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.

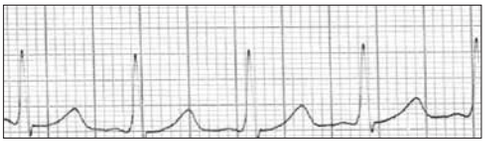

Figure 1. Resting electrocardiogram (DII) with delta wave and wide QRS complex morphology

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, aortobiiliac 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

hemorrhagic shock - 6.5%, pulmonary dysfunction -12.5%, and renal failure - 6.25% of cases. The average duration of hospitalization was 9.37±2.02 days, with an average intensive care unit stay of 3.81±2.25 days.

Patients cannot survive RAAA without operative treatment (1). The mortality rate of RAAA remains high over five decades despite the improvement of operative techniques (including the endovascular techniques in recent years) and perioperative management. The hospital mortality rate is 30-50%, but overall mortality of patients with RAAA is even higher (2).

Certain intraoperative factors have been shown to determine those at high risk of perioperative mortality. Many authors have shown that estimated blood loss and total blood transfusions have a negative impact on survival (3). If hemoperitoneum is present, the chances of patients' survival are very low due to ongoing hemorrhagic shock. We found hemoperitoneum and hemorrhagic shock in two patients. One of them died intraoperatively. Most of these patients have multiple additional risk factors as coronary artery disease and pulmonary dysfunction that effect the results in negative ways. Maximizing myocardial function with adequate preload, controlling oxygen consumption by the reduced heart rate and blood pressure product, ensuring adequate oxygenation, and establishing effective analgesia are important techniques to prevent myocardial ischemia postoperatively (4). Myocardial infarction developed in three (18.75%) of our patients at postoperative period and two of those (12.5%) died. 68.75% of our patients operated for RAAA were smokers. Smoking contributes to the rapid expansion and increases the risk of rupture. Another preoperative factors that have been associated with increased mortality include elevated creatinine and chronic renal failure (5).

We conclude that outcome can be improved by adequate preoperative care, routine use of intraoperative autotransfusion and improvements in postoperative care. Despite an improvement in overall mortality, RAAA remains highly fatal, and elective repair and early recognition are still the essential components in reducing mortality.

Table 1. Preoperative patients' characteristics

Female/male ratio	1/4.3
Average age, years	58.81±10.57
Smokers, %	68.75
Hypertension, %	62.5
Diabetes mellitus, %	25
Hemorrhagic shock, %	12.5
Ischemic heart disease, %	62.5
Diameter of RAAA, %	7.06±1.73
Median time from symptoms to surgery, hours	5.37±2.39
Cerebrovascular disease, %	6.25
Previous coronary artery bypass graft, %	6.25
Preoperative hemoglobin, %	
<7	12.5
7-10	37.5
≥10	50

RAAA - ruptured abdominal aortic aneurysm

Table 2. Operative data

Time of operation, hours	3.25±0.77
Tube grafts, %	62.5
Bifurcated grafts, %	37.5
Aortobiiliac bypass, %	25
Aortobifemoral bypass, %	12.5
Estimated blood loss (ml), %	
<1500	31.25
1500-2500	50
>3000	18.75
Blood transfusion, units	
Blood	4.93±1.56
Thrombocyte suspension	6

Table 3. Postoperative complications

Complication	İncidence		Mortality	
	n	percent	n	percent
Myocardial infarction	3	18.75	2	12.5
Pulmonary dysfunction	4	25	2	12.5
Renal failure	3	18.75	1	6.25
Peroperative hemorrhagic shock	2	12.5	1	6.25
Limb ischemia	1	6.25	-	-
Wound infection	2	12.5	-	-
Bleeding	1	6.25	-	-
Thrombocytopenia	1	6.25	-	-

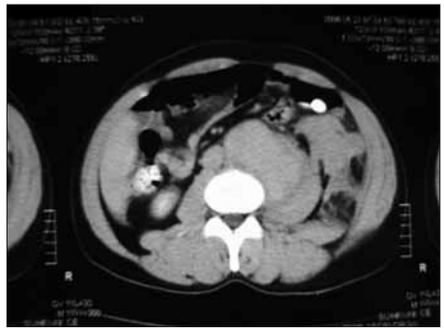

Figure 1. Computed tomographic view of the ruptured abdominal aortic aneurysm

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