Graves' disease combined with acute myocarditis and thyrotoxic periodic paralysis in a male:a case report and review of the literature

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Abstract

Graves' disease is the most common reason for hyperthyroidism which manifest as multi-system changes.Among these clinical manifestations acute myocarditis and thyrotoxic periodic paralysis are very rare and patients who are combined with both two haven't been reported yet.The etiology may attribute to autoimmunity.

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Abstract:

Graves' disease is the most common reason for hyperthyroidism which manifest as multi-systemchanges.Among these clinical manifestations acute myocarditis and thyrotoxic periodic paralysis are very rare and patients who are combined with both two haven't been reported yet.The etiology may attribute to autoimmunity.Physicians should check thyroid function when facing patients with hypokalemia and cardiac symptoms.

We report a rare case of Graves' disease combined with acute myocarditis and thyrotoxic periodic paralysis.The patient was a 25-year-old Chinese young male who was healthy previously.He was suddenly paralyzed when he woke up in the morning,then he felt palpitations and dyspnea.He was then sent to our emergency department by ambulance.Uponarrival,the electrocardiograph showed junctionaltackycardia and STdepression in all leads,laboratory findings showed extreme hypokalemia and elevated troponinI,he was given potassium tablets and also intravenous potassium supplement,then metoprolol was given to control heart rate,after this coronary CTA was perfomed and there was no abnormal findings of the coronary artery,so acute myocarditis

was diagnosed.Afterwards,he was admitted to our ED ward. We continued giving him potassium supplement and metoprolol,further testing showed Graves' disease and continued elevated cardiac enzymes and BNP. We suggested the patient to accept cardiac MR(CMR) and endocardial myocardial biopsy(EMB),but the patient refused further examination.By consulting an endocrinologist,we then gave him methiamazole to treat hyperthyroidism,andtrimetazidine,calciumdibutyryladenosinecyclophosphate for myocarditis.After 6 days,the patient's cardiac enzymes,BNP,potassium and electrocardiograph returned to normal. His symptoms were relievedand he was discharged from the hospital. During a 6-mouth follow up the patient continued taking metoprolol, trimetazidine and methiamazole. He was asymptomatic and checked his thyroid,liver,kidney function routinely to adjust his medicine.His hyperthyroid state was controlled.

Acute myocarditis and thyrotoxic periodic paralysis are two rare manifestations of Graves' disease.In our case,we almost missed the diagnosis of Graves' disease,we first thought acute myocarditis was the primary cause of the patient's clinical manifestations.So physicians should think of Graves' disease when patients have unknown reason of hypokalemia and elevated cardiac enzymes.

Key words: Graves'diease,myocarditis,thyrotoxic periodic paralysis,junctional tachycardia.

Key clnical message:

Physicians should not ignore Graves' disease when facing patients unknown paralysis.Physicians should know acute myocarditis is a rare combination of Graves' disease which etiology may contribute to autoimmunity.

1 Introduction:

We report a case of acute myocarditis and thyrotoxic periodic paralysis in the setting of new-onset Graves' disease. A previously healthy 25-year-old male presented with sudden paralysis,palpitations,dyspnea,elevated troponin I and extremehypokalemia.After thorough examination he was diagnosed with Graves' disease combined with acute myocarditis and thyroxic periodic paralysis.

I

2 Case report

A 25-year-old Chinese young male was suddenly paralyzed when he woke in the morning, by the same time he felt palpitations,dyspnea,nausea and vomiting gastric contents one time.He was then sent to our emergency department by ambulance. Upon arrival, the electrocardiograph was performed,which showed junctional tachycardia(heart rate 91 times per minute,figure1) ,ST depression in all leads. The emergent laboratory results showed potassium 1.7mmol/L, troponin I 0.32ng/ml. Acute myocardial infarction or acute myocarditis and hypokalemic periodic paralysis was considered,theemergency department gave him potassium supplement orally and intravenously,oxygeninspiration,aspirin and clopidogrel. Metoprolol was given to control heart rate. Regarding the young age and no risk factors contribute to acute myocardial infarction, the ED department suggested the patient to perform an emergent coronary CTA and a brain CT to exclude more dangerous disease,the results showed no abnormal findings of the coronary artery,and no abnormal findings of brain CT. So acute myocarditis was diagnosed. Then the patient was admitted to our ED ward. We diagnosed him as acute myocarditis and hypokalemic periodic paralysis(reason unknown),the next step was to find out the primary disease. The patient's family history did not reveal anything significant to his present condition. He was healthy did not take any drugs previously. He also denied any weight changes recently. Physical examination was performed. The patient was conscious, afebrile and blood pressure was 110/65mmHg.He was agitated and was sweating profusely.The muscle strength was only grade 2. According to the patient's high metabolic condition, we thought of a disease called hyperthyroidism which was the most common reason for young males to suffer from hypokalemic periodic paralysis. So we specially checked the thyroid. There was no exophthalmos of the patient's eyes and no restriction of the eye movements. There was no hand tremor.Palpation of the thyroid showed II degree's swelling of the thyroid gland and no abnormal findings on isthmus. There was also no tenderness. On auscultation of the thyroid,a bruit could be heard. Then we examined lungs,heart and abdomen, all were normal. Thyroid function tests revealed a hyperthyroid state,Graves' disease was considered:T3 17.51pmol/L(3.1-6.8pmol/L),T4 39.68pmol/L(12-22pmol/L),TSH

0.005 μIU/mL(0.27-4.2μIU/mL),thyroglobulin 94.77ng/mL,TGAB 18.35IU/mL(normal), TSHR-AB 13.76 IU/l(0-1.5 IU/l),TPOAB 77.67 IU/mL(0-34 IU/mL).To confirm the diagnosis of Graves' disease and exclude subacute thyroiditis,weperfomed thyroid ultrasonography which showed enlarged thyroid gland and rich blood flow signal,no tumor was found.We then performedthyroid static imaging,which showed the swelling of bilateral lobes and increased function(figure2).These examinations confirmed the diagnosis of Graves'disease.Other significant laboratory findings showed elevated troponin I and elevated BNP(troponin I 0.24ng/mL,BNP 196.24pg/ml).However,the patient still suffered from dyspnea,an echocardiograph was performed and there was no abnormal findings of the cardiac structure. We also considered virus myocarditis firstly. So we also checked the 13 common virus from throat swabs and there were no positive results. We considered autoimmunity may be the etiology. We then suggested him to perform cardiac MR and endocardial myocardial biopsy, but he refused further examination. At the same time we consulted an endocrinologist, the endocrinologist suggested him to accept radioactive iodinetherapy,but the patient preferred taking drugs.Wegave him methiamazole20mg/d to treat hyperthyroidism according to the suggestions, trimetazidine 60mg/d,metoprolol 50mg/d, calcium dibutyryl adenosine cyclophosphate40mg/d(IV) for myocarditis.His symptoms relieved in 6days.Again we checked troponinI,BNP and electrocardiograph.All returned back to normal.The patient was discharged from the hospital and we told him continuing taking methiamazole ,trimetazidine and metoprolol.

We performed a 6-mouth follow up in the emergency clinic and by phone calls, the patient continued taking metoprolol, trimetazidine and thiamazole. He was asymptomatic except some symptoms of thyrotoxicosis and checked his thyroid,liver,kidney function routinely to adjust his medicine. 45 days later,all his symtoms disappeared and thyroid function improved:T3 12.26pmol/L(3.1-6.8pmol/L), T4 28.37pmol/L(12-22pmol/L),TSH 0.07μIU/mL(0.27-4.2μIU/mL).After 80 days,euthyroid state was restored and his liver,kidney functions were in good state. He also checked electrocardiography and troponin I routinely and all were normal.Methimazole was adjusted to 5mg/d,metoprolol 23.75mg/d formaintenance.

3 Discussion:

Graves' disease is manifested as a hyperthyroid state,butis also an autoimmune process.According to the patient's thyroid function tests,hyperthyroidism was diagnosed. Measurements of serum levels of TRAb and thyroid ultrasonography represent the most important diagnostic tests for Graves' disease. [1]According to the latest guidelines[1],this patient had high TSHR-AB and his thyroid static imaging further confirmed the diagnosis of Graves' disease.The treatment for Graves' disease includingradioactive iodine (RAI), antithyroid drugs (ATDs) and thyroidectomy[2].For this patient,thyroidectomy was not suitable for him.The patient had acute myocarditis and thyrotoxic periodic paralysis,radioactive iodine therapy was more suitable for him to control his hyperthyroid state rapidly,although we had persuaded him to accept this therapy,he and his family still chose drugs for treatment.So according to the guidelines and endocrinologist's suggestions,we gave him methiamazole and let him check thyroid function routinely. We told him to accept radioactive iodine therapy if methiamazole could not control his hyperthyroid state.

Thyrotoxicosis rarely can cause thyrotoxic periodic paralysis (acute muscle paralysis and

severe hypokalemia)[3]. It is usually reported in young males.Glass J had reported an African-American male teenager with Graves' disease and paralysis.[4] So as for this patient,hewas a young male who suffered from sudden paralysis and severe hypokalemia.Thesemanifestations were consistent with the previous case report.The treatment should be the correction of hypokalemia and hyperthyroidism[5],then the symtoms will relieve. This patient was able to move as usual when his potassium levels returned back to normal.Obese patients are at an especially increased risk for this manifestation due to underlying insulin resistance, which enhances basal sodium-potassium ATPase function.[6] This patient was obese and he admitted drinking high amounts of carbohydrate drinks before he was admitted. This confirmed the previous reports and researches.

Acute myocarditis is an acute injury of the myocardium which manifested as arrhythmia,dyspnea and elevated cardiac enzymes.It is usually caused by a viral infection,but few is caused by autoimmunity. Patients with acute autoimmune myocarditis always combined with an autoimmune state,such as systemic lupus

erythematosus,rheumatoid arthritis and so on[7]. As Graves' disease is also an autoimmune disease,it can manifest as acute autoimmune myocarditis in the theory.But acute autoimmune myocarditis is rarely seen in patients with Graves' disease.It was only reported one time that acute autoimmune myocarditis was a manifestation of Graves' diease.[8]This patient's electrocardiograph,elevated troponin I,normal coronary arteries and symptoms were consistent with acute myocarditis[7].According to the latest diagnostic citeria,cardiac MR(CMR) and EMB should be performed.Regretfully,this patient refused these procedure.Although CMR and EMB was not performed ,according to the latest guidelines[7],his symptoms and electrocardiograph was consistent with acute myocarditis and a normal coronary artery revealed no inschemia,he could be diagnosed as acute myocarditis. We deduced his myocarditis was attribute to autoimmunity.The treatment of primary disease is of vital importance.

In our case,the patient was combined with both two rare manifestations of Graves' disease which was more seldom seen in clinical practice.It has not yet been reported. So now we write it down to share with all the physicians worldwide.If the physicians receive a patient with hypokalemia and elevated cardiac enzymes,they should not forget to check the thyroid function.

Author contributions

Mengmei Li and Jun Teng contributed to the final version of this report,other authors contributed to the diagnosis and treatment for the patient in the report.

Conflict of interest:

None of the authors have conflict of interest to declare in this article.

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None.

Ethical approval:

We have told the patient about the publication of his disease.He agreed and signed the patient consent form.

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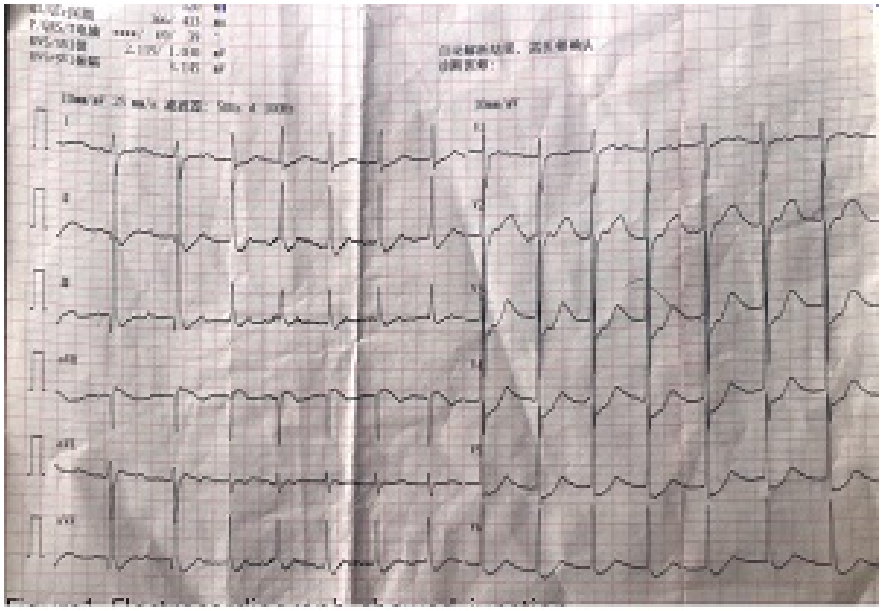
Figure1: Electrocardiograph showed junctional tachycardia(no sinus P waves was found,the heart rate was 91 times per minute which was within 60-130 times per minute),ST depression was seen in all leads.Junctional tachycardia could be seen in patients with acute myocarditis.

Figure 2 Thyroid static imaging results:15 minutes after injection of 99mTc-O4-7mCi,the bilateral thyroid gland were swelling and the function was increased which was consistent with Graves' disease.



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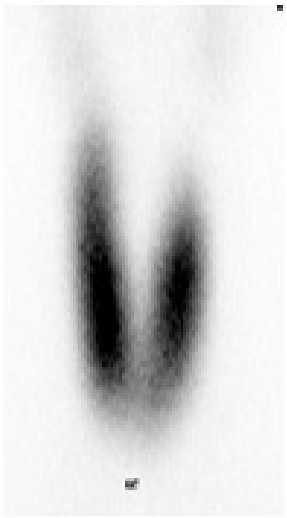

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