OLGU SUNUMU CASE REPORT

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Normalization of Electrocardiography Pattern Due to Anesthesia in a Patient with Wolff-Parkinson-White Syndrome During Non-Cardiac Surgery

Wolff-Parkinson-White Sendromu Olan Hastada Non-Kardiyak Cerrahi Sırasında Anesteziye Bağlı Ritim Normalleşmesi

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ABSTRACT Wolff-Parkinson-White (WPW) syndrome is a form of arrhythmia in which atrioventricular node is bypassed between the atrium and the ventricles by a second electrical communication pathway (accessory), with a short PR interval and a delta wave. During the anesthetic management of these patients the most important goal must be avoiding malignant arrhythmias such as paroxysmal supra ventricular tachycardia (PSVT), ventricular fibrillation (VF) and atrial fibrillation (AF). Inproper anesthetic management like superficial anesthesia, hypovolemia, electrolyte imbalance and anesthetic agents which may cause tachycardia or sympathetic discharge may lead to malignant arrhythmias. In this case report, the electrocardiography normalization and anesthetic management of a patient with WPW syndrome which has presented for non-cardiac surgery will be presented.

Keywords: Wolff-Parkinson-White syndrome; arrhythmias, cardiac; anesthesia

ÖZET Wolff-Parkinson-White (WPW) sendromu, atriyum ve ventrikül arasındaki atriyoventriküler nodun ikincil bir elektriksel bağlantı (aksesuar) yolağı ile by-pass edildiği, kısa PR intervalinin ve aksesuar yolaktaki iletimin göstergesi olarak delta dalgalarının gözlendiği bir aritmi tipidir. Bu hastalarda anestezi yönetimindeki en önemli amaç, paroksismal supraventriküler taşikardi (PSVT), atriyal ve ventriküler fibrilasyon (AF, VF) gibi malign aritmilerden kaçınmaktır. Yüzeyel anestezi gibi uygunsuz anestezi yönetimi, hipovolemi, elektrolit dengesizliği ve taşikardi ya da sempatik deşarja neden olabilecek anestezik ajanlar malign aritmilere yol açabilirler. Bu olgu raporunda WPW sendromu olan hastada non-kardiyak cerrahi sırasındaki anestezi yönetimi ve elektrokardiyografi normalleşmesi sunulacaktır.

Anahtar Kelimeler: Wolff-Parkinson-White sendromu; aritmiler, kardiyak; anestezi

Wolff Parkinson White (WPW) syndrome is a form of arrhythmia in which atrioventricular node is bypassed between the atrium and the ventricles by a second electrical communication pathway (accessory), with a short PR interval and a delta wave which represents the conduction via accessory pathway. The P wave is followed by a widened QRS complex. The phenomenon has been described in 1930 by Wolff-Parkinson and White.[1] Paroxysmal supra ventricular tachycardia (PSVT), ventricular fibrillation (VF) and atrial fibrillation (AF) are the most common arrhythmias in these patients.[2] In the anaesthetic management of these patients the most important point is to prevent situations which may lead to malign arrhythmias. In this case report, the ECG normalization of a patient

with WPW syndrome due to anaesthesia will be presented.

CASE REPORT

A 38-year-old man, 167 cm height and weighing 50 kg with ASA II physical status admitted for elective left hemicolectomy. Preoperative evaluation revealed that the patient has WPW Syndrome and implantable cardiac defibrillator (ICD). There were no other comorbidities. Preoperative electrocardiogram was suggestive of WPW syndrome with heart rate 72 beats/min, PR distance 0.08 sec and delta waves. In echocardiographic evaluation he had ejection fraction 55%, systolic pulmonary arterial pressure 28 mmHg with normal valve structures. Preoperative laboratory evaluations revealed LDH: 349 U/L, amylase: 104 U/L, CRP: 20.7 mg/L, Hb: 9.1 mg/dl, Hct: 28.2, INR: 1.39, t. bilirubin: 3.53 mg/dL, direct bilirubin: 0.50 mg/dL. Other tests revealed normal values.

In the operating theatre following ECG, noninvasive blood pressure (NIBP), peripheral arterial oxygen saturation (SpO_2) monitoring, radial artery cannulation were performed after skin infiltration with local anaesthesia and sedation with intravenous (iv) 2 mg midazolam. Cardiac index trending monitor (ProAQT, PULSION Medical Systems SE, Munich, Germany) was connected for advanced invasive haemodynamic monitoring in case of malignant arrhytmia and fluid therapy guidance. Cardiac output (CO), systemic vascular resistance (SVR), stroke volume variation (SVV), pulse pressure variation (PPV) values monitored continuously via ProAQT and recorded 20 min intervals (Table 1). Anaesthesia was induced with propofol 2 mg/kg, fentanyl 1μg/ kg, and rocuronium 0, 6 mg/kg and patient's trachea was intubated without any difficulty. Anaesthesia was maintained with 0,6-0,8 MAC sevoflurane and remifentanil infusion. Rocuronium was administered according to Train of Four (TOF) monitoring to avoid unnecessary neuromuscular blocker application. Mechanical ventilation was initiated with a tidal volume 6 mL/kg, frequency: 12 breaths/min, %40 oxygen-air mixture. Following two hours of operation the patient was extubated without using neither cholinesterase inhibitor nor sugammadex. Analgesia was provided with iv tenoxicam 20 mg and tramadol hydrochloride 50 mg. In the intraoperative period, disappearance of delta waves following induction of anaesthesia and normalization of ECG were detected following extubation, the regular ECG pattern of the patient was observed.

DISCUSSION

In patients with WPW syndrome, light anaesthesia, hypoxia, ischemia and electrolyte impairment may cause sympathetic discharge and trigger malignant arrhythmias. In these patients, the most prudent goal of anaesthesia should be avoiding sympathetic discharge related to these reasons.[1] In addition, some inhalation and intravenous agents may lead to triggering arrhythmias.[2] The intravenous agents propofol, barbiturates and benzodiazepines are the agents which have no effect on accessory pathway conduction and they can be used safely.[3] Ketamine stimulates indirectly the sympathetic nervous system and inhibits reuptake of norepinephrine following rapid injection. It causes an increase in heart rate, blood pressure and cardiac output, so it must be avoided in WPW syndrome.[3] Some inhalation agents like desflurane may lead to elevation in heart rate, blood pressure and catecholamine levels when it's concentration is increased rapidly.[4] It has been stated that isoflurane and sevoflurane are also safe inhalation agents.[5] Pancuronium due to vagal blocking effect and sympathetic stimulation and histamin releasing agents like atracurium should be also avoided.[4] Antimuscarinic agents atropine and glycopirolate may lead to tachycardia; ephedrine and other sympathomimetic agents should be avoided as well.[2] Opioid agents inhibit the noroendocrine stress response to surgical stimulation and can be used safely.[2] In this patient anesthesia was induced with propofol, fentanyl and rocuronium and all of these agents are considered as safe. Also the maintenance is done with sevoflurane and remifentanil which have no effect on AV conduction and supresses the neuroendocrine stress response and cause hemodynamic stability.

TABLE 1: Intraoperative haemodynamic changes and other parameters.

Time	HR (beats/min)	BP (mmHg)	CO (l/min)	SVR (dyne•sn cm-5)	SVV (%)	PPV (%)	SV (ml/beats)	BIS (%)	TOF (%)	MAC
0.min	58	120/65	4,24	1540	7	8	70	52	%11	0,5
20.min	57	111/61	4.06	1520	3	4	71	47	%46	0,7
40.min	58	113/69	4,10	1630	5	5	69	42	%73	0,8
60.min	54	107/62	3,5	1650	5	5	65	41	%8	0,7
80.min	53	97/55	3,45	1600	4	4	66	41	%31	0,8
100.min	54	125/65	3,93	1690	7	7	72	43	%55	0,8
120.min	54	116/60	4,21	1450	5	3	75	55	%55	0,8

HR: Heart rate; BP: Blood pressure; CO: Cardiac output; SVR: Systemic vascular resistance; SVV: Stroke volume variation; PPV: Pulse pressure variation; SV: Stroke volume; BIS: Bispectral index; TOF: Train of Four monitor; MAC: Minimum alveolar concentration.

Neostigmine is a dangerous agent in such patients because it prolongs the AV nodal conduction and activates the conduction in accessory pathway and may lead to atrial fibrillation with rapid ventricular rate.[6] Also antimuscarinic agent applied during the reversal of neuromuscular blockade to avoid muscarinic effects of neostigmine may lead to tachycardia. Sugammadex is another option in this patients and has been used in some cases safely but there have been case reports which caused persistent bradycardia and arrthymia.[7,8] So we avoided both of the agents by TOF monitoring, following a TOF value of 0.9 the patient was extubated safely and no recurarization was observed in the post anesthesia care unit (PACU).

The electrolyte imbalance and hypoxia has been prevented by analysis of arterial blood gas analyses and volume replacement was done according to SVV and PPV; CO values by avoiding hypovolemia and volume overloading.

It has been stated that propofol or inhalation agents have no effect on electrophysiological effects of the accessory pathway. But there have been cases in the literature in which normalization of ECG and disapperiance of delta vawes have been observed following propofol infusion.[9,10] The disapperiance of delta waves and normalization of QRS shows the depression of accessory pathway and in this patient this may be related to propofol, opioids or sevoflurane anesthesia. Also anesthesia may lead to supression of sympathetic discharge and normalization of ECG pattern. Further electrophysiological assessments are needed to evaluate these effects.

In conclusion, our case report shows a proper anaesthetic management may lead to normalization of ECG pattern although the patient has a abnormal ECG pattern preoperatively. Also current knowledge about the effects of anaesthetic agents on the accessory pathway in these patients needs further investigation.

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Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

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