCES

1. Abboud, B., Sargi, Z., Akkam, M., & Sleilaty, F. (2002). Risk factors for postthyroidectomy hypocalcemia. *Journal of the American College of Surgeons*, *195*(4), 456-461.
2. Bansal, B., Bansal, M., Bajpai, P., & Garewal, H. K. (2014). Hypocalcemic cardiomyopathy—different mechanisms in adult and pediatric cases. *The Journal of Clinical Endocrinology & Metabolism*, *99*(8), 2627-2632.
3. Benzarouel, D. Hasni, K. Ashab, H. & El, H. M., (2014). A reversible cause of cardiomyopathy: Hypocalcemia Annales de Cardiologie et d'Angéiologie 63(2), 102-106.
4. Boussaadani, S.R., Abourazzak, F. & Harzy, T. (2012). Hypocalcemia : Practical review. Rev Mar Rhum. 2(1), 4-9.
5. Gezmish, O., Tare, M., Parkington, H. C., Morley, R., Porrello, E. R., Bubb, K. J., & Black, M. J. (2010). Maternal vitamin D deficiency leads to cardiac hypertrophy in rat offspring. *Reproductive Sciences*, *17*(2), 168-176.
6. Jung, Y. J., Kim, S. E., Hong, J. Y., Lee, J. H., Park, D. G., Han, K. R., & Oh, D. J. (2013). Reversible dilated cardiomyopathy caused by idiopathic hypoparathyroidism. *The Korean journal of internal medicine*, *28*(5), 605.
7. Lee, S. W., Jung, Y. K., Jeon, U., Park, S. H., Lee, S. J., Sin, W. Y., & Jin, D. K. (2013). Takotsubo cardiomyopathy associated with severe hypocalcemia secondary to idiopathic hypoparathyroidism. *Korean circulation journal*, *43*(8), 573-577.
8. Sung, J. K., Kim, J. Y., Ryu, D. W., Lee, J. W., Youn, Y. J., Yoo, B. S., & Choe, K. H. (2010). A case of hypocalcemia-induced dilated cardiomyopathy. *Journal of cardiovascular ultrasound*, *18*(1), 25-27.
9. Moges, T., Shiferaw, Y., & Heye, T. (2017). Maternal vitamin D deficiency: a culprit for hypocalcaemia induced myocardial failure in a four-month old infant: a case report from Tikur Anbessa Specialized Hospital, Ethiopia. *Ethiopian journal of health sciences*, *27*(3), 299-304.
10. Tishkoff, D. X., Nibbelink, K. A., Holmberg, K. H., Dandu, L., & Simpson, R. U. (2007). Functional vitamin D receptor (VDR) in the t-tubules of cardiac myocytes: VDR knockout cardiomyocyte contractility. *Endocrinology*, *149*(2), 558-564.
11. Vlot, M., de Jong, M., de Ronde, P., & Tukkie, R. (2014). A surprising cause of reversible dilated cardiomyopathy. *BMJ case reports*, *2014*, bcr2013203512.
12. Yazici, M. U., Kesici, S., Demirbilek, H., Tanyıldız, M., Gumustas, M., & Bayrakci, B. (2018). Reversible Dilated Cardiomyopathy Due to Combination of Vitamin D–Deficient Rickets and Primary Hypomagnesemia in an 11-Month-Old Infant. *Journal of Pediatric Intensive Care*, *7*(01), 046-048.
13. Yilmaz, O., Kilic, O., Ciftel, M., & Hakan, N. (2014). Rapid response to treatment of heart failure resulting from hypocalcemic cardiomyopathy. *Pediatric emergency care*, *30*(11), 822-823.