CASE REPORT

Complete Atrioventricular Block in Children

by

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Abstract

Two cases (a 7 years and a 2 years old boy) suffering from complete atrioventricular block have been reported. Both of them were found accidentally without any subjective symptoms of atrioventricular block although the ventricular rate was less than 45 times per minute. The diagnosis was easily established with eletrocardiographic examinations. It was necessary to detect the underlying diseases, and for this purpose some appropriate laboratory examinations were done. Rheumatic fever and viral myocarditis were suspected to be the underlying disease in these cases. The follow up during 7 years for the case with rheumatic fever and 3 months for the case with viral myocarditis showed that the abnormalities were permanent, and they did not require any specific treatment for the atrioventricular block itself.

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Introduction

Atrioventricular block is a cardiac mechanism resulting from defective conduction of the impulse from the atria to the ventricle through the atrioventricular node (Friedberg, 1966; Nadas, 1972). The most severe type is the third degree, the total or complete atrioventricular block. In such a case, there is damage or disfunction of the atrioventricular conduction system in a certain place so that none of the atrial impulses reaches the ventricle; the atria and the ventricle beat independently and do not have a synchronous contraction. The ventricle continues to beat in response to a new pacemaker in the atrioventricular bundle below the site of block.

Complete atrioventricular block may be either congenital or acquired; the acquired form may be permanent or temporary whereas the congenital form can be diagnosed in utero by the fetal electrocardiogram and sound tracing, with simultaneous electrocardiogram of the mother (Friedberg, 1966). Anatomic interruption of the conduction pathway can not always be found and it may only be a functional disturbance. Clinical manifestation of this disease is wide in variety. Subjective symptoms are often absent and usually discovered accidentally during a routine physical examination for another disease. Sometimes the patients are found in the state of Adams-Stokes syncope attack or sign and symptoms of an underlying heart disease. Prevention as well as quick and prompt treatment of the syncope attack is essential.

The purpose of this paper is to report 2 boys suffering from complete atrioventricular block with different backgrounds, admitted to the Child Health Department, Medical School, Gajah Mada University/Dr. Sarjito General Hospital, Yogyakarta, with special attention to the diagnostic approach and follow up. The diagnosis of this disease is relatively easy to establish by electrocardiographic examination, but determination of the etiology is more important so that a rational and adequate management can be given.

Case Reports

CASE 1 : D.F., a 25 months old boy was brought to the doctor on May 29, 1984 because of a fever, cough and cold during 3 days. The doctor then refered him to the Dr. Sarjito General Hospital because of bradycardia. Two weeks ago he had high fever for 5 days accompanied by cough and cold. He had been brought to a pediatrician 2 times but did not recover completely and 3 days ago he was again feverish. He had seldom been ill and had always been active before hand. During his illness he did not look weak, was still active as before, never suffered from dyspnea, cyanosis or edema and never fainted. His growth and development was satisfactory. Basic DPT immunisation were given 2 times at the age of 6 and 9 months. On the first examination his general condition appeared to be good, so was his nutritional state. The child was active. The pulse rate was 70/minute, regular; the respiration rate 30/minute, regular; the temperature 37° C and the blood pressure 140/80 mmHg. No cardiac enlargement was noted. A mild brady-

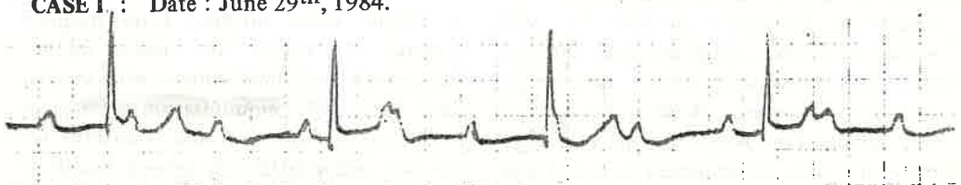
cardia was found but there was no heart murmur heard on auscultation. Neither right nor left heart failure was found. Electrocardiographic record showed a total atrioventricular block : the atrial and the ventricular rate was 154/minute and 78/minute respectively. There was no continuation between the P wave and the QRS complex; the QRS complex did not widen. The boy was discharged. A congenital complete atrioventricular block was suspected.

On the follow up 2 weeks later his general condition remained good but there was a decrease of the QRS frequency. It became namely 55-60 times per minute, whereas the P wave was still 150/minute. The patient was then admitted to the hospital for further examinations. Neither pseudomembrane nor diphtheria bacillus were found on throat examination. Chest x-ray showed no heart enlargement. Blood electrolytes, SGOT and SGPT were normal. Lactic dehydrogenase (LDH) was 378 unit/liter (normally 80 - 240 U/1). Creatine phosphokinase examination was not performed because of technical difficulties. The white blood count was 9.300/cu mm, the hemoglobin 10 gram/dl and the blood sedimentation rate for the first and second hour was 10 and 21 milimeter respectively. Myocarditis was suspected and a cardiac monitoring was fitted. Intravenous Ampiclox, antidiphtheria serum, and prednison 2 mg/kg. body weight/day were administered. The general condition remained good, the ventricular rate was variable: 44 to 48 times per minute during sleep and 56 times per minute when awake, and it became 80 times per minute when he cried. After a week the LDH level decreased to 290 U/1, Creatine kinase NAC-activated (CK-NAC) 123 U/1 (normally 20-70 U/1). The electrocardiographic pattern remained showing a ventricular bradycardia with a complete atrioventricular block, the atrial rate decreased to 100-120 times per minute and the ventricular rate remained 48 to 56 times per minute in serial electrocardiographic examination. LDH, CK-NAC and CK-MB (Creatine kinase myocardial type) were within normal limits two weeks after the initial examination. Follow up examination during 3 months showed a stable condition and his general condition remained good.

Electrocardiogram.

Conclusion : 1. Ventricular bradycardia.
2. Complete Atrioventricular block.

CASE I : Date : June 29th, 1984.

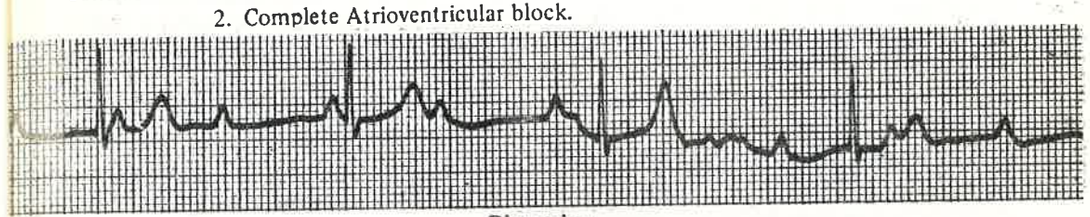


CASE 2 : A.W., a 7 years old boy sometimes complained of intermittent joint pain since he was 4 years old. One week before his admission to the hospital he had fever and cough and complained of recurrent joint pain without swelling. There were no complaints of dyspnea or edema. He was refered to the hospital by a general practisioner on Desember 7, 1976. On examination his pulse rate 48 times per minute, regular; the temperature was 37°Celcius, the respiration rate 32 times per minute and the blood pressure 125/80 mm.Hg. The heart was enlarged to the left and downward reaching the medial of the anterior axillary line; a 3rd grade pansystolic murmur with the maximal punctum at the apex was noted. Neither right nor left heart failure were found. Antistreptolysine-O level increased and chest x-ray showed a cardiomegaly with a cardiothoracic ratio a 0,58. The electrocardiographic record showed a complete atrioventricular block : the atrial rate was 105/minute and the ventricular 43/minute. The patient was treated as suffering from rheumatic fever and he got penicilline protection given regularly until now.

A 7.5 years follow up showed no sign of rheumatic fever exacerbation, his growth remained normal and he was able to participate normally in physical activities without complaints. The systolic murmur was still present but there was reduction of its intensity. The last electrocardiographic record remained showing a complete atrioventricular block with the atrial and the ventricular rate of 84 and 38 times per minute respectively without any sign of syncope.

CASE II.: Date : April 22nd, 1982.

Conclusion : 1. Ventricular bradycardia.
2. Complete Atrioventricular block.



Discussion

Complete atrioventricular block in children is very rare. Nadas (1972) reported 61 cases during 28 years from 1935 to 1963. In the Pediatric Cardiology sub-department Dr. Sarjito General Hospital/Medical School Gajah Mada University, beside fatal cases caused by diphtheria, only 2 cases were found during 8 years (1976 to 1984). The cause of complete atrioventricular block is usually congenital anomalies. Recently, open cardiac surgery is also considered as one of its main causes (Nelson, 1979). The congenital type might be caused by a defect of the main branch of the His bundle (Nora and Wolfe, 1976). An International study of about 600 cases of congenital complete atrioventricular block showed that 70% of them had no other heart anomaly (Nelson, 1979).

Miller and Rodriguez-Coronel (1968) found 45 cases of severe congenital atrioventricular block and 29% of them had also congenital cardiac anomalies. Of 61 cases presented by Nadas (1972), 15 had congenital heart anomalies, 11 had normal hearts, 17 cases had probable and 18 had posible congenital heart disease. The most frequently associated cardiac malformation were "corrected" transposition of the great arteries (ventricular inversion), single ventricle, and patent ductus arteriosus. Isolated ventricular septal defect is seldom associated with complete atrioventricular block (Nelson 1979). Other cardiac malformations often reported as the causes of atrioventricular block are secundum atrial defect, tetralogy of Fallot, mitral atresia, ventricular septal defect with pulmonary stenosis and endocardial

fibroelastosis. Acquired complete atrioventricular block is rarely found in children. Nadas (1972) found this total atrioventricular block in rheumatic heart disease, occurring temporarily. Rheumatic heart disease seldom causes complete atrioventricular block in children, though prolongation of the P-R interval occurs commonly. Heart block may be found during and after diphtheria, scarlet fever, measles, rubella, typhoid fever, pneumonia, typhus, viral myocarditis, mumps, and influenza (Miller and Rodriguez-Coronel, 1968). Progressive myocardial degeneration and fibrosis can give atrioventricular block. James as cited by Miller and Rodreguez-Coronel (1968) found a fatal case of progressive muscular distrophy of which the death was caused by atrioventricular block. Trauma, inflammation, granuloma, malignant cells infiltration to atrioventricular node and severe electrolyte imbalance have also been reported as the cause of complete atrioventricular block. Complete atrioventricular block, therefore, may be due to congenital or acquired anatomic interruption of the conduction pathway or to physiologic alteration. In the two cases mentioned above the underlying diseases were rheumatic disease and a previous upper respiratory tract infection. Further evaluation showed permanent atrioventricular block and no other symptoms developed that might disturb the child's activity. Children with complete atrioventricular block might have a normal or near normal working capacity and can tolerate strenuous muscular exercise, especially when the ventricular rate is not very slow. Thus it is usually asymptomatic (Miller and Rodriguez-Coronel, 1968). Exercise can increase cardiac rate usually by 10 to 20 beats per minute (Nelson, 1979; Nadas, 1972) or 20 per cent (Friedberg, 1966). Complete atrioventricular block is often overlooked because the ventricular rate is relatively fast, 40 to 56, and in diphtheritic heart block it may even exceed 80/minute (Friedberg, 1966). Several laboratory examinations are necessary to perform, such as peripheral blood smear, blood sedimentation rate, hemoglobin, hematocrit, SGOT, SGPT, Lactic dehydrogenase, Creatine kinase, Creatine kinase Myocardial type (CK-MB) and blood electrolytes. It is also necessary to perform a chest X-ray Photo. To determine the etiology, bacteriologic examination can be done from blood, pharyngeal swab, feces and urine. Virologic examination is occasionally also indicated. These examination should be done immediately. In our two cases, laboratory examination were not complete because of technical difficulties.

Treatment

Treatment is rarely indicated for the atrioventricular block itself. Treatment of the causative factor may result in abolition of the atrioventricular block; for example digitalis should be discontinued if the heart block is caused by digitoxicity, in myocarditis it is important to treat the underlying disease. Careful and continuous observation is essential because of the possible development of Adams-Stokes syndrome (Friedberg, 1966). Asymptomatic cases with the heart rate of greater than 40/minute during sleep do not require treatment but require regular control. Hard physical activity must be restricted. When the heart rate is less than 40/minute, it may be necessary to give oral isoproterenol or ephedrine to increase the ventricular rate, intermittent or continuously (Nora and Wolfe, 1976). Atrioventricular block may require more specific treatment when it is associated with symptoms, especially dizziness, faintness or Adams-Stokes seizures or with refractory heart failure. Treatment may be necessary for the acute attack or to prevent recurrent attacks. Anti Diphtheria serum was given to case I, although there was no diphtheria bacillus found in the pharyngeal swab. This measure was done because he had a fever 3 weeks before and the most frequent cause of atrioventricular block in this age is diphtheria.

Within recent years the administration of chlorothiazide has been used in some patients who have a slow ventricular rate and complete atrioventricular block. This help partly by reducing edema and partly by causing potassium diuresis, resulting in a low total body potassium level, thus increasing cardiac irritability and causing a higher ventricular rate (Nadas, 1972).

It is better to hospitalized a patient with mild symptomatology or in those who have no symptoms but who have ventricular rates lower than 50/minute. The ventricular rate response to a standardized exercise load and to Isuprel administered intravenously must be evaluated. If there is no satisfactory exercise response, but Isuprel increases the rate by more than 50%, sublingual Isuprel administration should be given as therapeutic trial. If, even with the use of Isuprel, attack of dizziness or Adams-Stokes syndrome persists, a pacemaker should be inserted (Nadas, 1972).

Adams-Stokes attack must be managed immediately.When there is no pulse, blood pressure or cardiac sound, precordial thump must be performed immediately.If this procedure is ineffective, cardiopulmonary resuscitation should be initiated. As soon as possible the heart should be paced with an external cardiac pacemaker. If this is not promptly available, an intravenous infusion should be started with 1 mg. of isoproterenol in 200 cc. 5% dextrose solution at initial rates of 15 to 30 drops (5 to 10 micrograms) per minute. Frequently, it requires 5 to 50 micrograms per minute. Sometimes 0,1 to 0,4 mg. of Isuprel is injected intramuscularly or even intravenously if there is a delay in setting up an intravenous infusion, or 0,3 to 0,5 cc. of epinehrine may be injected intramuscularly, intravenously or even intracardially if cardiac arrest persists. Implantation of a pacemaker is recommended when these medicaments are ineffective. If implantation of a pacemaker is not performed, the patient should be placed on long-term oral isoproterenol therapy (Friedberg, 1966).

Conclusion

Two cases consisting of 7 and 2 years old boys suffering from complete atrioventricular block with rheumatic fever and viral myocarditis as suspected underlying diseases have been reported. They were found accidentally with bradycardia without any subjective symptoms. Although the majority of complete atrioventricular block are congenital, it is always necessary to detect the possibility of acquired causes, so that an adequate and rational follow up can be done.

The two cases showed permanent complete atrioventricular block and they did not require any specific treatment. Specific treatment for atrioventricular block are limited for cases with symptoms caused by bradycardia. The treatment is usually directed to the underlying diseases.

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