Long-term follow up after transcatheter closure of atrial septal defect and patent foramen ovale in adults

Dolgoročno spremljanje odraslih bolnikov po transkatetrskem zapiranju okvare preddvornega pretina in odprtega ovalnega okna

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Izvleček

Izhodišča: Namen raziskave je bil proučiti dolgoročne elektrokardiografske in ehokardiografske spremembe ter opredeliti zaplete pri perkutanem zapiranju okvare preddvornega pretina in odprtega ovalnega okna pri odraslih.

Metode: Retrospektivno smo analizirali klinične, elektrokardiografske in ehokardiografske spremembe pri 137 zaporednih bolnikih v 10-letnem obdobju po perkutanem zapiranju okvare preddvornega pretina (51 bolnikov) in odprtega ovalnega okna (86 bolnikov).

Rezultati: Pri skupini bolnikov z odprtim ovalnim oknom nismo zabeležili pomembnih sprememb po posegu. Pri obeh skupinah nismo zabeležili pomembnih sprememb srčne frekvence in ritma ter trajanja intervalov PR in QRS. Pri skupini z okvaro preddvornega pretina smo zabeležili premik srčne osi v levo ($p = 0{,}017$), zmanjšanje ocenjenega sistoličnega tlaka v pljučni arteriji ($p = 0{,}024$), zmanjšanje zgodnje diastolične dopplerske hitrosti na trikuspidalni zaklopki ($p = 0{,}002$), zmanjšanje desnih srčnih votlin ($p = 0{,}0004$) in normalizacijo gibanja prekatnega pretina ($p < 0{,}0001$). Večina zapletov, povezanih s posegom, je bila zgodnjih in blagih, zabeležili pa smo 3 resne zgodnje zaplete.

Zaključki: Pri skupini z odprtim ovalnim oknom nismo zabeležili pomembnih elektrokardiografskih in ehokardiografskih sprememb. Pri skupini z okvaro preddvornega pretina pride do pomembnega in hitrega izboljšanja morfoloških in hemodinamičnih sprememb srca. Zapleti, povezani s posegom, so večinoma blagi in zgodnji, možni pa so tudi resni pozni zapleti. Zato je potrebno te bolnike spremljati dolgoročno.

Abstract

Background: The aim of our study was to define long-term electrocardiographic and echocardiographic changes and complications after transcatheter closure of atrial septal defect and patent foramen ovale in adults.

Methods: The clinical, electrocardiographic and echocardiographic follow-up of 137 consecutive patients that underwent transcatheter closure of atrial septal defect (51 patients) or patent foramen ovale (86 patients) in a 10-year period was analyzed retrospectively.

Results: In the patent foramen ovale group, we observed no significant postprocedural changes. There were no changes in heart rate, heart rhythm and PR or QRS duration in both groups. In the atrial septal defect group, we observed a leftward shift in the heart axis ($p = 0.017$), a decrease in the estimated systolic pulmonary artery pressure ($p = 0.024$), decreased tricuspid early diastolic flow velocity ($p = 0.002$), a decrease in the right chamber dimensions ($p = 0.0004$) and interventricular septal movement normalization ($p < 0.0001$). Most of the complications were mild and occurred early after the procedure. Three early serious complications were documented.

Conclusions: No electrocardiographic or echocardiographic changes occurred after patent foramen ovale closure. Atrial septal defect closure is related to significant early morphological and hemodynamic improvement. Postprocedural complications are usually early and mild but serious late complications can occur. For that reason, long-term follow up is recommended in these patients.

Introduction

Atrial septal defect (ASD) is one of the most common congenital cardiac lesions occurring in up to 10 % of patients with congenital heart disease at birth.[1] Secundum type ASD is the most common type, occurring in up to 60 % of ASD patients.[1] Traditionally, the only treatment modality in patients with hemodynamically important secundum type ASD was open heart surgery. The first interventional transcatheter closure of ASD was described in 1976[2] and is now a widely available form of treatment for relevant lesions in pediatric and adult populations.

Patent foramen ovale (PFO) is present in 25 to 27 % of the adult population[3,4] and is usually an unimportant echocardiographic or autopsy finding. It is presumed that in one out of 1000 patients with PFO, a cryptogenic embolic stroke occurs as a result of paradoxical embolism through PFO.[3,5] Though, the number could be a lot higher because it is often difficult to ascertain where the embolism originated from. In patients with doubtlessly proved paradoxical embolism, the transcatheter closure of PFO is the usual mode of treatment. On the other hand, there is no evidence that PFO closure is any better than medical therapy alone as was shown in a recently published CLOSURE I trial.[6]

In both subgroups of patients, the data on the long-term results of the transcatheter procedure is lacking. Most studies are small and only sporadic cases of complications, such as new-onset atrial fibrillation, conduction abnormalities, aortic and mitral regurgitation, device embolisation, pericardial effusion or tamponade, residual right-to-left cardiac shunt, recurrent stroke or transient ischemic attack, are described.[7-16] In both subgroups of patients, the electrocardiographic and echocardiographic changes after the procedure are not well defined.[17-21]

The aim of our study is to define these changes and immediate and long-term postprocedural complications after percutaneous transcatheter occlusion of either secundum type ASD or PFO.

Patients and methods

Patients. From January 2001 to December 2010, 137 consecutive adult patients with PFO (86 patients) or secundum type ASD (51 patients) were treated with percutaneous transcatheter defect occlusion at the Department of Cardiology of the University Medical Centre Ljubljana, Slovenia. Patient data was collected and analysed retrospectively. The inclusion criteria for PFO patients were age under 55 and cryptogenic embolic stroke verified by positive magnetic resonance imaging. The inclusion criteria for ASD patients were the presence of a hemodynamically significant defect with anatomy appropriate for percutaneous occlusion. Amplatzer septal occluder was used in all patients. We studied ASD and PFO patients in the same study because the procedure and the type of the closure device for both defects were very similar.

Follow up. Patients were followed up at regular intervals. Clinical data before the procedure, six months after the procedure and later on at yearly intervals were collected. No patients were lost to follow up.

Standard 12-lead electrocardiographs were recorded before the procedure, six months after the procedure and later on at yearly intervals. Heart rate, rhythm, axis, PR interval and QRS duration data were collected.

Transthoracic echocardiography was performed before the procedure, one day after, one and six months after the procedure and later on at yearly intervals. The right atrial size, right ventricular size and function, and coronary sinus anatomy were assessed from the apical four-chamber view. The interventricular septal movement, left ventricular size and function, as well as the pericardial space, were observed from the parasternal views. We assessed the valvular function. The right heart chambers' size and function were estimated qualitatively.[22] The systolic pulmonary artery pressure was estimated from the continuous wave Doppler measurements of the tricuspid regurgitant flow velocity. Transesophageal echocardiography was performed before and during the procedure. Before the procedure, the

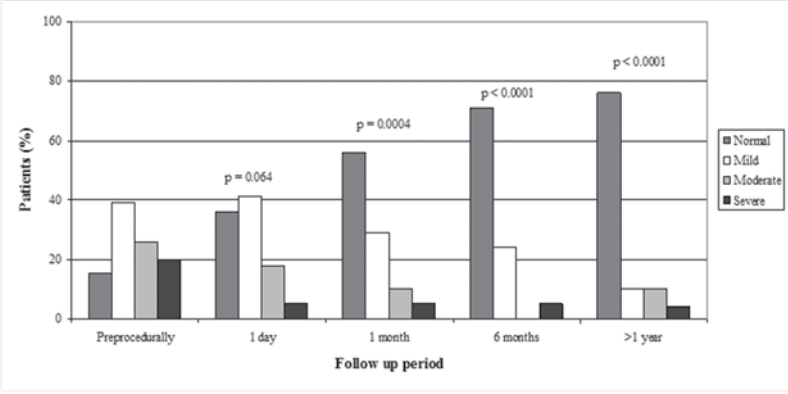

Figure 1: Distribution of right atrial size 1 day, 1 month, 6 months and > 1 year after atrial septal defect closure.

presence of PFO and right-to-left shunt was confirmed using contrast echocardiography. In ASD patients, the size and anatomy of the defect was observed. During the procedure, the position of the occluder device was assessed in all patients.

Statistical analysis. The Kolmogorov-Smirnov test was used to verify normal distribution. Normally distributed continuous variables were expressed as means and standard deviations. In non-normal distributed continuous variables, the data was expressed as a median together with the 25th and 75th percentiles (inter-quartile range). Categorical data were summarized as frequencies and percentages. For comparisons of continuous variables, the paired Student t-test was used for normally distributed variables and the Wilcoxon matched-pair test for non-normally distributed variables. The data for categorical variables were analyzed by using the Fisher exact test. For all tests, a two-tailed p value less than or equal to 0.05 was considered statistically significant. Data was analyzed with the Statistical Package for Social Sciences version 16 (SPSS Inc, Chicago, Illinois).

Results

The baseline patient characteristics are shown in Table 1.

Electrocardiography. In the PFO group, the mean heart rate before the defect occlusion was 69 ± 11 beats per minute. Mean heart axis before the procedure was 43 ± 31°. Mean PR interval before the procedure was 152 ± 26 ms and mean QRS duration was 89 ± 10 ms. All these parameters did not change during follow-up. All PFO patients were in sinus rhythm before the procedure and remained in sinus rhythm during follow up.

In the ASD group, the mean heart rate before the defect occlusion was 78 ± 13 beats per minute. Mean PR interval before the procedure was 147 ± 25 ms and mean QRS duration was 100 ± 17 ms. None of these parameters changed during follow-up. However, we observed a statistically significant change of heart axis from 80 ± 36° before the procedure to 58 ± 37° six months after the procedure (p = 0.017). Forty-six ASD patients (90 %) were in sinus rhythm before the procedure and remained in sinus rhythm throughout the follow-up period. Only one patient converted to sinus rhythm from atrial fibrillation postprocedurally.

Echocardiography. The baseline echocardiographic characteristics of both study subgroups are summarized in Table 2.

In the PFO group, none of the measured echocardiographic parameters changed throughout the follow–up period. None of the PFO patients had pericardial effusion or coronary sinus abnormality pre- or postprocedurally.

In the ASD group, we found a statistically significant decrease in the estimated systolic pulmonary artery pressure. From 35 ± 7 mmHg (+ central venous pressure) preprocedurally, the pressure decreased to 30 ± 12 mmHg (+ central venous pressure) one day after the procedure (p = 0.024). The decrease in pulmonary artery pressure remained statistically significant throughout the follow–up period. We also documented an increase in the early diastolic pulsed-wave Doppler velocity at the mitral orifice from 85 ± 19 cm/s before the procedure to 99 ± 24 cm/s one month after the procedure (p = 0.004). The early diastolic pulsed wave Doppler velocity at the tricuspid orifice decreased from 85 ± 33 cm/s before the procedure to 52 ± 19 cm/s one day after the procedure (p = 0.002). Both Doppler parameter differences remained statistically significant throughout the follow-up period. In ASD group we also found a postprocedural decrease in the right heart chamber dimensions and normalization of interventricular septal movement (Figs 1–3). None of the other me-

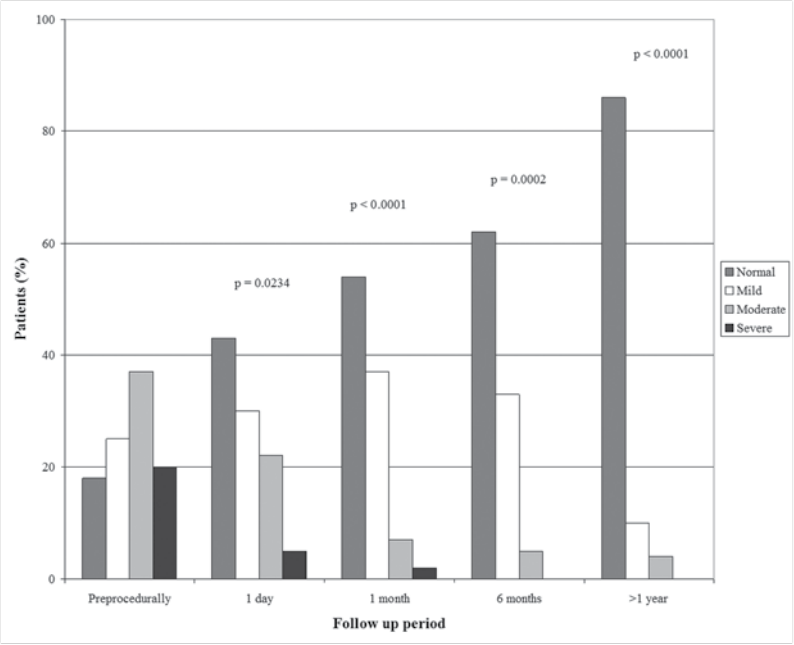

Figure 2: Distribution of right ventricular size 1 day, 1 month, 6 months and > 1 year after atrial septal defect closure.

asured echocardiographic parameters changed throughout the follow–up period.

In the ASD group, three patients had mild pericardial effusion on the first day after the procedure. Later on, we observed no new-onset pericardial effusions. None of the effusions were hemodynamically significant. None of the patients in the ASD group had coronary sinus abnormality throughout the follow–up period.

Complications. The device implantation success rate was 98.6 %. In the PFO group, one patient suffered from hypotensive (vasovagal) reaction during the procedure. We observed two serious complications in two patients. After the procedure, one patient needed surgery because of arteriovenous fistula on the femoral artery access site. Another patient suffered from epigastric artery rupture with mild hemorrhagic shock. We observed two early postprocedural device embolisations without serious consequences.

In the ASD group, we observed one serious complication in a patient with the rupture of the pulmonary vein and left atrium with cardiac tamponade. After urgent surgical intervention, the patient underwent an uneventful recovery.

No thromboembolic events or late device-related erosions were observed in either subgroup of patients. Also, no significant valvular regurgitation was detected postprocedurally.

Discussion

PFO group. No significant electrocardiographic changes were observed postprocedurally in the PFO group. There were no heart rhythm or heart axis changes and no conduction abnormalities, which suggests that the anatomical position of the PFO septal occluder does not interfere with the conduction system of the heart. On the other hand, Johnson et al. reports changes in atrial conduction postprocedurally but the incidence of the clinically significant heart block was low.[23] Mareedu et al. observed paroxysmal atrial fibrillation in one patient postprocedurally.[4]

Similarly, no echocardiographic changes were found postprocedurally in the PFO group. This was expected because PFO is not a hemodynamic anomaly.

ASD group. There were no significant changes in the heart rate, heart rhythm, PR interval or QRS interval pre- or postprocedurally. Interestingly, Mainzer et al. observed an improvement in the right bundle branch block pattern in 53 % of adult patients after ASD closure.[20] Some authors report a 2 to 7 % incidence of different degrees of atrioventricular block early postprocedurally.[11,14,24] Usually, atrioventricular blocks are transient and only rarely pacemaker implantation is needed.[14] Some studies report a relatively high percentage of new-onset atrial fibrillation postprocedurally in up to 4.5 % of patients[9,11,17] and other report increased supraventricular ectopy.[24] In our study, only one patient converted from atrial fibrillation preprocedurally to sinus rhythm postprocedurally. However, we observed a significant leftward heart axis shift postprocedurally. We presume this change to be a consequence of hemodynamic relief of the right ventricle after the closure of the left-to--right shunt. A similar finding is reported by Dhillon et al.[25]

On the first day after the procedure, both right atrial and right ventricular chamber dimensions decreased significantly and remained significant throughout the fol-

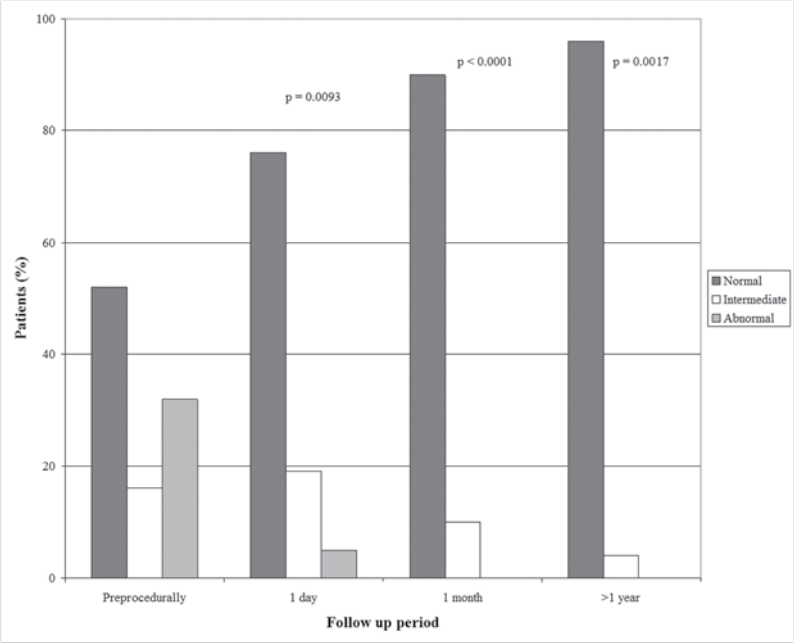

Figure 3: Distribution of interventricular septal motion 1 day, 1 month, 6 motnhs and > 1 year after atrial septal defect closure

low–up period. The same right chamber dimension reduction was observed by other authors,[19,21] but as late as six weeks after the procedure[21] or even six months after the procedure.[26] Dhillon et al. reports preserved right ventricular function after transcatheter ASD closure and impaired right ventricular function after surgical ASD repair.[25] These observations support the potential for favorable right heart remodeling after ASD transcatheter closure, despite longstanding right chamber volume overload.[19,26] Also, in percutaneous ASD closure the patient does not suffer from negative influence of extracorporeal circulation as in surgical ASD closure. Similarly, we observed normalization of the interventricular septal movement on the first day postprocedurally, which remained significant throughout the follow–up . period. Mainzer et al. observed interventricular septal movement normalization as late as one month after the procedure.[20]

We found no changes in the left ventricular dimensions postprocedurally, yet some authors report left ventricular enlargement after the ASD closure, probably due to normalization of the left ventricular load after the closure of the left-to-right cardiac shunt.[19-21] We also found no changes in right or left ventricular function after the ASD closure, probably due to the early intervention in individuals with preserved ventricular function. However, some authors report left ventricular ejection fraction improvement after the procedure.[21]

In the present study, we observed a significant drop in the estimated systolic pulmonary artery pressure as early as on the first day after the ASD closure and the drop remained significant throughout the follow--up period. Other authors report a similar drop in systolic pulmonary artery pressure.[9,17,18,20] We also found a significant drop

Table 1: Baseline characteristics of the study cohort (N = 137).

	PFO patients	ASD patients
N (%)	86 (62)	51 (38)
Mean age, years	40.35 ± 8.88	40.35 ± 8.88
Male sex (%)	36 (41)	16 (31)
Sinus rhythm (%)	100	84
Positive TCD (%)	98	
Mean defect diameter (mm) (central position, %)	/	16.9 ± 5.9 (68)
Mean occlusion device diameter (mm)	23.96 ± 5.09	26.96 ± 6.27
ASA at discharge (%)	97	95
Clopidogrel at discharge (%)	56	36
Warfarin at discharge (%)	8	12
Median follow up (days) (inter-quartile range)	951 (624–1852)	1835 (660–2619)

PFO, patent foramen ovale; ASD, atrial septal defect; TCD, transcranial Doppler; ASA, acetylsalicilic acid

in the tricuspid early diastolic blood flow velocity on the first day after the procedure. On the other hand, mitral early diastolic flow velocity increased significantly as late as one month postprocedurally. Both changes of flow velocities remained significant throughout the follow-up period. We understand these changes as a consequence of the reduction of transtricuspid flow and as an increase in transmitral flow after ASD closure. In conclusion, we observed significant morphological and hemodynamic improvement that occurred already within the first day after the procedure and continued later on. These echocardiographic changes reflect the physiological changes after ASD

Table 2: Baseline echocardiographic characteristics of study group.

	PFO patients	ASD patients
Right atrial enlargement (%)		
Normal	91	15
Mild	9	39
Moderate	0	26
Severe	0	20
Interventricular septal movement		
Normal	96	52
Intermediate	4	16
Abnormal	0	32
Right ventricular enlargement (%)		
Normal	95	18
Mild	5	25
Moderate	0	37
Severe	0	20
Right ventricular function (%)		
Normal	100	97
Mildly decreased	0	3
Left ventricular enlargement (%)		
Normal	100	97
Moderate	0	3
Left ventricular function (%)		
Normal	100	97
Moderatly decreased	0	3
Tricuspid valvular regurgitation (%)		
None	71	20
Mild	29	55
Moderate	0	20
Severe	0	5
Mitral valvular regurgitation (%)		
None	78	58
Mild	21	37
Moderate	1	5
Aortic valvular regurgitation (%)		
None	94	95
Mild	5	5
Moderate	1	0

PFO, patent foramen ovale; ASD, atrial septal defect.

closure, and similar changes can be seen after surgical closure of the defect.[25]

Complications. The device implantation success rate in our study was as high as 98.6 %, and similar to other published series with success rates ranging from 90 to 100 %.[4,7,9,11,15,18,19,21] These observations suggest that transcatheter closure of ASD or PFO can be performed with a high overall procedural implantation success rate in appropriately selected patients.

Most complications in our study were mild and occurred within the first 24 hours after the procedure.

In the PFO group, we observed no recurrent neurologic events. Other authors report a 0 to 4.4 % rate of recurrent neurologic events.[7-9,15] Cifarelli et al. defined age and atrial septal aneurysm as risk factors for recurrent neurological events[8], while Fischer et al. observed a high recurrent neurologic event rate as a consequence of an inappropriate antiplatelet regimen postprocedurally.[15] The low recurrent neurologic event rate in these studies suggests that a combined acetylsalicilic acid and clopidogrel antiplatelet regimen is optimal. In our cohort, some patients received single antiplatelet regimen at discharge because of a high risk of bleeding . Some patients received a single antiplatelet medication in combination with warfarin because of co-existing indication for anticoagulant treatment, in most cases due to coexisting atrial fibrillation. On the other hand, the CLOSURE I trial found no difference in the occurrence of recurrent neurologic events between patients treated with medical therapy alone and those treated with percutaneous closure of the defect.[6] We observed no significant postprocedural aortic or mitral regurgitation, no thromboembolic events and no coronary sinus abnormalities in either group of patients. There are some reports of sporadic cases of thrombus formation on the device early[19] and late after the procedure.[9]

None of the PFO patients had pericardial effusion postprocedurally, while three ASD patients had pericardial effusion on the first day after the procedure (2.2 % in both groups). Effusions resolved spontaneously without any intervention. Other authors report a lower 0.8–1.2 % incidence of pericardial effusion postprocedurally.[9,19]

Three patients (2.2 %) suffered from serious complications. All of them occurred early after the procedure and were related to procedural technique and not to device--related thromboembolic events. All serious complications occurred early in the learning curve.

Some authors report cases of serious late complications, such as pulmonary venous obstruction that occurred 3.5 years after transcatheter closure of ASD[10], or late severe mitral valve insufficiency that occurred three months after ASD closure.[12] Another life threatening late complication is device erosion of adjacent structures. The incidence of such erosions in the United States is 0.1 %, and patients at increased risk are those with deficient aortic rim and/or superior rim and patients with oversized devices.[27] We observed none of such late complications. Nevertheless, because late complications can be serious, these patients should not be discharged completely.

The most common surgical complications after ASD device implantation in a study reported by Sarris et al. also occurred mostly within 48 hours postprocedurally.[28] In addition, some patients needed surgery as late as eight years after device implantation, thus imposing the need for lifelong follow-up of these patients.

This overview of possible complications after percutaneous transcatheter ASD or PFO closure suggests that this procedure is a reasonably safe and feasible technique that can be performed in selected individuals. Recent studies suggest that in PFO patients careful risk and benefit evaluation should be performed before closing the defect since the procedure seems not to be more efficient that conservative medical treatment alone. Further larger studies should confirm or exclude these observations.

Study limitations. The main disadvantage of our study is the retrospective study design. Also, the number of enrolled patients is relatively low. For this reason as well as the reasons mentioned earlier we included both PFO and ASD patients in the same analysis. To assess procedural success rate in PFO

group more accurately, we should perform transcranial Doppler studies postprocedurally.

Conclusions

In a retrospective study, we analyzed rhythm, morphological and hemodynamic changes and complications after percutaneous transcatheter closure of PFO or ASD in adults. These changes were minimal in PFO patients. On the other hand, we observed significant and early morphological and hemodynamic improvement in ASD patients that continued throughout the follow up. Most of the complications in both groups of patients were mild and occurred peri- or early postprocedurally. In our cohort, no late complications were observed.

Abbreviations

ASD, atrial septal defect
PFO, patent foramen ovale

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