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Myocardial Infarction in Children

Meki Bilici, Mehmet Ture and Hasan Balik

Abstract

Myocardial infarction (MI) is a clinical condition that develops associated with a sudden reduction or interruption of the blood flow of the vessels supplying the heart for various reasons. The electrocardiographic, echocardiographic and enzymatic diagnostic criteria of MI have been well defined in adults, in children there are some difficulties. Although seen more often in the presence of congenital heart disease (CHD), MI may also be seen in patients without CHD. Unlike atherosclerotic coronary artery disease in adult patients, ischaemia and infarct in children are often associated with coronary artery anomalies and CHD. In addition, congenital prothrombotic diseases, vasculitis, surgical or interventional procedures may also cause ischaemia and infarct. Subendocardial ischaemia, especially aortic stenosis characterised by hypertrophy in the left ventricle is often seen in hypertrophic cardiomyopathy or hypertensive patients. The most important risk factors in neonates and infants are the presence of CHD, coronary artery anomalies and perinatal asfixia. The most frequently seen causes of pediatric myocardial infarction (PMI) are abnormal left coronary artery originating from the pulmonary artery (ALCAPA) and Kawasaki disease. Another often seen cause of PMI is patients who underwent arterial switch operations.

Keywords: children, myocardial infarction, coronary artery anomalies

1. Myocardial infarction in children

Myocardial infarction (MI) is a clinical condition that develops in association with a sudden reduction or interruption of the blood flow in coronary vessels supplying the heart for various reasons. Coronary artery spasm and myocardial ischaemia are seen in the early stage of occlusion. If the relevant coronary artery is not rapidly re-channelled or cannot be re-vascularised, then MI develops [1]. Myocardial infarction is a common event in adults, but is not common among children. Furthermore, although the electrocardiographic, echocardiographic and
enzymatic diagnostic criteria of MI have been well defined in adults, in children there are some difficulties [2, 3]. As the cardiac structure changes with age, there are sometimes difficulties in the electrocardiographic diagnostic criteria of ischaemia.

Although MI is seen more often in the presence of congenital heart disease (CHD), it may also be seen in patients without CHD. Unlike atherosclerotic coronary artery disease in adult patients, ischaemia and infarct in children are often associated with coronary artery abnormalities and CHD [4]. In addition, congenital prothrombotic diseases, vasculitis, surgical or interventional procedures may also cause ischaemia and infarction [5]. Subendocardial ischaemia, especially aortic stenosis characterised by hypertrophy in the left ventricle is often seen in hypertrophic cardiomyopathy or hypertensive patients [2].

The most important risk factors in neonates and infants are the presence of CHD, coronary artery abnormalities and perinatal asfixia [5, 6]. The most frequently seen causes of Paediatric myocardial infarction (PMI) are abnormal left coronary artery originating from the pulmonary artery (ALCAPA) and Kawasaki disease [7, 8]. Patients undergoing arterial switch operations are also at increased risk for PMI [9].

2. Anamnesis

The anamnesis in Paediatric myocardial infarction (PMI) and Paediatric myocardial ischaemia and physical examination findings show differences from adult cases. The anamnesis of infants and young children is taken from the family and carers [2]. The complaints usually reported in this period are generalised findings such as feeding problems, lack of appetite, irritability, diarrhoea, vomiting, cold extremities, pallor and tachypnea. Older children may be able to describe chest pain well and can explain the spread of pain. A compressive of chest pain spreading to the left arm and shoulder should suggest chest pain with cardiac origin [10, 11]. However, some children may not be able to describe the character of the chest pain.

In the physical examination, patients are generally anxious, pale and interactive. They may have dyspnea or tachypnea. If tachycardia, hypotension or cardiogenic shock develop, these can be determined [2]. In the cardiac examination, rhythm irregularity and gallop rhythm can be determined. Extremities may be cold and the pulse may be weak on the electrocardiography (ECG), ventricular arrhythmia or cardiac block may be determined [2, 12–14]. Patients with ventricular arrhythmias may have symptoms of palpitations, syncope and loss of conscious [12].

3. Cardiac chest pain

The anamnesis has great value in the determination of whether or not the chest pain is from cardiac origin. In the case of a child presenting with chest pain, it must be determined from the family when the pain started, how often the child has experienced chest pain, how long the pain lasts, where the pain radiates to, the relationship with exercise, factors that increase or decrease the pain, whether or not there is any relationship with feeding or respiration, whether there is any trauma anamnesis, whether or not there is any fever, or accompanying
complaints such as shortness of breath, sweating, palpitations or nausea [10, 11]. It must also be determined whether the child or any family member has any CHD and whether or not any family member has recently experienced any chest pain, or MI.

Chest pain, which is one of the most significant symptoms for adults presenting to the Emergency Dept, is generally has a benign character in children. However, it is extremely important to decide whether or not the pain frequently seen in children is of cardiac origin [15]. Chest pain with cardiac origin in childhood can be classified in 3 groups; as structural heart diseases, inflammatory causes and dysrhythmias [10]. Structural heart diseases can lead complaints associated with an increased need for oxygen or a reduction in coronary blood circulation. These include events such as hypertrophic obstructive cardiomyopathy or aortic stenosis because of an obstruction in the left ventricle outlet tractus. Coronary artery abnormalities may also cause coronary ischaemia.

Chest pain with cardiac origin generally presents in situations where an increase in cardiac output is required. It is typically in the precordial or substernal region, in a constricting form and radiates to the left arm, neck and jaw. In some cases, there may also be shortness of breath, sweating, nausea, vomiting or syncope. In infants, the findings may be seen as feeding difficulties, crying and screaming (Table 1), [2, 6, 15, 16].

After the anamnesis and physical examination, ECG examination must be made in all patients and X-ray imaging should be applied in order to exclude any respiratory causes [15]. In cases where the pain is thought to be of cardiac origin, troponin and creatine kinase myocardial band (CK-MB) levels must be examined and if necessary echocardiographic evaluation should be made [15, 17, 18].

<table>
<thead>
<tr>
<th>Neonates</th>
<th>Older children</th>
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<tr>
<td>Feeding problems</td>
<td>Fatigue</td>
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<tr>
<td>Lack of interest in surroundings</td>
<td>Lack of appetite</td>
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<tr>
<td>Irritability</td>
<td>Paleness</td>
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<tr>
<td>Diarrhoea</td>
<td>Dyspnoea</td>
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<tr>
<td>Sweating</td>
<td>Tachypnea</td>
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<td>Vomiting</td>
<td>Tachycardia</td>
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<td>Pallor</td>
<td>Hypotension</td>
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<tr>
<td>Tachypnea</td>
<td>Weak pulse</td>
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<tr>
<td>Dyspnoea</td>
<td>Rhythm irregularity</td>
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<td>Sudden paroxysmal abdominal pain</td>
<td>Gallop rhythm</td>
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<td>Cold extremities</td>
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<td></td>
<td>Shock</td>
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<td></td>
<td>Ventricular arrhythmia</td>
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<td>Heart block</td>
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Table 1. Symptoms and physical examination findings in Paediatric myocardial infarction.
4. Electrocardiography in Paediatric myocardial infarction

The 12-lead ECG is an integral part of the evaluation of coronary artery disease [16]. Elevation of the J point which joins the QRS and the ST segment is the first finding of myocardial ischaemia [7]. Compared to baseline, an elevation of 1–2 mm is seen in the J point and the ST segment in myocardial ischaemia. An elevation of >2 mm should rouse suspicion of MI (Figures 1 and 2). When ST elevation is determined, there should be a progression through differential diagnosis of benign early repolarisation, pericarditis, MI, bundle branch block and left ventricle aneurism [18]. While pathological ST elevation does not show variability, J point and ST elevation seen in early repolarisation in healthy adults is generally corrected with isoproteronol infusion or exercise [19].

The presence of PR segment depression is an ECG finding which is valuable in the differentiation of myopericarditis from MI in favour of myopericarditis [18]. The positive predictive value of PR depression seen in chest and extremity derivations has been determined to be 96.7% [19].

T-wave alterations generally accompany ST segment alterations in AMI. Initially, T-waves may be long and sharp (hyperacute T-wave). These changes determined on ECG show myocardial injury (Figure 3). ST depression may reflect the reciprocal effect of the region in the derivation corresponding to approximately 180° [7]. The standard leads does not show ST segment elevation in patients with true posterior wall MI. Instead ST elevation, ST segment depression may be seen as a reciprocal change in V4R-V2 on the ECG [20, 21].

Electrocardiographic findings that show recent MI are pathological Q-waves. A long Q-wave from 3 small squares (0.12 secs) in whichever derivation should be evaluated as pathological Q. In addition, broad Q-wave in V1 and V2 may be seen in patients with left ventricle hypertrophy. In short, the evaluation of anamnesis, physical examination and laboratory findings together with the ECG findings is important for every patient.

Generally pathological Q-waves are seen to emerge within the first 12–48 hours on at least 2 adjacent leads, and when they are present at inferior, lateral or anterior derivations, they have

Figure 1. 2–3 mm ST elevations in DII, DIII, aVF, V5, and V6 leads of electrocardiography show that myocardial infarction.
Figure 2. Coronary angiography revealed total occlusion of the left anterior descending (LAD) and distal circumflex (CX) coronary arteries (red arrows).

Figure 3. In this patient with aortic stenosis, ST depression on V4–6 and negative T wave show that coronary ischemia.
got extremely high value for the diagnosis of MI [1, 21]. However, in Paediatric patients, the determination of Q-wave in just one derivation could even be sufficient to determine MI [21]. While these Q-waves show infarct of the myocardial wall, high R waves in V1 and/or V2 (negative Q-waves) may represent true posterior wall MI. On the ECG of approximately half of cases, pathological Q-waves have a tendency to regress with time. In newborn infants, the presence of Q-waves in derivations DII, DIII and AVF may be normal. Furthermore, if ECG leads are placed on the upper part of the chest, Q-waves can be incorrectly shown in V5–6 that can cause misinterpretation of an integral part of the evaluation of coronary artery disease [16]. Elevation of the J point that joins the QRS and the ST segment is the first finding of myocardial ischaemia [7]. Compared to baseline, an elevation of 1–2 mm of the J point and the ST segment can be determined in myocardial ischaemia. An elevation of >2 mm should rouse suspicion of MI [7].

When the clinician has suspicions on the presence of pathological Q-wave, ECG in deep inspirium can be helpful. There is a change in the voltage of the Q-waves in physiological cases while there is no change of voltage at deep inspirium in pathological Q waves [7]. Towbin et al. reported that the presence of Q-waves wider than 35 ms on ECG was the most valuable finding for MI and a diagnosis of transmural MI diagnosis should not be made in patients with no Q-wave abnormalities [2].

Towbin et al. evaluated the ECG and clinical findings in the pre-MI records of 37 patients who died because of MI. In this retrospective study, it was reported that findings of MI were determined on the pre-mortem ECG records of 28 children who suffered fatal acute MI, while it was reported in 9 cases who died because of chronic MI [3]. It was also reported by the same authors that when MI was determined in a hypertrophic heart, the infarction was determined at the hypertrophic ventricle. This showed that in PMI, the presence of hypertrophy was a risk factor for MI. At least one of the criteria shown in the Table 2 was determined to be present in 30 patients included into the study by Towbin et al [2]. Furthermore, no finding was determined on ECG in approximately 19% of the cases in that study, ECG was not sufficient to make a diagnosis of PMI alone. The evaluation of the patient should be made together with the anamnesis, physical examination, ECG and laboratory data.

Furthermore, Nakanishi et al. showed that deep Q-waves were a good marker for MI in Kawasaki patients. It was also determined in the same study that T-wave inversion in derivations II, III and AVF showed MI in the inferior wall [22].

| 1. New appearance of wide Q waves >35 ms in duration |
| 2. Increased amplitude or duration [>35 ms] of pre-existing Q waves |
| 3. New onset Q waves in serial tracings |
| 4. Q waves notching |
| 5. ST segment elevation [≥2 mm] and prolonged QT interval corrected for heart rate [>440 ms] with any other criteria. |

Table 2. Electrocardiographic findings significant for MI in children, as reported by Towbin et al.
In the adult guidelines of MI criteria, it is stated that there should be ECG changes in more than one lead [23]. However, in Paediatric cases, when there are ECG changes in one derivation, there should be suspicion of MI [3]. Moreover, the observation on ECG of more than one of these changes, such as ST elevation, Q-wave changes, ST depression or T-wave inversion should more strongly suggest MI diagnosis.

5. Cardiac enzymes

An increase in the level of enzymes released into circulation from cells exposed to injury is important in the diagnosis of MI. These enzymes are creatine kinase myocardial band (CK-MB) and troponin [7, 10]. In all Paediatric cases thought to have myocardial damage, the troponin level should be examined. Values more than 2 ng/ml value are especially more valuable for cardiac origin [17]. Even in cases of mild damage in myocardial cells, an increase in enzyme levels may be seen [19]. In addition, the events causing an increase in troponin levels must be known (Table 3) [6].

6. Echocardiographic evaluation of Paediatric acute myocardial infarction

<table>
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<th>Acute heart failure</th>
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<tr>
<td>Cardiac contusion</td>
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<td>Myopericarditis</td>
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<td>Pulmonary embolism</td>
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<td>Sepsis</td>
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<td>Strenuous exercise</td>
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<td>Sympatomimetic drugs</td>
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<td>Tachyarrhythmia</td>
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Table 3. Non-coronary events which increase troponin.

The increasing experience with echocardiography [echo] in recent decades has greatly facilitated the diagnosis of acute myocardial infarction [AMI], as echo is an inexpensive, readily available, ambulatory, non-invasive method [24]. Echo is useful, not only in the diagnosis of AMI but also in prognosis, the monitoring of complications and in follow-up. In Paediatric AMI patients, echo provides very valuable information in the determination of segmentary wall movement abnormalities and in the diagnosis of CHD, pericarditis, myocarditis, Kawasaki disease, cardiomyopathy, aortic stenosis and ALCAPA which often accompanies chest pain. In adult studies, abnormal wall movement findings have been determined in 91% of patients applied with echo in the early stage in
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Myocardial Infarction
Myocardial Infarction
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