

compared to baseline homocysteine levels ($12.9 \pm 5.4 \mu\text{mol/l}$ vs $11.7 \pm 3.6 \mu\text{mol/l}$ $p=0.045$), there was also a significant decrease in homocysteine levels at the end of 3rd month of metoprolol use when compared to baseline homocysteine levels ($12.9 \pm 5.4 \mu\text{mol/l}$ vs $10.4 \pm 3.1 \mu\text{mol/l}$ $p=0.011$) and there was a significant difference in homocysteine levels between between 3rd and 6th months ($11.7 \pm 3.6 \mu\text{mol/l}$ vs $10.4 \pm 3.1 \mu\text{mol/l}$ $p=0.010$).

In accordance with previous findings (2-5), we found that beta-blocker therapy significantly decreased homocysteine levels in patients with CAE. Additionally, we have showed for the first time that calcium-channel blocker therapy significantly decreased homocysteine levels in patients with CAE.

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A case with Wolf-Parkinson-White syndrome first presented with a devastating event: aborted sudden cardiac death

İlk bulgusu ani ölüm olan Wolf-Parkinson-White sendromu tanılı bir olgu

Wolf-Parkinson-White (WPW) syndrome is a cardiac conduction disorder that can present with potentially life-threatening consequences. Sudden death very rarely occurs as the initial presentation of WPW syndrome. Sudden cardiac death (SCD) is defined as a natural and unexpected death due to cardiac causes that occurs within 1 hour of onset of symptoms (1). Wolff-Parkinson-White syndrome is a cardiac conduction disorder that can present with potentially life-threatening consequences. Sudden death rarely occurs as the initial presentation of WPW syndrome (2). Unpredictable lethal complications were found in the asymptomatic WPW patients, creating uncertainty whether these patients should receive aggressive evaluation and interventional therapy.

In this case report, we presented a female children with WPW syndrome admitted to our hospital with sudden cardiac death as an initial finding of her disease.

A 13-year-old female patient admitted to our emergency department due to sudden loss of consciousness when walking in the school garden for ceremony rehearsal activity. She said to her friends that she had palpitation and chest pain just before the loss of consciousness. The headmaster immediately called out an ambulance. In about 5 minutes, ambulance arrived at the school. Ambulance paramedics reported that when they arrived, the patient was in cardiopulmonary arrest; they applied cardiopulmonary resuscitation. The patient was brought from the school to our emergency department in about 10 minutes by the ambulance. During the transportation, paramedics kept on doing cardiopulmonary resuscitation. The physical examination in the emergency department revealed that the patient was unconscious and had cardiovascular instability with clinical evidence of low cardiac output. Electrocardiography revealed ventricular fibrillation. Ventricular fibrillation was treated promptly with electrical defibrillator (1 joule/kg) and the patient returned to normal sinus rhythm. Arterial blood gas analysis revealed severe metabolic acidosis and HCO₃ replacement was performed. Intravenous fluid and dopamine infusions were started and the patient was referred to pediatric intensive care unit from emergency room. She did not have any previous history of drug usage, trauma or any infection. Before this event, she did not experience any episode of syncope, palpitation or chest pain. Family history was unremarkable. Mechanic ventilatory support was provided in intensive care unit. On physical examination at the admission to pediatric intensive care unit, the patient was unconscious with the Glasgow score of 4 (E1M3V_e). Heart rate, arterial blood pressure and body temperature were 120/min, 110/70 mmHg, 36,7 °C respectively. Pupils were isochoric; direct and indirect pupil light reflexes were positive on both eyes. There was not any sign causing suspicion of trauma. On cardiac examination, S1 and S2 were normal and there was no heart murmur. The physical examination findings of other systems were unremarkable. For brain edema, developed secondary to hypoxia, head cooling was applied. Blood ions, renal and liver function tests were all within normal limits. Computed brain tomography was normal. The electrocardiogram showed sinus rhythm with ventricular pre-excitation (short PR interval, wide QRS complex with delta wave) (Fig. 1). Prediction of accessory pathway from the resting surface electrocardiogram (ECG) according to QRS polarity was left-lateral. Because QRS polarity was positive on leads V1 and III. On the first day of hospitalization, generalized tonic-clonic convulsions were observed and antiepileptic treatment was started. Patient needed prolonged mechanical ventilatory support of 30 days due to Adult respiratory Distress Syndrome (ARDS) developed secondary to aspiration pneumonia. Due to prolonged need for respiratory support and inefficient cough and gag reflex, patient underwent open surgical tracheostomy. Patient was fed via nasoduodenal catheter then via gastrostomy. During follow up, Glasgow score improved from 4 to 8 (E4M3V1). Unfortunately, patient had severe neurological sequelae. On physical examination 40 days after admission, there was spasticity on all extremities, more prominent on upper extremities. Deep tendon reflexes were increased in all extremities. These findings were consisted with first motor neuron lesion. Baclofen, phenytoin and physical therapy program was prescribed for the bedridden patient. Cranial magnetic resonance imaging revealed multiple foci of hypointense lesion on T1-weighted images and hyperintense lesion on T2-weighted images and these findings were consisted with hypoxic ischemic encephalopathy. During follow-up, we did not observe any arrhythmia including atrial fibrillation. Electrophysiologic study (EPS) and ablation was planned.

Although SCD rarely occurs at youth, when it does, it is a devastating event for both the family and the medical community. The SCD is defined as a natural and unexpected death due to cardiac causes that occurs within 1 hour of onset of symptoms (1).

The WPW syndrome is a recognized cause of sudden death. Sudden death rarely occurs as the initial presentation of WPW syndrome (2). The exact prevalence of sudden death in WPW syndrome is unknown.

Previous natural history studies have reported sudden cardiac death rates of 0.0% to 0.6% per year for patients with WPW. In a recent study of less than 35-year-old SCD victims, WPW was identified on 10.5% of the available ECGs (3).

Previous studies have shown that ventricular fibrillation can be the first event of the WPW syndrome. In the study of and Timmermans et al (4) ventricular fibrillation was the first manifestation of the WPW syndrome in 53% of their series. Similar to our case, most of sudden deaths have the peculiarity to occur during exercise.

At the time of the curative treatment of WPW syndrome by radiofrequency ablation, it is important to detect the forms at risk of sudden death. Patients incidentally found to have WPW electrocardiogram morphology are difficult to manage. Whether such patients should receive further invasive or non-invasive approaches of all patients is debatable, especially in asymptomatic patients, because of the low incidence of sudden cardiac death in this group. Natural history studies suggest an excellent prognosis and encourage non-intervention. However, such studies are logistically difficult and suffer problems of patient dropout and inadequate follow-up. Because sudden death may be the first, although infrequent, clinical manifestation of the WPW syndrome, conservative therapy policy is under arguments and it has been proposed that all patients should receive intervention. Rinne et al. (5) have presented evidence favoring invasive electrophysiological testing in all patients with WPW syndrome and palpitation. Risk stratification is performed to determine which individuals with WPW syndrome are at risk for sudden cardiac death. Sudden cardiac death in these individuals is due to the propagation of an atrial arrhythmia to the ventricles at a very high rate. Individuals with WPW syndrome in whom the delta waves disappear with increases in the heart rate are considered at lower risk of SCD. This is because the loss of the delta wave shows that the accessory pathway cannot conduct electrical impulses at a high rate. These individuals will typically not have fast conduction down the accessory pathway during episodes of atrial fibrillation. Risk stratification is best performed via programmed electrical stimulation (PES). High-risk features that may be present during PES include an effective refractory period of the accessory pathway less than 270 ms, multiple pathways, septal location of pathway, and inducibility of supraventricular tachycardia. Individuals with any of these high risk features are generally considered at increased risk for SCD and should be treated accordingly (6).

In resuscitated patients with WPW syndrome who have normal left ventricular function at echocardiography and no ECG abnormalities suggesting additional electrical disease, ablation of their overt accessory pathways prevented cardiac arrest recurrences (7). Response to long-term antiarrhythmic therapy for the prevention of further episodes of tachycardia in patients with WPW syndrome remains quite variable and unpredictable. Some drugs may paradoxically make the reciprocating tachycardia more frequent. Dual-drug therapy has been used, eg, procainamide and verapamil (class IA and IV), or quinidine and propranolol (class IA and II). Class IC drugs are good choices, but class IC drugs should not be given if the patient has structural heart disease. Class IC drugs are typically used with an AV nodal blocking agent. The best plan is to treat symptomatic WPW syndrome patients with ablation to cure the tachycardia and eliminate the potential dangerous effects of drugs. Electrophysiologic study and ablation were planned for our case. Up to now, she is taking the antiarrhythmic treatment of propafenone and β -blocker.

The risk of sudden death is always present with WPW syndrome, and it is the motivating force in the evaluation and treatment of this syndrome.



Figure 1. Resting electrocardiogram (DII) with delta wave and wide QRS complex morphology

Current diagnostic modalities are accurate in identifying patients with WPW syndrome, but lack the sensitivity to predict sudden cardiac death.

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Ruptured abdominal aortic aneurysms: a five-year experience

Rüptüre abdominal aort anevrizmaları: Beş yıllık deneyimimiz

Between March 2001 and December 2006, 16 consecutive patients underwent surgery urgently in our department for ruptured abdominal aortic aneurysm (RAAA). Thirteen of the patients were male 81.25% and three were female 18.75%. Average age was 58.81±10.57 years with a range between 36 to 74 years (Table 1). The time interval between the first symptoms of RAAA and patient's admission to surgical incision was 5.37±2.39 h. The average diameter of RAAA evaluated by ultrasonography during patient's admission to our emergency unit was 7.06±1.73 cm. Ultrasonography and computed tomography (CT) were performed for all the patients (Fig. 1). All procedures were performed transperitoneally and all aneurysms were at infrarenal region (Table 2). Ruptures were predominantly retroperitoneal (75% of cases) and less often intraperitoneal (25% of cases). Vascular reconstruction included interposition of the tube graft 62.5% of cases, aorto-biiliac bypass 25% of cases, aortobifemoral bypass 12.5% of cases. Operative and early postoperative mortality was defined as death within 30 days of surgery.

Early postoperative mortality was seen in 5 patients (31.25%) and operative mortality was seen in one patient (6.25%). The overall operative and early postoperative mortality rate was 37.5%. Main causes of patients' death (Table 3) (n=6) were myocardial infarction in 12.5% of cases, peroperative