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Perioperative Type I Aortic Dissection during Conventional Coronary Artery Bypass Surgery: Risk Factors and Management

Bulend Ketenci, Yavuz Enc, Batuhan Ozay, Rafet Gunay, Serdar Cimen, Alper Gorur, Abdullah Kemal Tuygun, Murat Sargin, Sibel Sari, Mahmut Murat Demirtas

Siyami Ersek Thoracic and Cardiovascular Surgery Centre, Istanbul, Turkey

ABSTRACT

Objectives: Perioperative iatrogenic type I aortic dissection (PIAD) is a rare but potentially fatal complication of conventional coronary artery bypass surgery (CCABG). Prompt recognition and repair of PIAD may significantly improve outcomes.

Methods: We reviewed the hospital records of patients with PIAD occurring as a complication of CCABG at Siyami Ersek Thoracic and Cardiovascular Surgery Center from January 2001 through June 2007. During this period, 10,130 CCABG were performed and 21 patients (0.20%) with PIAD were identified. We compared variables for these 21 patients with 603 patients without PIAD (control group).

Results: PIAD occurred intraoperatively in 19 patients (90%) and during the early postoperative period (first 6 hours) in 2 patients (10%) who underwent CCABG. Dissections were noticed after removal of the aortic cross-clamp in 11 patients, during aortic cannulation in 3 patients, and after removal of the partial-occlusion clamp in 5 patients. Patients with and without PIAD differed significantly in regard to sex ($P = .05$), history of hypertension ($P = .001$), and history of severe concomitant peripheral arterial disease (PAD) ($P = .001$). The diameter of the aorta was significantly wider in patients with PIAD. (3.83 ± 0.9 vs 2.93 ± 0.46 cm, $P = .019$). The occurrence of high cardiopulmonary bypass (CPB) pressure (≥120 mmHg) was significantly higher in the PIAD patients than the non-PIAD patients (28.6% vs 3.3%, $P = .0001$). Seven PIAD patients (33.3%) died preoperatively and 3 (14.2%) died postoperatively.

Conclusion: PIAD is frequently fatal. Risk factors for PIAD during or after CCABG include female sex, history of PAD and hypertension, increased aortic diameter, and high CPB pressure.

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Correspondence: Bulend Ketenci, Gazi Muhtar Pasa Korusu Mazhar Osman Sk. 25/4 Feneryolu, Kadıköy 34724, Istanbul Turkey; +0905053762401; fax: 902164146276 (bulendketenci@gmail.com).

INTRODUCTION

Perioperative iatrogenic type I aortic dissection (PIAD) is a rare but potentially fatal complication of cardiac surgery, with a reported frequency of 0.12%-0.16% [Blakeman 1988; Still 1992]. In conventional coronary artery bypass grafting (CCABG), the ascending aorta is subjected to manipulations such as cannulation, cross-clamping, partial clamping, and proximal anastamoses of grafts. Each manipulation could cause disruption of the intima of the ascending aorta, resulting in pseudoaneurysm or acute dissection of the ascending aorta. Cannulating the ascending aorta for cardiopulmonary bypass (CPB) and for the delivery of antegrade cardioplegia often disrupts the 3-layered aorta through manipulation and flow. PIAD can occur during the perioperative period or even after patient discharge [Murphy 1983]. Despite the potentially catastrophic nature of PIAD, its associated predisposing risk factors, clinical presentation, and outcomes have not been systematically studied. Predisposing factors for aortic dissection include aortic valve pathology [Larson 1984; Epperlein 1994], arterial hypertension, and Marfan syndrome [Robicek 1994]. Potential locations of injury during surgery include sites with cross-clamp injury [Litchford 1976], proximal anastomosis lines (suture lines from CABG) [Archer 1986], and the cannulation site [Williams 1974]. In short, especially in the presence of concomitant vessel-wall abnormalities, manipulation of the ascending aorta, such as that which occurs with cardiopulmonary bypass (CPB), can cause subsequent PIAD [Chavanon 2001]. The most important concern in management of this frequently fatal complication is prevention by modification of perioperative risk factors.

The purpose of this study was to review and identify the factors associated with the risk of PIAD during on-pump CCABG and to improve strategies for PIAD management and prevention.

MATERIALS AND METHODS

Definition

In this retrospective study, PIAD was considered to be present in any case of dissection involving any portion of the aorta between the aortic annulus and the take-off of the innominate artery. We analyzed cases of PIAD in 10,130 patients CCABG patients who underwent surgery

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in our institution during the period from January 2001 through June 2007. The study protocol was approved by the institutional review board (ethics committee) and was consistent with the principles of the Declaration of Helsinki.

Of 10,130 patients who underwent CCABG at our center during this period, 21 suffered perioperative or early postoperative (first 6 hours after CCABG) PIAD and underwent PIAD repair involving the ascending aorta or aortic arch. We compared variables for these patients with those of a control group of 603 CCABG patients who did not suffer PIAD. Among 10,130 CCABG patients, patients chosen for the control group were those on whom proximal anastomosis was performed by use of lateral clamping and in whom aortic venting had been used. The study and control patients were matched according to age and number of proximal anastomoses. Patients in whom the maximal arterial pressure was as high as 120 mmHg during CPB were considered hypertensive during CPB. The study and control group characteristics were standardized.

For all 624 study patients, we collected data on preoperative, perioperative, and postoperative variables and used multivariate regression analysis to evaluate the potential effects of these variables and perioperative risk factors.

Surgical Technique

The usual technique for CCABG surgery was used for all CPB procedures, which were performed while the patient was under moderate hypothermia (28°C to 32°C). Proximal anastomoses were performed after distal anastomoses under lateral aortic clamp. We performed proximal anastomoses, using 6-0 polypropylene, while the patient's heart was beating. With a Beck clamp placed on aorta for anastomoses, extracorporeal circulation (ECC) flow was decreased to 500 mL/min. The flow is also decreased when the surgeon is placing and removing the lateral clamp. Similarly, the systemic pressure is kept below 100 mmHg during this time.

When PIAD occurred, an aortic cannula was placed in the femoral artery and the patients underwent total circulatory arrest (TCA). During, TCA retrograde a cerebral perfusion technique was used for cerebral protection in 11 patients.

Data Analysis

The Number Cruncher Statistical System (NCSS) 2007 statistical software package was utilized for statistical analysis. Data are given as mean ± SD, and categorical data were reported as percentages. Differences between e groups were analyzed by unpaired Student *t*-test or Fisher exact test when appropriate. A χ^2 or Pearson χ^2 test was used for comparison of categorical variables between the groups. A logistic regression test was used to determine the influential factors of dissection. Significant differences were considered to exist for $P < .05$. A confidence level of 95% was required to consider correlations and statistical values to be significant.

RESULTS

Of 10,130 patients who underwent CCABG at our center during the 6.5-year study period, 21 consecutive patients (group I) underwent PIAD repair involving the ascending aorta or aortic arch. The demographic characteristics of these patients are summarized in Table 1. PIAD occurred intraoperatively in 19 patients (90%) and during the early postoperative period (first 6 hours) in 2 patients (10%).

Among the PIAD patients (group I), 10 (47.65%) were female, and the mean age was 62.1 ± 8 years. Six of the 21 patients (28.6%) were diabetic, and 14 (66%) were hypertensive. In addition, 11 patients (52.4%) also had severe coexisting peripheral arterial disease (PAD). In the non-PIAD patients (group II, n = 603), 21.4% were female, the mean age was 59.6 ± 7.6 years, 18.2% were diabetic, and 28.4% of all patients were hypertensive. The incidence of severe concomitant PAD was only 3% in this group. Ejection fraction did not differ significantly between groups (52.31% ± 7.80% in group I vs 52.73 ± 10.78% in group II) <<Query 2>>. We did find significant differences between the 2 groups, however, regarding patient sex ($P = .05$), history of hypertension ($P = .001$), and history of severe concomitant PAD ($P = .001$). These 3 preoperative risk factors were significantly higher in patients in whom PIAD occurred (Table 1).

Data for perioperative variables are summarized in Table 2. The diameter of the aorta, measured by preoperative echocardiography in all patients, was significantly larger in patients with PIAD (3.83 ± 0.9 cm in group I vs 2.93 ± 0.46 cm in group II; $P = .019$). When we compared the perioperative hemodynamic parameters of the patients in both groups, we found that the occurrence of high (≥120 mmHg) CPB pressure was significantly higher in PIAD patients (group I) than in group II (28.6% vs 3.3%, respectively, $P = .0001$).

PIAD sites are summarized in Table 3. Dissection originated at the aortic cannula in 5 patients, the cross-clamp site in 5 patients, the partial-occlusion clamp in 4 patients, the proximal anastomosis in 3 patients, and the aortic vent in 4 patients. PIAD were first observed after removal of the aortic cross-clamp in 11 patients, during aortic cannulation in 3 patients, and after removal of the partial-occlusion clamp in 5 patients. The mean time from onset of dissection until reestablishment of normal perfusion was 19 ± 3.5 minutes. In 2 patients who developed PIAD in the postoperative period, the diagnosis of PIAD was determined by transthoracic echocardiography performed when malperfusion (pulselessness in the lower extremities) was seen. The mean time from onset of dissection until reestablishment of normal perfusion was not included in analyses because the onset time of the dissection could not be determined with certainty.

Postoperative characteristics of the patients are summarized in Table 4. Seven patients died perioperatively (33.3%) and 3 patients died during the postoperative period (14.2%). Overall mortality from PIAD was 47.6%. Five of 7 patients who died perioperatively could not be weaned from CPB owing to cardiac failure, and 2 of them died perioperatively owing to severe bleeding. Of the 2 patients who died during the postoperative period, 1 died from neurological complications and 1 from intractable fibrillation. Six of 14 patients (28.5%)

Table 1. PIAD Patient Characteristics*

Patient No.	Age, y/Sex	Surgical Procedure	Dissection Origin	Type of Repair	Repair Cannulation Site	Complication	Results
1	51/F	CABG	Partial occlusion clamp	Supracoronary graft	CFA/RA	Bleeding/ARF/ hemiplegia	Death, day 19
2	57/F	CABG	Vent area	Elephant trunk	CFA/RA	Death	Death, perioperative
3	58/F	CABG	Cannulation site	Supracoronary graft	CFA/RA	Hemiparesia	Discharge, day 15
4	55/F	CABG	Cross-clamp area	Supracoronary graft	CFA/RA	Death	Death, perioperative
5	69/F	CABG	Vent Area	Supracoronary graft	CFA/RA	Death	Death, perioperative
6	63/M	CABG	Partial occlusion clamp	Supracoronary graft	CFA/RA	Hemiparesia	Discharge, day 46
7	67/M	CABG	Cross-clamp area	Supracoronary graft	CFA/RA	Death	Death, perioperative
8	64/M	CABG	Partial occlusion clamp	Supracoronary graft	CFA/RA	Death	Death, perioperative
9	60/M	CABG	Cannulation site	Supracoronary graft	CFA/RA		Discharge, day 8
10	59/M	CABG	Cross-clamp area	Supracoronary graft	CFA/RA	Bleeding/ atelectasis	Discharge, day 14
11	74/F	CABG	Cross-clamp area	Supracoronary graft	CFA/RA	Bleeding/ reintubation	Discharge, day 20
12	54/M	CABG	Cannulation site	Supracoronary graft	CFA/RA	Pneumonia	Discharge, day 8
13	66/F	CABG	Cross-clamp area	Supracoronary graft	CFA/RA	Death	Death, perioperative
14	62/M	CABG	Vent area	Supracoronary graft	CFA/RA	Death	Death, perioperative
15	74/M	CABG	Proximal anastomoses	Local repair	Ascending aorta/RA	Bleeding/ broncospasm/ hemiparesia	Discharge, day 15
16	40/M	CABG	Proximal anastomoses	Supracoronary graft	CFA/RA		Discharge, day 9
17	67/F	CABG	Cannulation site	Supracoronary graft	CFA/RA	Bleeding/ compartment syndrome	Death, day 13
18	69/M	CABG	Cannulation site	Supracoronary graft	CFA/RA	Pleural effusion/ hemiplegia	Discharge, day 18
19	71/M	CABG	Partial occlusion clamp	Supracoronary graft	CFA/RA	Bleeding/TIA	Death, day 9
20	64/F	CABG	Proximal anastomoses	Supracoronary graft	CFA/RA	Hemiparesia	Discharge, day 15
21	62/F	CABG	Vent area	Supracoronary graft	CFA/RA	ARF/ hemiplegia	Discharge, day 20

*CABG indicates coronary artery bypass graft; CFA, common femoral artery; RA, right atrium; ARF, acute renal failure; TIA, transient ischemic attack.

who required treatment in the postoperative intensive care unit (ICU) underwent bleeding revision during the early postoperative period. Intraaortic balloon pump usage and lengths of ICU and hospital stay were significantly higher in patients with PIAD. The incidence of reversible and irreversible neurological impairment was also significantly higher in this group ($P = .0001$).

DISCUSSION

The incidence of PIAD during heart surgery is low, with an occurrence rate of about 0.12% [Blakeman 1988; Ruchat 1998; Fleck 2003]. The overall mortality rate of PIAD is 47.6%, significantly higher than that for primary acute type A dissections, which is about 20%-33%. [Kouchoukos 1997]. The Interna-

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Table 2. Patient Preoperative and Perioperative Variables*

	Group I (n = 21)	Group II (n = 603)	*P*
Age, years	62.1 ± 8	59.6 ± 7.6	NS
Female sex, n (%)	10 (47.6%)	129(21.4%)	.05
Diabetes mellitus, n (%)	6 (28.6%)	110 (18.2%)	NS
Hypertension, n (%)	14 (66%)	171 (28.4%)	.0001
Peripheral arterial disease, n (%)	11 (52.4%)	18 (3%)	.0001
Ejection fraction, %	52.31% ± 7.80%	52.73% ± 10.78%	NS
Aortic diameter, cm	3.83% ± 0.9%	2.93% ± 0.46%	.019
Cardiopulmonary bypass pressure ≥120 mmHg, n (%)	6 (28.6%)	20 (3.3%)	.0001
Anastomoses, n	1.85 ± 0.64	1.88 ± 0.25	NS

*NS indicates not significant.

tional Registry Of Acute Aortic Dissection [Collins 2004] reported an increased risk of cardiac complications in patients with spontaneous aortic dissection (SAD) compared to those with PIAD. Because of the urgency of SAD, patient cardiac function and coronary status are usually not evaluated in SAD patients. Patients with PIAD, however, undergo surgery for another cardiac condition and thus their cardiac function is fully evaluated. Nevertheless, mortality of PIAD is higher than that of SAD. This higher mortality may be attributable to the fact that patients with PIAD tend to be older than those with SAD, and advanced age is a known risk factor for death [Nienaber 2003; Collins 2004]. The second reason may be that surgeons are unprepared for PIAD, a situation that requires sudden decision-making and immediate provision of adequate perfusion by switching the cannulation to an appropriate site. In addition, cross-clamp and CPB time inevitably increase significantly in patients with coexisting cardiac disease. Therefore, the prevention of PIAD and immediate diagnosis when it occurs are the most important points in the management of PIAD patients.

In our study ICU and hospital stays were markedly prolonged in the surviving PIAD patients compared to patients who underwent CCABG without PIAD, a finding that we assumed to be a result of the complexity of the procedure and not of the CCABG itself.

Aortic dissections have been repeatedly reported as complications originating from cross-clamp injury [Litchford 1976] or from intimal tears at the suture line of a CABG [Archer 1986] or from the site of cannulation [Williams 1974]. Thus, intimal injury can result from direct laceration and mechanical compression, with increased risk with severe atherosclerosis and cystic medial necrosis [Epperlein 1994].

Table 3. Origins of Perioperative Iatrogenic Type I Aortic Dissection

Origin of dissection	n (%)
Cannulation site	5 (23.8)
Cross-clamp area	5 (23.8)
Partial occlusion clamp	4 (19)
Vent area	4 (19)
Proximal anastamoses	3 (14.2)
Total	21

Further independent predictors are aortic valve pathology [Larson 1984] and aortic wall fragility, dilation, and thinning [Mizutani 2003]. As in most previous reports, we observed that female sex, hypertension, dilatation and thinning of the aorta, and PAD, which indicates severe and diffuse atherosclerosis, were significant preoperative risk factors in our patients (Table-5). Among perioperative variables, we found that high CPB pressure (≥120 mmHg) was a significant risk factor. Because preoperative risk factors could not be changed, perioperative management in these high-risk patients became a priority. When systemic atherosclerosis is widespread and intense, the compliance of the arterial tree deteriorates and stiffness of the arterial system leads to resistance to the vasodilatation effect of drugs. In PIAD, surgical trauma is the main trigger mechanism, in conjunction with pathological conditions of the aorta that are often present in the population undergoing CCABG surgery. Many sites of manipulation may become the initiating point of the dissection. Therefore, general preventive measures include perioperative evaluation of the ascending aorta. Calcification or dilation of the aorta should alert the surgeon and lead him/her to select the site of the anastomosis carefully, manipulate the aorta gently, and choose appropriate clamps. The cannulation site should be distant from palpable aortic plaque to avoid separation of the plaque from the aortic wall, and systolic pressure should be below 100 mmHg to prevent excessive stress to the aortic wall [Gravlee 2000].

Table 4. Postoperative Patient Variables

	Group I (n = 21)	Group II (n = 603)	*P*
Intensive care unit stay, days	7.38 ± 8.08	1.8 ± 5.7	.005
Hospital stay, days	10.9 ± 11	6.97 ± 6.5	.008
Intraaortic balloon pump, n (%)	4 (19%)	13 (2.2%)	.0001
Bleeding revision, n (%)	6 (28.5%)	20 (3.3%)	.0001
Cerebrovascular accident, n (%)	7 (33.3%)	11 (1.8%)	.0001
Death, n (%)	10 (47.6%)	16 (2.7%)	.0001

Table 5. Risk Factors for Perioperative Iatrogenic Aortic Dissection*

Variable	Study Group (n = 21)	Control Group (n = 603)	*P*	*P*	Exp (B)	95% CI for Exp (B)	
						Lower Bound	Upper Bound
Female sex, n (%)	10 (47.6%)	129 (21.4%)	0.05	.012	7.916	2.733	10.627
Hypertension, n (%)	14 (66%)	171 (28.4%)	0.0001	.049	.137	.019	.993
Peripheral arterial disease, n (%)	11 (52.4%)	18 (3%)	.0001	.0001	.04	.02	.09
Diameter of aorta, cm	3.83 ± 0.9	2.93 ± 0.46	0.019	.003	2.219	1.45	7.315
≥120 mmHg CPB pressure, n (%)	6 (28.6%)	20 (3.3%)	.0001	.008	4.219	2.13	7.768

*Ex (B) indicates beta exponential distribution.

If possible, the intrapericardial aorta is chosen for aortic cannulation because this segment best resists tearing or dissection. Many surgeons insert 2 concentric purse-string sutures into the aortic wall. Surgeon opinions differ as to whether these sutures should be shallow, deep, or full-thickness bites [Kirklin 1993]. In our institution, most surgeons incise and then dissect away the adventitia within the purse-string suture. To minimize clamp trauma to the aorta, most surgeons avoid using a partial-occluding clamp, except in pediatric patients. Optimal arterial blood pressure during cannulation (mean arterial pressure of about 70-80 mmHg, systolic pressure of about 100 to 120 mmHg) is important: if pressure is too high, there is a greater chance of tears and dissection and blood loss and spray; if it is too low, the aorta tends to collapse, making incision and insertion of the cannula more difficult and increasing the risk of damaging the back wall of the aorta [Gravlee 2000]. In our study the presence of high CPB pressure significantly increased the risk of PIAD.

Some perioperative observations helped us to recognize the occurrence of PIAD. First and foremost was an unexpectedly increased CPB arterial line pressure when the systemic pump was activated. This increase often coincided with profoundly decreased systemic pressure measured by the indwelling arterial catheter. Other indications are the observation by the perfusionist of decreased venous drainage to the CPB circuit. At the surgical field, a hematoma may develop near the arterial cannulation site or there may be bleeding around the purse-string sutures [Gravlee 2000]. However, intraoperative dissection is most commonly noticed after removal of the aortic cross-clamp or the partial-occlusion clamp [Still 1992].

Another important factor for immediate diagnosis and management of PIAD is the time of occurrence. Studies have found that dissections were most frequently identified after removal of the aortic cross-clamp [Still 1992] or during or at the initiation of CPB [Fleck 2006]. In our study, the PIAD was identified after removal of the aortic cross-clamp in 11 patients, during aortic cannulation in 3 patients, and after removal of the lateral clamp in 5 patients. Therefore, care should be taken to place the heel of the partial-occlusion clamp slightly away from the aortic cannula. Torsion on the partial-occlusion clamp closely adjacent to the aortic cannula may injure the intima. To decrease pressure and the risk of tear, pump flow should be reduced whenever cross-clamps or partial-occlusion clamps are applied [Kirklin 1993].

When PIAD occurs, arterial cannulation must immediately be moved to another appropriate site, except in patients with limited dissection that can be repaired by use of a closed plication technique. In our clinic, the arterial cannula was moved to the femoral artery in all cases. Because of time limitations, we did not have the chance to prepare the axillary artery as a cannulation site. However, when it is difficult to confirm that these sites were intact and would not cause further malperfusion, transapical cannulation to the true lumen can enable speedy and certain perfusion to the true lumen [Mizutani 2003].

Change of cannulation site during CPB, mean arterial pressure less than 50 mmHg during change of cannulation site, and the time of diagnosis of dissection are accepted as the major risk factors for PIAD [Mizutani 2003]. When dissection is diagnosed at the beginning of CPB the prognosis is good, but when diagnosis occurs at the end of surgery or CPB, the prognosis is accepted to be poor owing to a long period of malperfusion. In our patients, PIAD were noticed after removal of the aortic cross-clamp in 11 patients, during aortic cannulation in 3 patients, and after removal of lateral clamp in 5 patients. The mean time from onset of dissection until reestablishment of normal perfusion was 19 ± 3.5 minutes. The length of this time interval may be a reason for the high overall mortality (47%) and also the high rate of neurological impairment observed in our patients.

As previously mentioned, the most common locations for aortic dissection during cardiac surgery are sites of cross-clamp injury [Larson 1984], proximal anastomosis lines (suture lines from CABG) [Archer 1986], and sites of cannulation [Williams 1974]; other risk factors include the used of CPB in on- or off-pump CABG [Chavanon 2001], manipulation of the ascending aorta in the presence of concomitant vessel wall abnormalities [Chavanon 2001], proximal anastomosis with lateral clamp or under cross-clamp [Stanger 2002], and aortic valve surgery [Murphy 1983]. The potential preoperative risk factors for perioperative aortic dissection are history of hypertension, severe atherosclerosis, and a thin or dilated ascending aorta and aortic-valve pathology [Murphy 1983].

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The diagnosis of PIAD was made on the basis of bluish discoloration of the aorta in conjunction with high impedance at the beginning of CPB or cannulation. Subintimal extravasations of blood produce a false lumen that rapidly bleeds at sites of proximal venous anastomoses, cannulation, or cardioplegia.

The appropriate repair of PIAD depends on the extent and location of the dissection. For tears proximal to aortic cannulation that do not involve proximal vein anastomoses, repair can be accomplished by primer repair. When proximal anastomosis is involved, Dacron graft interposition is necessary. In our clinic, supracoronary aorta graft replacement was performed in 19 cases, elephant-trunk technique in 1 case, and patchplasty with synthetic graft material in 1 case. All patients underwent TCA after the aortic cannula was placed in the femoral artery. If the aorta can be cross-clamped distal to the dissection without moving the aortic cannula, the repair can be performed without recannulation. Generally, however, the dissection occurs distal to the cannulation site, making more distal cannulation necessary.

CONCLUSION

PIAD is a frequently fatal complication of CCABG. Preexisting aortic wall diameter and pathology, hypertension, PAD, and the intraoperative manipulation of the aorta contribute to the pathophysiology of this condition. Our results showed that female sex, history of PAD and hypertension, and high pressure during CPB are risk factors for PIAD. In such patients, the surgeon and his team must be alert for signs of PIAD, keep CPB pressures in the normal range, and prevent trauma to the aorta. Extreme caution for manipulation may be mandatory if the aorta is dilated. Awareness of the high risk of aortic dissection during surgery in such patients may prevent aortic dissection.

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