

World Journal of *Clinical Cases*

World J Clin Cases 2022 April 26; 10(12): 3639-3968



EVIDENCE REVIEW

- 3639 Tilt and decentration with various intraocular lenses: A narrative review
Chen XY, Wang YC, Zhao TY, Wang ZZ, Wang W

REVIEW

- 3647 Role of zonula occludens in gastrointestinal and liver cancers
Ram AK, Vairappan B

MINIREVIEWS

- 3662 Pathophysiological mechanisms of hepatic stellate cells activation in liver fibrosis
Garbuzenko DV

ORIGINAL ARTICLE**Retrospective Cohort Study**

- 3677 Predictors of unfavorable outcome at 90 days in basilar artery occlusion patients
Chiu YC, Yang JL, Wang WC, Huang HY, Chen WL, Yen PS, Tseng YL, Chen HH, Tsai ST

Retrospective Study

- 3686 Role of multidetector computed tomography in patients with acute infectious colitis
Yu SJ, Heo JH, Choi EJ, Kim JH, Lee HS, Kim SY, Lim JH
- 3698 Efficacy and prognostic factors of neoadjuvant chemotherapy for triple-negative breast cancer
Ding F, Chen RY, Hou J, Guo J, Dong TY
- 3709 Relationship between subgroups of central and lateral lymph node metastasis in clinically node-negative papillary thyroid carcinoma
Zhou J, Li DX, Gao H, Su XL
- 3720 Nomogram to predict postoperative complications in elderly with total hip replacement
Tan XJ, Gu XX, Ge FM, Li ZY, Zhang LQ
- 3729 Flap failure prediction in microvascular tissue reconstruction using machine learning algorithms
Shi YC, Li J, Li SJ, Li ZP, Zhang HJ, Wu ZY, Wu ZY

Observational Study

- 3739 Surgery in platinum-resistant recurrent epithelial ovarian carcinoma
Zhao LQ, Gao W, Zhang P, Zhang YL, Fang CY, Shou HF

- 3754 Anorectal dysfunction in patients with mid-low rectal cancer after surgery: A pilot study with three-dimensional high-resolution manometry

Pi YN, Xiao Y, Wang ZF, Lin GL, Qiu HZ, Fang XC

Randomized Controlled Trial

- 3764 Effect of wrist-ankle acupuncture on propofol dosage during painless colonoscopy: A randomized controlled prospective study

He T, Liu C, Lu ZX, Kong LL, Li Y, Xu Z, Dong YJ, Hao W

META-ANALYSIS

- 3773 Melatonin intervention to prevent delirium in hospitalized patients: A meta-analysis

You W, Fan XY, Lei C, Nie CC, Chen Y, Wang XL

- 3787 Risk factors for hospital readmissions in pneumonia patients: A systematic review and meta-analysis

Fang YY, Ni JC, Wang Y, Yu JH, Fu LL

CASE REPORT

- 3801 Anti-programmed death 1 antibody in the treatment of coexistent *Mycobacterium fortuitum* and lung cancer: A case report

Zhang CC, Chen P

- 3808 Acute pancreatitis-induced thrombotic thrombocytopenic purpura: A case report

Wang CH, Jin HF, Liu WG, Guo Y, Liu Z

- 3814 Successful management of life-threatening aorto-esophageal fistula: A case report and review of the literature

Zhong XQ, Li GX

- 3822 Isolated coagulopathy without classic CRAB symptoms as the initial manifestation of multiple myeloma: A case report

Zhang Y, Xu F, Wen JJ, Shi L, Zhou QL

- 3828 Evaluation of intracoronary function after reduction of ventricular rate by esmolol in severe stenotic myocardial bridge: A case report

Sun LJ, Yan DG, Huang SW

- 3834 Pediatric living donor liver transplantation using liver allograft after *ex vivo* backtable resection of hemangioma: A case report

Li SX, Tang HN, Lv GY, Chen X

- 3842 Kimura's disease in soft palate with clinical and histopathological presentation: A case report

Li W

- 3849 Combined targeted therapy and immunotherapy in anaplastic thyroid carcinoma with distant metastasis: A case report

Ma DX, Ding XP, Zhang C, Shi P

- 3856** Successful multimodality treatment of metastatic gallbladder cancer: A case report and review of literature
Zhang B, Li S, Liu ZY, Peiris KGK, Song LF, Liu MC, Luo P, Shang D, Bi W
- 3866** Ischemic colitis after receiving the second dose of a COVID-19 inactivated vaccine: A case report
Cui MH, Hou XL, Liu JY
- 3872** Cryoballoon pulmonary vein isolation and left atrial appendage occlusion prior to atrial septal defect closure: A case report
Wu YC, Wang MX, Chen GC, Ruan ZB, Zhang QQ
- 3879** Surgical treatment for a combined anterior cruciate ligament and posterior cruciate ligament avulsion fracture: A case report
Yoshida K, Hakozaki M, Kobayashi H, Kimura M, Konno S
- 3886** Successful robot-assisted partial nephrectomy for giant renal hilum angiomyolipoma through the retroperitoneal approach: A case report
Luo SH, Zeng QS, Chen JX, Huang B, Wang ZR, Li WJ, Yang Y, Chen LW
- 3893** Cryptococcal antigen testing of lung tissue homogenate improves pulmonary cryptococcosis diagnosis: Two case reports
Wang WY, Zheng YL, Jiang LB
- 3899** Combined use of extracorporeal membrane oxygenation with interventional surgery for acute pancreatitis with pulmonary embolism: A case report
Yan LL, Jin XX, Yan XD, Peng JB, Li ZY, He BL
- 3907** Dynamic navigation system-guided trans-inferior alveolar nerve implant placement in the atrophic posterior mandible: A case report
Chen LW, Zhao XE, Yan Q, Xia HB, Sun Q
- 3916** Anti-glomerular basement membrane disease with IgA nephropathy: A case report
Guo C, Ye M, Li S, Zhu TT, Rao XR
- 3923** Amniotic membrane transplantation in a patient with impending perforated corneal ulcer caused by *Streptococcus mitis*: A case report and review of literature
Hsiao FC, Meir YJJ, Yeh LK, Tan HY, Hsiao CH, Ma DHK, Wu WC, Chen HC
- 3930** Steriod for Autoimmune pancreatitis complicating by gastric varices: A case report
Hao NB, Li X, Hu WW, Zhang D, Xie J, Wang XL, Li CZ
- 3936** Antithrombotic treatment strategy for patients with coronary artery ectasia and acute myocardial infarction: A case report
Liu RF, Gao XY, Liang SW, Zhao HQ
- 3944** Mesh plug erosion into the small intestine after inguinal hernia repair: A case report
Xie TH, Wang Q, Ha SN, Cheng SJ, Niu Z, Ren XX, Sun Q, Jin XS
- 3951** Recurrence of infectious mononucleosis in adults after remission for 3 years: A case report
Zhang XY, Teng QB

3959 Vertical direction impaction of kissing molars: A case report

Wen C, Jiang R, Zhang ZQ, Lei B, Yan YZ, Zhong YQ, Tang L

LETTER TO THE EDITOR

3966 Comment on “Outcomes of different minimally invasive surgical treatments for vertebral compression fractures: An observational study”

Ma L, Luo ZW, Sun YY

ABOUT COVER

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Evaluation of intracoronary function after reduction of ventricular rate by esmolol in severe stenotic myocardial bridge: A case report

Long-Jun Sun, Ding-Guang Yan, Shu-Wei Huang

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Abstract

BACKGROUND

Severe stenotic myocardial bridges (MBs) have been reported to lead to intracoronary ischaemia, but the physiological evaluation of MBs using intracoronary function evaluation indicators after intraoperative drug treatment has not been fully established.

CASE SUMMARY

We performed through snuff fossa for coronary angiography in a patient with chest tightness after repeated exercise, and the results showed that the middle part of the anterior descending branch was a MB with 100% systolic compression. The intracoronary function evaluation (defined as the ratio of distal coronary pressure to aortic pressure with zero microcirculation resistance) was instantaneous wave-free ratio (IFR) without drug and fractional flow reserve (FFR) with adenosine. The IFR was 0.73, and the FFR was 0.66. Then esmolol 0.02 µg/kg/min was intravenously injected. The IFR and FFR were measured again when the heart rate dropped to 60 beats/min. The IFR was 0.83, and the FFR 0.65.

CONCLUSION

This case report is a case of isolated MB with severe stenosis. After intraoperative drug treatment decreased the ventricular rate, an increase in the coronary function evaluation index was immediately observed to confirm the effective improvement of coronary blood flow.

Key Words: Myocardial bridge; FFR; IFR; Drug therapy; Coronary artery disease; Angiography; Coronary; Case report

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Core Tip: Myocardial bridge is one of the causes of myocardial ischaemia, and some studies have found that the degree of ischaemia is positively correlated with the degree of systolic compression. In this case of severe stenosis, after intraoperative drugs reduced the ventricular rate of the patient, the coronary ischaemia improved. This is a relatively novel change in coronary function that was determined by evaluation indices.

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INTRODUCTION

Coronary arteries are usually distributed on the surface of the epicardium, and occasionally segmental blood vessels run through the myocardium[1]. When this blood vessel is compressed by 70% in systole and 35% in late diastole, it is called a myocardial bridge[2]. Referring to relevant studies, Myocardial bridge (MB) can cause ischaemia in some patients, and the degree of systolic compression is inversely proportional to coronary flow reserve[3]. It has been proposed that drugs are the first choice for the treatment of MB. For isolated MB with severe stenosis, it is necessary to evaluate the state of coronary blood flow. The main evaluation methods are fractional flow reserve (FFR) and instantaneous wave-free ratio (IFR). FFR is defined as the ratio of distal coronary artery pressure to aortic pressure under the condition of maximal dilatation of small vessels and microvessels in the coronary artery supply area and no significant increase in central venous pressure under the action of adenosine[4]. IFR refers to the ratio of distal mean pressure to mean arterial pressure in diastolic nonwaveform interphase stenosis. This principle uses the blood pressure with the lowest diastolic coronary artery resistance to approximately replace coronary artery blood pressure under the action of adenosine[5,6]. In this case, we describe a case of coronary angiography *via* the distal radial artery pathway. The results showed that the anterior descending branch MB was associated with 100% systolic compression (Figure 1). After intraoperative drug control of the ventricular rate, the improvement of the coronary ischaemic state was confirmed by changes in the evaluation index of coronary artery function.

CASE PRESENTATION

Chief complaints

A 37-year-old Chinese male was admitted to the hospital with repeated chest tightness for two years.

History of present illness

After taking aspirin, tigenol and atorvastatin calcium before the operation, the patient still showed chest tightness. Since the chest tightness was not alleviated, coronary angiography was performed.

History of past illness

The patient had a history of previous hypertension for 4 years, was not taking medications, denied a history of diabetes and other chronic illnesses, had no history of long-term smoking or hyperlipidaemia, and had no family history of heart disease or other related risk factors for coronary heart disease.

Personal and family history

No family history of heart disease.

Physical examination

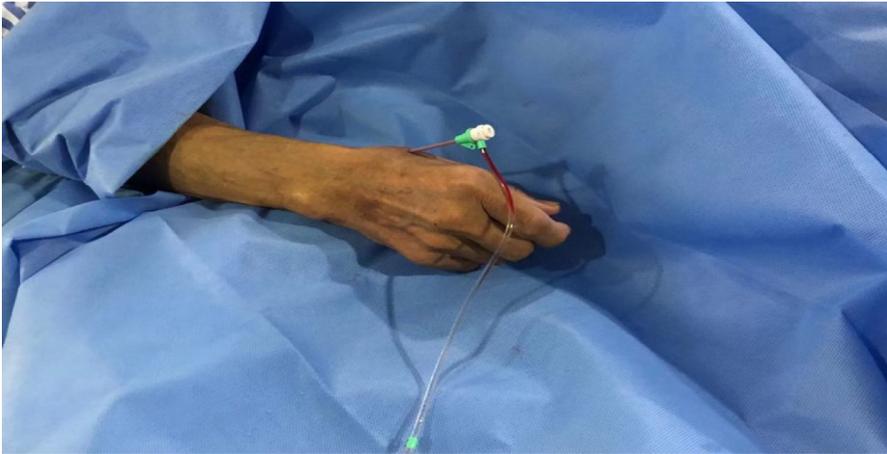
Physical examination revealed an auscultation heart rate of 74 beats/min and no pathological murmur was found in each valve auscultation area. There was no enlargement of the heart boundary of percussion and no tremor in palpation.

Laboratory examinations

No obvious abnormality in laboratory examination.

Imaging examinations

Twenty-four hours before the operation, the electrocardiogram was normal, and echocardiography



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Figure 1 16cm terumo sheath tube through snuff fossa for coronary angiography.

showed that the left ventricular systolic function was normal.

FINAL DIAGNOSIS

The results showed that the MB of the middle part of the left anterior descending branch was accompanied by 100% systolic compression, and the rest of the vessels did not have any other significant stenosis (Figure 2).

TREATMENT

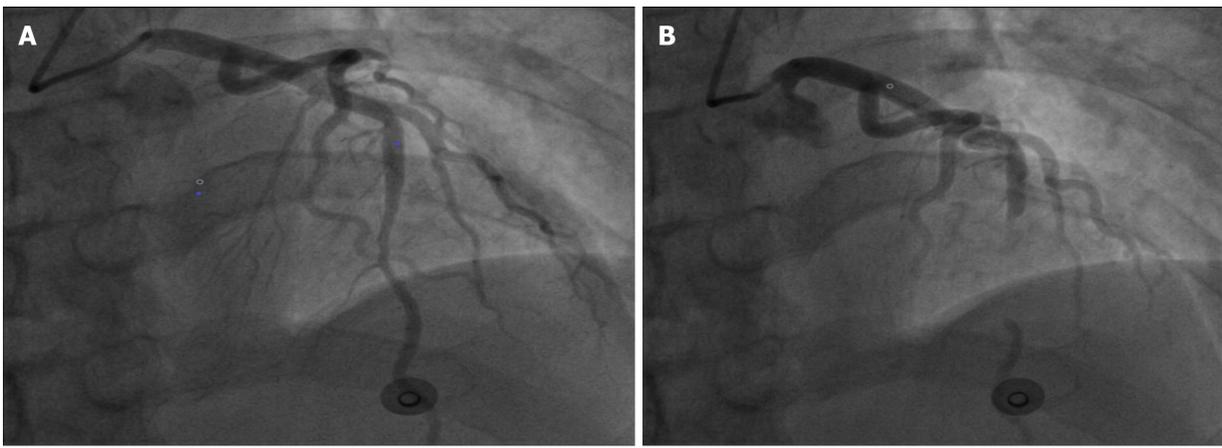
Considering that the symptoms of chest tightness and discomfort may be related to severe stenotic myocardial bridges, intracoronary function evaluation was performed. The pressure was adjusted to 1 when the pressure guide wire entered the root of the aorta. The pressure was measured after the guide wire passed through the diseased segment, and an IFR of 0.72 was measured. Using an adenosine intravenous pump, an FFR of 0.66 was measured after the patient's blood pressure dropped by 10%. The critical values of FFR and IFR were 0.80 and 0.89, respectively. A value less than the critical value indicates that a myocardial bridge caused significant haemodynamic changes. After myocardial bridge surgery, β -blockers were routinely used to reduce the heart rate of the patient. During the operation, we waited for the completion of the basic metabolism of adenosine and injected esmolol 0.02 $\mu\text{g}/\text{kg}/\text{min}$ intravenously. After the heart rate dropped to 60 beats/min (basal heart rate 75 beats/min), changes in coronary blood flow could be observed. The results of the retest were FFR = 0.65 and IFR = 0.83 (Figure 3).

OUTCOME AND FOLLOW-UP

At the end of the operation, the puncture site was pressed with an elastic bandage to stop bleeding. Three hours later, there were no complications, such as blood oozing and haematoma. The patient was discharged 3 h after operation. After discharge, the patient was treated with metoprolol 47.5 mg qd for one month, and the symptoms of chest tightness were significantly relieved after follow-up.

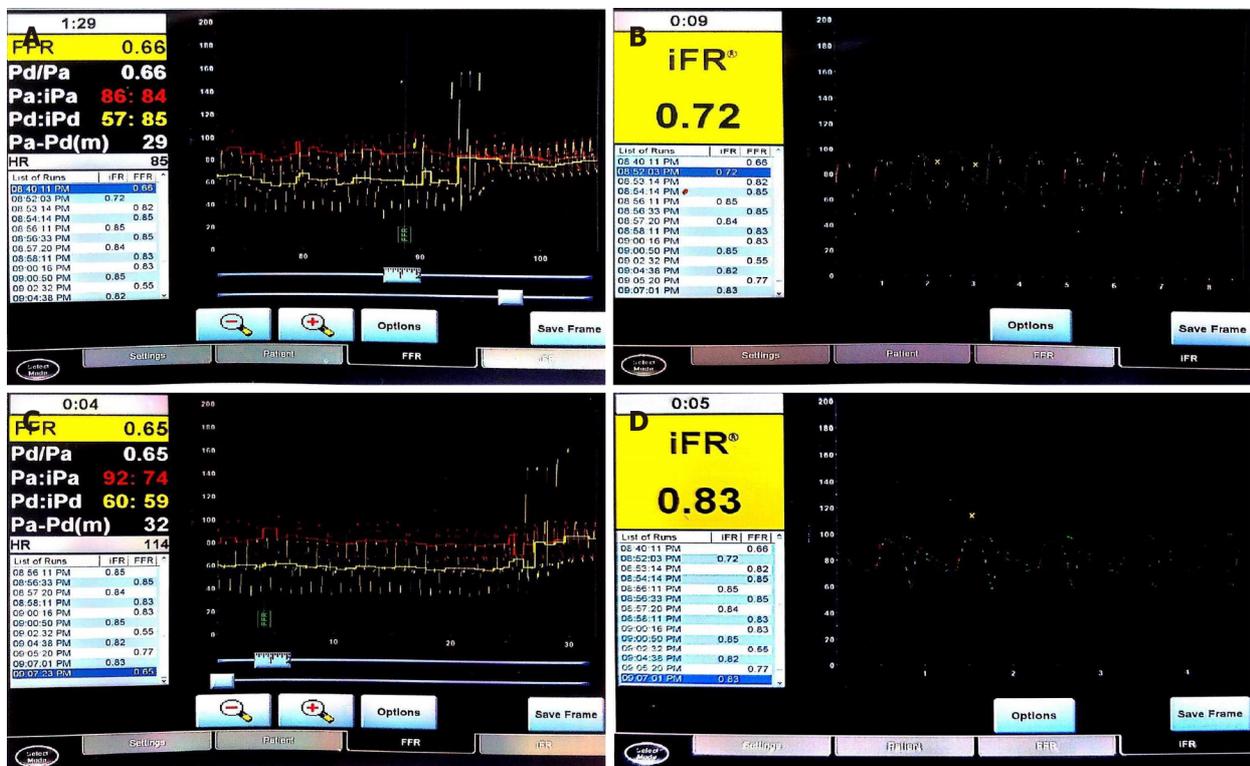
DISCUSSION

The myocardial bridge is a segment of the coronary artery in the myocardium. Long-term studies have suggested that MB only blocks systolic coronary blood flow[7]. However, some studies have reported that MB is associated with stable angina pectoris, acute coronary syndrome and malignant arrhythmias that may lead to sudden death[8]. Therefore, a full understanding of the haemodynamic significance of MB during surgery is of great significance to guide treatment. There have been a large number of studies evaluating the intracoronary function of myocardial bridges. First, Teragawa *et al*[8] used FFR and IFR to confirm that myocardial bridges can cause coronary ischaemia and angina pectoris in



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Figure 2 Coronary angiography results. A: Lumen shape of diastolic anterior descending brac. B: The shape of the lumen of the anterior descending branch during systole.



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Figure 3 Fractional flow reserve results before drug therapy. A: Hyperemia fractional flow reserve is 0.66; B: Resting instantaneous wave-free ratio is 0.72; C: Hyperemia fractional flow reserve is 0.65; D: Resting instantaneous wave-free ratio is 0.83.

patients. Second, Ryan and Escaned[9] measured the FFR of patients with myocardial bridges stimulated by baseline and dobutamine. This study ultimately found that the average FFR measurement increased artificially due to excessive systolic blood pressure, and diastolic FFR should be the first choice. Compared with adenosine, dobutamine seems to be more accurate in evaluating myocardial bridge FFR, highlighting the importance of muscle strength in the development of vascular compression. Third, Klues found that a myocardial bridge causes coronary haemodynamic abnormalities by combining intracoronary Doppler blood flow with pressure measurement. This is characterized by a continuous decrease in diastolic diameter, an increase in blood flow velocity and retrograde blood flow, and a decrease in blood flow reserve. However, there are few reports on the evaluation of the intracoronary function of myocardial bridges after intraoperative drug treatment. In this case, the MB was located near the middle part of the anterior descending branch. Here, the muscle bridge is longer, so the degree of systolic compression is more serious. Angiography also indicated that

the muscle bridge had a systolic compression of 100%. To understand the blood flow changes of severely stenotic myocardial bridges, intraoperative coronary functional evaluation was used. The decrease in coronary flow reserve has a significant inhibitory effect on diastolic coronary blood flow. There are several possible mechanisms for this effect. One such mechanism is that the blood flow in the proximal end of the myocardial bridge with severe systolic compression can be stopped or even retrograde. This results in a partial decrease in the distal perfusion pressure of the bridging vessel and leads to ischaemia[9]. Vascular contraction and compression of the myocardial bridge segment causes turbulence and high shear stress, which leads to the disturbance of vascular endothelial function, an increase in the expression of vasoactive substances and morphological changes in endothelial cells and smooth muscle cells in this region. This leads to self-repair of the vascular endothelium, thickening of the vessel wall, stenosis of the lumen and a decrease in coronary blood flow reserve. The more serious the systolic vascular compression of the myocardial bridge is, the worse the diastolic diameter recovery, the higher the intracoronary filling pressure, and the lower the blood supply rate., All of these conditions seriously affect the main perfusion period of the coronary artery. Another mechanism by which the coronary flow reserve effects diastolic coronary blood flow occurs when the change in diastolic blood flow is more obvious. An obvious abnormality in flow velocity further leads to a decrease in coronary flow reserve. In this case, the coronary blood flow reserve decreased seriously in the severely narrow myocardial bridge, and the symptoms of myocardial ischaemia, such as chest tightness, became more obvious once the heart rate increased. In the face of isolated MB with such severe stenosis, in addition to percutaneous coronary intervention, coronary artery bypass grafting and surgical unroofing, the main treatment is still drugs. To understand the improvement of coronary artery ischaemia in patients with severe myocardial bridge stenosis, esmolol 0.02 µg/kg/min was injected intravenously during the operation. When the heart rate dropped to 60 beats/min, the FFR and IFR were measured again. FFR was 0.65 and IFR was 0.83. The IFR was significantly higher than it was before treatment, suggesting that the coronary flow reserve was better than before. One possible reason is that esmolol inhibits the automaticity of the sinoatrial node, prolongs atrioventricular conduction and reduces heart rate. Thus the diastolic period is prolonged, the time of myocardial bridge blood supply is prolonged, and the myocardial contractility and myocardial oxygen consumption are reduced. The average peak value, diastolic peak value and maximum instantaneous peak velocity in the myocardial bridge were significantly increased[10]. A second possible reason is that β-blockers can also reduce systemic and intramural pressure to reduce vascular compression in vitro, They can also reduce the indirect effect of sympathetic drive, further improve the state of coronary ischaemia, and relieve ischaemic symptoms, such as chest tightness and chest pain, in patients[11]. Other possible reasons could be a decrease in heart rate, decrease in peak blood flow in early diastole, prolongation of diastolic platform, disappearance of reverse blood flow, recovery of normal diastolic blood flow velocity, and relief of coronary flow reserve.

CONCLUSION

For isolated MB with severe stenosis, it is novel to observe the improvement of coronary blood flow after intraoperative drug therapy is added to reduce ventricular rate. At present, the main treatment is still drugs. However, the applicable types of evaluation methods and the effectiveness of long-term treatment need to be further evaluated by large-scale studies.

FOOTNOTES

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