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Original Research

Bedside Temporary Transvenous Pacemaker Insertion in the Emergency Department: A Single-Center Experience

Bihter Senturk,[1] Servan Kucuk,[2] Sevilay Vural,[3] Erdal Demirtas,[2] Figen Coskun[2]

[1]Department of Cardiology, Faculty of Medicine, Dokuz Eylul University, Izmir, Turkey
[2]Department of Emergency Medicine, Faculty of Medicine, Dokuz Eylul University, Izmir, Turkey
[3]Department of Emergency Medicine, Faculty of Medicine, Yozgat Bozok University, Yozgat, Turkey

Abstract

Objectives: Insertion of a temporary transvenous pacemaker (TTPM) is one of the life-saving interventions performed in the emergency department (ED). The aim of the study was to determine demographic, clinical characteristics, and in-hospital outcomes of patients who underwent TTPM insertion due to hemodynamically unstable bradyarrhythmia in the ED.

Methods: In our study, 234 consecutive patients who underwent TTPM insertion at the bedside in the ED between January 2014 and October 2019 were included in the study. Etiological characteristics, electrocardiographic (ECG) findings, requirements for permanent pacemaker (PPM), and in-hospital mortality of the patients were analyzed retrospectively.

Results: Extrinsic causes were the most common etiology of unstable bradyarrhythmia (57.6%). Most extrinsic causes were drug therapy-related factors (60.7%). Bradyarrhythmia persisted in 60% of patients after extrinsic causes were eliminated. The most common ECG finding was a high-degree atrioventricular block (62%). PPM was implanted in 44% of patients. In-hospital mortality rate was 19.7%. In the multivariate regression analysis, the left ventricular ejection fraction (LVEF) and diastolic blood pressure (DBP) measured at admission ($p<0.001$ and $p<0.001$, respectively) were determined to be independent predictors for in-hospital mortality.

Conclusion: First diagnosis and intervention in the ED are of great importance for patients with unstable bradyarrhythmia. The fastest possible TTPM insertion in the ED can reduce mortality by reducing the exposure time to hypoperfusion of vital organs, especially in patients with reduced LVEF and low DBP. Furthermore, it should be kept in mind that an underlying latent conduction system disease can also be present in bradyarrhythmias thought to occur potentially due to extrinsic factors.

Keywords: Temporary pacemaker; emergency; bedside; bradyarrhythmia.

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Introduction

Insertion of temporary transvenous pacemaker (TTPM) is an essential procedure for patients with impaired hemodynamics, life-threatening bradyarrhythmias and is one of the most common interventions performed in the emergency department (ED).[1] In patients with bradyarrhythmia, a low heart rate gives rise to decreased cardiac output and hypoperfusion of vital organs, which can lead to dizziness, shortness of breath, angina, syncope, acute heart failure, unstable hemodynamic status, and even sudden cardiac

Address for correspondence: Bihter Senturk, MD. Dokuz Eylul University, Faculty of Medicine Department of Cardiology, 35340, Izmir, Turkey
Phone: +90 232 412 41 30 **E-mail:** drbihter@hotmail.com

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death.[2] Restoring the cardiac depolarization and ensuring effective myocardial contraction by inserting a TTPM provide sufficient cardiac output, and thus end-organ perfusion is maintained.[3,4] Heterogeneous conditions can cause unstable bradyarrhythmias requiring TTPM insertion, such as idiopathic and degenerative diseases of the conduction system,[5] drug overuse and adverse effects,[6-9] electrolyte imbalance,[8,10] and acute myocardial infarction (AMI).[11] The aim of the study was to determine demographic, etiologic, and electrocardiographic (ECG) characteristics of patients who underwent TTPM insertion in the ED and to identify predictors of in-hospital mortality.

Methods

The Non-invasive Investigation Ethics Committee of Dokuz Eylul University approved this study (date: 13.04.2020, approval number: 2020/07-27). In our study, 234 consecutive patients between January 2014 and October 2019 with hemodynamically unstable bradyarrhythmia treated with insertion of TTPM at the bedside in the ED without using fluoroscopy were included. The patients' data were analyzed retrospectively, the characteristics of the patients, etiologies, ECG findings, echocardiographic, and laboratory data were recorded. In addition, complications during the procedure, whether permanent pacemaker (PPM) was implanted, and in-hospital mortality were noted.

ECG Findings of Patients

ECG findings of patients were classified as atrioventricular conduction dysfunction (AVCD) and sinoatrial node dysfunction (SAND). AVCD was divided into a high-degree atrioventricular block (HAVB) and atrial fibrillation/flutter with a slow ventricular rate (AF/AFL with SVR). HAVB was comprised of complete AVB (3rd degree AVB) and 2nd degree AVB. SAND consisted of sinus pause and sinus bradycardia.

Etiology of Bradyarrhythmia

Etiologies of bradyarrhythmia were classified as intrinsic and extrinsic.[12] Intrinsic factors involved idiopathic or degenerative conduction system disease (CSD) and AMI. Extrinsic factors involved drug therapy-related factors and metabolic abnormality-related factors, that is, isolated electrolyte imbalance or combination of electrolyte imbalance and drug therapy. Situations with hyperkalemia medication treatment were defined as potassium >5.5 mol/L, while isolated hyperkalemia was defined as potassium >6 mmol/L.[13,14]

Reversibility of Bradyarrhythmia

Reversibility of bradyarrhythmia was defined as resolution of bradyarrhythmia following drug discontinuation, treatment of potentially reversible causes (metabolic abnormality and acute myocardial ischemia), and absence of bradyarrhythmia recurrence.

Statistical Analysis

All statistical procedures were performed with SPSS software (version 25.0, SPSS Inc., Chicago, IL, institutional software). Normality was assessed with the Kolmogorov–Smirnov test. Continuous variables are expressed as mean±SD or median (25th–75th percentile). Categorical variables are presented as number and percentage. The comparison between groups was performed using the independent samples *t*-test for normally distributed continuous variables, the Mann–Whitney *U*-test for non-normally distributed continuous variables, and the Chi-square test for categorical variables. Logistic regression analyses were conducted to define predictors of mortality. Variables with $p<0.1$ on univariate analysis were included in logistic regression analysis. $P\leq0.05$ was considered statistically significant.

Results

Patient Characteristics

Two hundred and thirty-four patients were included in this study. About 85% of patients were the elderly (≥65 years). The median age of patients was 78.5. About 56% of the patients were women. Most common comorbidities were hypertension (HT) (74.8%), diabetes mellitus (DM) (37.2%), and coronary artery disease (CAD) (30.8%), respectively (Table 1).

ECG Findings of Patients

AVCD was involved in the majority of the cases (73.1%) followed by SAND (26.9%). Among AVCDs, HAVB was the most common ECG finding (84.8%) followed by AF/AFL with SVR (15.2%). Complete AVB accounted for 86.9% of HAVB. Among SANDs, sinus pause (79.3%) represented the majority followed by sinus bradycardia (20.6%) (Table 1).

Etiology of Bradyarrhythmia

Extrinsic factors were the most common etiology of bradyarrhythmia (57.6%). The majority of extrinsic causes were drug therapy-related factors (60.7%) (including beta-blocker [BB], non-dihydropyridine calcium channel blocker [CCB], digoxin, and amiodarone) followed by metabolic abnormality-related factors (39.3%). The only electrolyte imbalance determined was hyperkalemia. Hyperkalemia with medication therapy combinations comprised 21.5% of extrinsic factors, while isolated hyperkalemia comprised 17.8% of extrinsic factors.

Among drug-induced bradyarrhythmias (DIB), BBs were the most common drugs used (41.5%), followed by CCBs (19.5%), digoxin (9.8%), BB+digoxin (7.3%), BB+CCB (4.9%),

Table 1. Demographic, clinical, and laboratory characteristics of patients (*n*=234)

Age (years)[a]	**78.5 (69.7–84)**
Gender (female), *n* (%)	131 (56%)
Hypertension, *n* (%)	175 (74.8%)
Diabetes mellitus, *n* (%)	87 (37.2%)
Coronary artery disease, *n* (%)	72 (30.8%)
Systolic blood pressure (mmHg)[a]	107 (80–132)
Diastolic blood pressure (mmHg)[a]	65 (49–82)
Etiology of bradyarrhythmias	
I. Extrinsic	135 (57.6%)
a. Drug therapy-related factor	82 (60.7%)
b. Metabolic abnormality-related factor	53 (39.3%)
– Combination of electrolyte imbalance and drug therapy	29 (21.5%)
– Isolated electrolyte imbalance	24 (17.8%)
II. Intrinsic	99 (42.3%)
a. Acute myocardial infarction	50 (50.5%)
b. Idiopathic or degenerative conduction system disease	49 (49.4%)
ECG findings of patients, *n* (%)	
I. AVCD	171 (73.1%)
a. HAVB	145 (84.8%)
– Complete AVB	126 (86.9%)
– 2nd degree AVB	19 (13.1%)
b. AF/AFL: With a slow ventricular rate	26 (15.2 %)
II. SAND	63 (26.9 %)
a. Sinus pause	50 (79.3%)
b. Sinus bradycardia	13 (20.6%)
LVEF (%)	50.8±8.9[b]
Sodium (mmol/l)[a]	137 (134–140)
Potassium (mmol/l)[a]	4.6 (4.1–5.4)
Creatinine (mg/dl)[a]	1.37 (0.98–2.0)
Hemoglobin (g/dl)[a]	12.1 (10.7–13.7)
WBC (10^3/uL)[a]	10.4 (8–13.5)
Platelet (10^3/uL)[a]	205 (166–260)

[a]Median (25th–75th percentile); [b]Mean±standard deviation. AVCD: Atrioventricular conduction dysfunction; SAND: Sinoatrial node dysfunction; HAVB: High-degree atrioventricular block; AVB: Atrioventricular block; AF/AFL: Atrial fibrillation/atrial flutter; LVEF: Left ventricular ejection; WBC: White blood cell.

CCB+digoxin (4.9%), BB+amiodarone (4.9%), amiodarone (3.7%), CCB+amiodarone (2.4%), and BB+CCB+digoxin (1.2%). Among intrinsic etiologies, AMI and CSD had similar frequency (50.5% and 49.4%, respectively) (Table 1). AMI included ST-segment elevation myocardial infarction (STEMI) and acute coronary syndromes without persistent ST segment elevations (NSTE-ACS). Primary percutaneous coronary intervention (pPCI) was performed on all STEMI patients.

About 72% of AMI patients had inferior STEMI, 26% had anterior MI, and 2% had NSTE-ACS. In all the inferior STEMI cases, the culprit lesion was located in the right coronary artery (RCA); in all of the anterior STEMI cases, the culprit lesion was located in the left anterior descending artery (LAD); and in a single patient with NSTE-ACS, the culprit lesion was located in the left main coronary artery, and surgical revascularization was planned. The success rate of primary PCI procedures was 80% (86.1% for inferior STEMI and 69.2% for anterior STEMI). Bradyarrhythmias resolved in 95% of patients treated with successful pPCI for STEMI.

Complications

No deaths occurred as a direct result of TTPM insertion. Lead dislocation requiring lead revision with fluoroscopy was observed in 11.1% of patients. Tamponade resolved by pericardiocentesis occurred in 1.7% of patients. Pneumothorax that did not require insertion of a chest tube developed in 0.8% of patients. None of the patients developed complications requiring urgent surgery.

Reversibility of Bradyarrhythmia and PPM Implantation

Bradyarrhythmia was reversible in 39.3% of patients. The incidence of reversible bradyarrhythmia was 76% in AMI-associated bradyarrhythmias (AAB), 45.3% in metabolic abnormality-induced bradyarrhythmias (MAIB), and 36.6% in DIB. No reversibility was observed in CSD-associated bradyarrhythmia (CAB) (Table 2). About 27% of patients with irreversible bradyarrhythmia died before PPM could be implanted. Eventually, PPM was implanted in 44% of all patients. PPM was implanted in all patients with CAB, 55% of those with DIB, and 9% of those with MAIB. None of the patients with irreversible AAB had PPM implanted because they all died.

The type of PPM was decided according to the guideline.[15] PPM without defibrillator feature was implanted in patients with the left ventricular ejection fraction (LVEF) >35% and intracardiac defibrillator (ICD) or cardiac resynchronization therapy-ICD was implanted in those with LVEF ≤35%. Types of PPM implanted were DDD-PPM (67.9%), VVI-PPM (23.3 %), DDD-ICD (3.9%), VVI-ICD (0.9%), and CRT-ICD (3.9%), respectively (Table 2).

In-hospital Mortality

In-hospital mortality rate was 19.7%. Approximately 11% of deaths occurred after PPM implantation. Mortality rates

Table 2. In-hospital outcomes of patients

The rate of reversibility for all bradyarrhythmias	**92 (39.3%)**
The rate of reversibility in bradyarrhythmias with different etiologies	
Acute myocardial infarction	38 (76%)
Metabolic abnormality-related factors	24 (45.3%)
Drug therapy-related factors	30 (36.6%)
Idiopathic or degenerative conduction system disease	0
Rate of PPM implantation, *n* (%)	103 (44%)
Types of PPM, *n* (%)	
DDD-PPM	70 (67.9%)
VVI-PPM	24 (23.3%)
DDD-ICD	4 (3.9%)
VVI-ICD	1 (0.9%)
CRT-ICD	4 (3.9%)
Rate of in-hospital mortality, *n* (%)	46 (19.7%)
Mortality rates according to different etiological factors, *n* (%)	
Metabolic abnormality-related factors	23 (43.4%)
Acute myocardial infarction	14 (28%)
Drug therapy-related factors	8 (9.8%)
Degenerative or idiopathic conduction system disease	1 (2%)

PPM: Permanent pacemaker; ICD: Intracardiac defibrillator; CRT: Cardiac resynchronization therapy.

according to different etiologies are presented in Table 2. Mortality rate was 43.4% for MAIB, 28% for AAB, 9.8% for DIB, and 2% for CAB. The mortality rate in patients with inferior STEMI was 16.6% and in patients with anterior STEMI was 53.8%. A single patient with NSTE-ACS with a left main coronary artery lesion died before revascularization.

The most common etiologies were extrinsic causes in both survivors and non-survivors (55.3% and 67.4%, respectively, p=0.137), and the most common ECG finding was AVCD in both groups (72.9% and 73.9%, respectively, p=1.00) (Table 3).

No significant difference was found between the age and gender of the survivor and the non-survivor groups. The frequencies of HT, DM, CAD, and AMI were similar in non-survivors and survivors. LVEF, hemoglobin level, baseline systolic, and diastolic blood pressure (DBP) were lower in the non-survivor group (p<0.001, p=0.002, p<0.001, and p<0.001, respectively) (Table 3). In the multivariate logistic regression analysis, LVEF and DBP measured at admission to ED (p<0.001 and p<0.001, respectively) were determined to be independent predictors of in-hospital mortality (Table 4).

Discussion

Bradyarrhythmias requiring TTPM are caused by intrinsic and extrinsic factors. While idiopathic or degenerative CSD was in first place among the etiology of bradyarrhythmia in other studies,[16,17] extrinsic factors were the most common etiology in this study. Most extrinsic causes comprised factors related to drug therapy. Since patients were the elderly and had multiple comorbidities, it was not surprising that drug therapy was frequent in our patient group. This is the most important reason for the inclusion of extrinsic etiologies in the first place in our study. Although the frequency of etiologic factors was different, the incidence of PPM implantation was like in this study.[16,17] Bradyarrhythmia persisted in 60% of patients with extrinsic etiology, although the drugs responsible were discontinued, and hyperkalemia was corrected. In a study examining DIB, it was found that bradyarrhythmia was truly caused by drugs in only 52% of patients, and about half of patients with DIB needed a PPM.[9] Similarly, PPM was implanted in 55% of patients with DIB in this study. It was suggested that AVCD usually does not take place without structural heart disease, even if it was triggered by drugs.[7] This situation suggests the presence of a latent CSD in extrinsic etiology. The advanced age of patients also supports the existence of underlying degenerative CSD in extrinsic etiology. Hyperkalemia can also induce bradyarrhythmias in patients with latent CSD.[18] Furthermore, in this study, about 70% of the patients with hyperkalemia had chronic kidney disease (CKD). CKD can lead to fibrosis and calcification of the cardiac conduction system by disrupting calcium metabolism and cause degenerative CSD.[19] Therefore, bradyarrhythmia can persist, although hyperkalemia was corrected. In addition, in about a fifth of patients with extrinsic etiology, hyperkalemia was accompanied by drug therapy. Situations, where hyperkalemia with the use of AV node blocker medications causes bradyarrhythmia is a new clinical entity called BRASH syndrome.[14] In BRASH syndrome, generally, bradyarrhythmia may develop with lower potassium levels,[13] and bradyarrhythmia is proposed to be due to the synergistic effect between hyperkalemia and medications.[14] This study noted that nearly 45% of patients with hyperkalemia accompanying drug treatment had potassium levels between 5.5 and 6.0. For this reason, the limit for hyperkalemia combined with drug therapy was lowered.

HAVB was determined to be the most common ECG finding in patients with TTPM.[17,20,21] In addition, HAVB was

Table 3. Comparison of survivors and non-survivors

	Survivor (*n*=188)	Non-survivors (*n*=46)	*P*-value
Age (years)[a]	78 (69–84)	79.5 (67.7–85)	0.851
Gender (female), *n* (%)	105 (55.9%)	26 (56.5%)	0.935
Hypertension, *n* (%)	142 (75.5%)	33 (71.7%)	0.733
Diabetes mellitus, *n* (%)	72 (38.3%)	15 (32.6%)	0.474
Coronary artery disease, *n* (%)	55 (29.3%)	17 (37%)	0.403
Acute myocardial infarction, *n* (%)	36 (19.1%)	14 (30.4%)	0.141
Etiologies of bradyarrhythmias, *n* (%)			
Extrinsic	104 (55.3%)	31 (67.4%)	0.137
Intrinsic	84 (44.7%)	15 (32.6%)	
ECG findings of patients, n (%)			
AVCD	137 (72.9%)	34 (73.9%)	1.000
SAND	51 (27.1%)	12 (26.1%)	
Systolic blood pressure (mmHg)[a]	116 (90–137)	86 (60–110)	**<0.001***
Diastolic blood pressure (mmHg)[a]	70 (52–82)	50 (32–65)	**<0.001***
LVEF (%)[b]	53.2±6.7	40.8±9.8	**<0.001***
Creatinine (mg/dl)[a]	1.28 (0.97–1.92)	1.66 (1.04–2.18)	0.066
Sodium (mmol/l)[a]	137 (134-139)	137.0 (132–140)	0.660
Potassium (mmol/l)[a]	4.65 (4.1–5.21)	4.77 (4.1–5.74)	0.466
Hemoglobin (mg/dl)[a]	12.3 (10.9–13.8)	11.4(9.8–12.9)	**0.002***
WBC (10^3/uL)[a]	10.3 (7.8–13.1)	11.2 (8.2–15.0)	0.206
Platelet (10^3/uL)[a]	207 (171–257)	196 (149–276)	0.486

[a]Median (25th–75th percentile); [b]Mean±standard deviation. **p*<0.05. ECG: Electrocardiography; TPM: Temporary pacemaker; AVCD: Atrioventricular conduction dysfunction; SAND: Sinoatrial node dysfunction; LVEF: Left ventricular ejection fraction; WBC: White blood cell.

Table 4. Multivariate logistic regression analysis for the predictors of in-hospital mortality

Variables	Univariate OR (95% CI)	*P*-value	Multivariate OR (95% CI)	*P*-value
Systolic blood pressure	0.976 (0.965–0.987)	**<0.001***	0.986 (0.959–1.013)	0.297
Diastolic blood pressure	0.960 (0.942–0.977)	**<0.001***	0.954 (0.931–0.977)	**<0.001***
LVEF	0.841 (0.801–0.883)	**<0.001***	0.837 (0.794–0.883)	**<0.001***
Creatinine	1.120 (0.885–1.417)	0.346	—	—
Hemoglobin	0.785 (0.645-0.915)	**0.002***	0.906 (0.745-1.103)	0.327

**p*<0.05. OR: Odds ratio; CI: Confidence interval; LVEF: Left ventricular ejection fraction.

found to be the most common type of bradyarrhythmia in patients admitted to the ED due to hemodynamic impairment.[22] In our study, in accordance with these studies, 62% of the bradyarrhythmias were HAVB. AVCD was observed with a similar frequency in the survivors and non-survivors. In a study investigating the risk factors for mortality in patients with TTPM, like our study, no relationship was found between ECG findings and in-hospital mortality.[21]

The frequency and characteristics of unstable bradyarrhythmia in patients with AMI may have changed due to advances in PCI techniques and treatment strategies. Despite improvements in treatment strategies, AMI can still be complicated by HAVB in patients.[23] AMI did not reach first place in the etiology of bradyarrhythmia requiring TTPM.[16,17] AMI was in third place in this study. However, only patients who underwent TTPM insertion in the ED were included in our study. It should be noted that most

of the patients with AMI usually undergo TTPM insertion during the pPCI procedure in the catheter laboratory. Patients with inferior STEMI are at higher risk of HAVB; patients with inferior STEMI have 2- to 4-fold increased risk compared to those with anterior STEMI.[24,25] In accordance with this data, most AMI in our study was inferior AMI. Bradyarrhythmia, in general, occurs as a result of interruption of the perfusion of the AV nodal artery that usually originates from the RCA in inferior STEMI,[26] while generally it usually caused by impaired perfusion of the His-Purkinje system due to occlusion of the septal branches of the LAD in anterior STEMI, an indicator of poor prognosis. The mortality rate is high in anterior STEMI as a result of increased myocardial damage, larger infarct size, and decreased LVEF.[26,27] Concordant with the previous studies,[11,23] in this study, the mortality rate for anterior STEMI was more than 3 times that of inferior STEMI. The need for PPM implantation was significantly different between AMI and non-AMI groups.[20] Bradyarrhythmia is mostly reversible in patients with AMI.[23] In our study, none of the AMI patients with persistent bradyarrhythmia had PPM implanted because these patients died due to MI or its complications, while 56% of the patients in the non-AMI group had PPM implanted.

Complications associated with TTPM insertion are not uncommon.[28] Therefore, TTPM insertion is performed in patients with bradyarrhythmia only if symptoms or signs of hemodynamic impairment are present in our center. TTP placement was safe with relatively low complication rates, although it was performed at the bedside without the usage of fluoroscopy. The low complication rate may be due to the experienced cardiologists performing or supervising the procedures[17,29] and the use of internal jugular vein access in all patients.[30]

In-hospital mortality rates of the patients in this study were found to be higher compared to similar studies.[20,31] This situation could be explained by the high number of referrals of elderly patients with multiple comorbidities to our center since it is a tertiary center. The relationship between reduced LVEF and mortality is known in patients who require pacemakers.[32] LVEF was found to be an independent predictor of mortality in our study. Low DBP values are associated with poor prognosis in cardiovascular diseases. Coronary perfusion takes place predominantly in diastole; for this reason, low DBP values can reduce coronary blood flow. It was shown that low DBP values were associated with subclinical myocardial ischemia and major adverse cardiovascular outcomes.[33] Furthermore, Axler[34] reported that low DBP value was the predictor of mortality in patients with cardiogenic shock. In this study, DBP values measured at the time of admission to ED were identified as an independent predictor of in-hospital mortality in patients with bradyarrhythmia requiring TTPM.

Limitations of the Study

This study has several limitations. The main limitations of our study are its retrospective design, the relatively small number of patients and patients being recruited from a single center. TPM procedures performed within the specified date range were found through the hospital records. Because the procedure was performed urgently and at the bedside, there may be other cases not reported in hospital records. Furthermore, since our center is a tertiary reference center, patients may be more complex than those encountered in real clinical practice. Therefore, multicenter prospective studies with larger numbers of patients are needed.

Conclusion

We determined several important results in our study. First diagnosis and intervention in the ED are of great importance for patients with unstable bradyarrhythmia. The fastest possible TTPM insertion in the ED can reduce mortality by reducing the exposure time to hypoperfusion of vital organs, especially in patients with reduced LVEF and low DBP at the time of admission to the ED. Furthermore, it should be kept in mind that an underlying latent conduction system disease can also be present in bradyarrhythmias thought to occur potentially due to extrinsic factors.

Disclosures

Ethics Committee Approval: The research protocol was approved by the Non-invasive Investigation Ethics Committee of Dokuz Eylul University (date: 13.04.2020, approval number: 2020/07-27).

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

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