A 16-year-old boy with bronchial asthma and prinzmetal angina: case report

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Introduction

Prinzmetal’s angina is a very rare disease in children and adolescents. Adults’ studies suggest that vasospastic angina is more common in patients with bronchial asthma than in the general population.

Case presentation

A 16-year-old boy with a history of bronchial asthma was admitted to the hospital after successful resuscitation from asystole. On the day of admission, he had a severe left shoulder pain and developed cardiac arrest. He was complaining of left shoulder pain throughout the previous year. During his hospital stay, a second cardiac arrest took place with inferior ST elevation of the electrocardiography recorded after the second successful resuscitation. Diagnostic coronary angiography revealed multiple spasms throughout the coronary bed, which was completely resolved after intracoronary nitroglycerine administration. The patient was diagnosed Prinzmetal’s vasospastic angina, and the symptoms disappeared gradually with up-titration of a calcium channel blocker and a nitrate.

Discussion

Previous studies have suggested that the pathogenesis of Prinzmetal’s vasospastic angina may be similar to that of bronchial asthma, as we see in the presentation of this young patient.

Keywords

Case report • Vasospastic angina • Prinzmetal angina • Bronchial asthma • Coronary vasospasm • Adolecent

Learning points

- Prinzmetal’s angina and bronchial asthma may share similar pathogenesis; which include dependence on circadian variation, autonomic dysfunction, and inflammation.
- Bronchial asthma may increase the risk of new-onset coronary vasospastic angina. Therefore, we should be cautious even when the patient has atypical symptoms.

Timeline

- During 10 days before hospital admission: Frequent asthma episodes
- At first presentation: Severe left shoulder pain, cardiac arrest
- On the 6th day in hospital: Shoulder pain, another cardiac arrest
- On the 7th day: ST elevation episodes on 24 h electrocardiography monitoring
- On the 8th day: Diffuse coronary spasm on coronary angiography
- On 8th day: Treatment with amlodipin and nitrates initiated
- During 6 months: Slow up-titration of medication, which resulted to decrease of the frequency of shoulder pain episodes
- In a year: Almost no shoulder pain and good physical growth
- In 1.5 years: Patient quitted the medications and didn’t survive another cardiac arrest

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Case summary

A 16-year-old male patient was admitted to the hospital after successful resuscitation from cardiac arrest due to asystole. He had a medical history of bronchial asthma for 3 years duration and was currently taking Salmeterol/Fluticasone 50/100 for a year and Salbutamol as needed. His parents reported that during the last 10 days he had more frequent asthma episodes. He was complaining of left shoulder pain on the day of admission and had taken a non-steroidal anti-inflammatory drug for pain relief. Shortly afterwards, he fell unconscious as a result of cardiac arrest. After successful resuscitation with standard cardiopulmonary resuscitation with chest compressions and epinephrine, he was transferred to the hospital. On admission, the patient was pale and confused. SaO₂ 90%, heart rate 62 b.p.m., blood pressure (BP) 70/40 mmHg, normal heart sounds with no additional murmurs, and his chest was clear on auscultation. There was no elevation of cardiac enzymes. And his electrocardiography (ECG) was unremarkable. Echocardiography was performed, which also showed no abnormalities. In the absence of any cardiac abnormalities, his case was managed as exacerbation of bronchial asthma and he was transferred to the paediatric department. During his stay on the ward, he continued to complain of left shoulder pain from time to time. A shoulder X-rays found no abnormalities. On the 6th day of the hospital stay, he suffered another episode of asystole and cardiac arrest following severe left shoulder pain. This time there was small ST elevation recorded on inferior leads and inverted T waves on precordial leads on ECG after resuscitation (Figure 1). This episode was also not associated with any elevation of cardiac enzymes. A 24-h ECG Holter monitor was performed the next day and showed transient ST elevations on inferior leads during the day (Figure 2). ST-T changes were not related to

![After the second resuscitation](image-url)

**Figure 1** ECG after the second resuscitation.
physical activity, and no arrhythmia or pauses were recorded. A diagnostic coronary angiogram showed spontaneous diffuse vasospasm after the second injection of contrast. This was completely resolved after intracoronary nitroglycerine administration (Figure 3, Supplementary material online, Videos S1 and S2).

The patient was diagnosed with Prinzmetal’s angina.

His past medical records revealed that he had been complaining of left shoulder pain throughout the past year at almost every doctor’s appointment. X-rays were unremarkable, and the pain was dismissed. There were multiple discussions regarding implantable cardioverter-defibrillator (ICD) vs. pacemaker implantation. Pacemaker was preferred over ICD, because during all the events asystole was recorded, with no evidence of a ventricular arrhythmia, and also because of cost effectiveness, as state insurance didn’t cover all expenses. The parents refused both ICD and pacemaker implantation.

The treatment with small doses of nitrates and Amlodipine was initiated (Amlodipine 2.5 mg and Isosorbide dinitrate 5 mg). Up-titration proved to be challenging as the patient had low BP, which

Figure 2 Twenty-four hours Holter monitoring, which revealed transient ST elevations during the day.
In asthma, there is autonomic dysfunction, which is expressed with marked sensitivity to cholinergic constrictors and decreased sensitivity to adrenergic dilators (decrease of responsiveness of β2 receptors) and hyper-reactivity to α adrenergic receptors, which relies on the pathogenesis of vasoconstriction in Prinzmetal’s angina.5,6

In both cases, inflammation and smooth muscle dysfunction may play a significant role, inflammatory factors may be high in both cases.7–12 In one retrospective study, asthma significantly increased the risk of new-onset CVsA independent of other comorbidities and was significantly higher in previous users of oral or inhaled corticosteroids. It is considered that such correlation is rather due to the severity of the disease, which has led to corticosteroid prescription, than is an adverse effect of prescribed corticosteroid.4 Our patient had frequent asthma episodes with frequent exacerbations and was prescribed inhaled corticosteroids.

ECG changes during attack and the extent of coronary vasospasm may predict the type and severity of arrhythmia. Bradyarrhythmias are found to be more common in cases of inferior ST segment elevation,4 ventricular arrhythmias in anterior ST elevation, and lethal arrhythmias, such as ventricular fibrillation and advanced heart block are usually seen in cases of multivessel spasm.13,14 In the case of our patient, who developed asystole, ST elevation was recorded in inferior leads after the attack and during Holter monitoring, and coronary multivessel spasm was noticed during coronary angiography.

### Supplementary material

**Supplementary material is available at European Heart Journal – Case Reports online.**

**Consent:** The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient’s next of kin (parents) in line with COPE guidance.

**Conflict of interest:** none declared.

**References**

1. Lim CW, Hia CP, Chia BL, Quek SC. Variant angina in a 6-year-old boy. Int J Cardiol 2007;115:624–626. PubMed PMID: 17067693
2. Wilkes D, Donner R, Black I, Carabello BA. Variant angina in an 11 year old boy. J Am Coll Cardiol 1985;5:761–764. PubMed PMID: 3973275.
3. Rich MW. An association between Prinzmetal’s angina pectoris and obstructive lung disease. Am J Cardiol 2005;96:1612–1613.
4. Hung MJ, Mao CT, Hung MY, Chen TH. Impact of asthma on the development of coronary vasospastic angina: a population-based cohort study. Medicine (Baltimore) 2015;94:e1880. PubMed PMID: 26496346; PubMed Central PMCID: PMC4620748
5. Yasse H, Kugysma K. Coronary spasm: clinical features and pathogenesis. Intern Med 1997;36:760–765.
6. Lewis MJ, Short AL, Lewis KE. Autonomic nervous system control of the cardiovascular and respiratory systems in asthma. Respir Med 2006;100:1688–1705.
7. Hung MJ, Cheng WJ, Cheng CW, Li LF. Comparison of serum levels of inflammatory markers in patients with coronary vasospasm without significant fixed coronary artery disease versus patients with stable angina pectoris and acute coronary syndromes with significant fixed coronary artery disease. Am J Cardiol 2006;97:1429–1434. PubMed PMID: 16679078
8. Itoh T, Mizuno Y, Harada E, Yoshimura M, Ogawa H, Yasue H. Coronary spasm is associated with chronic low-grade inflammation. Circ J 2007;71:1074–1078.

**Figure 3** Coronary angiography demonstrating multivessel spasm.
9. Hung MJ, Hsu KH, Chang NC, Tsimikas S, Hung MY. Prevalence of coronary artery spasm after stent placement and its association with inflammation. *Int J Cardiol* 2015;179:252–255.

10. Tang XQ, Sun WP, Xu HB, Liu WB, Wang TS, Liu Hj. The changes in the levels of IL-6, IL-17, and IL-21 in the acute stage of childhood asthma. *Clin Lab* 2013;59:1381–1387.

11. Takemura M, Matsumoto H, Niimi A, Ueda T, Matsuoka H, Yamaguchi M, Jinnai M, Muro S, Hirai T, Ito Y, Nakamura T, Mio T, Chin K, Mishima M. High sensitivity creatine protein in asthma. *Eur Respir J* 2006;27:908–912.

12. Hartley R, Berair R, Brightling CE. Severe asthma: novel advances in the pathogenesis and therapy. *Pol Arch Med Wewn* 2014;124:247–254.

13. Nakamura M, Takeshita A, Nose Y. Clinical characteristics associated with myocardial infarction, arrhythmias, and sudden death in patients with vasospastic angina. *Circulation* 1987;75:1110–1116.

14. Myerburg RJ, Kessler KM, Mallon SM, Cox MM, DeMarchena E, Interian A Jr, Castellanos A. Life-threatening ventricular arrhythmias in patients with silent myocardial ischemia due to coronary artery spasm. *N Engl J Med* 1992;326:1451–1455.