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A Multidisciplinary Approach to Atrial Fibrillation

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Abstract : We report a case with mitral regurgitation and atrial fibrillation and discuss the management of atrial fibrillation. The successful result of combining mitral valve repair and simple pulmonary vein isolation in a single procedure is presented.

Key words : Mitral regurgitation, Atrial fibrillation, Pulmonary vein isolation

เรื่องย่อ : แนวทางการรักษา **Atrial Fibrillation**

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รายงานผู้ป่วย 1 ราย ที่มีลิ้นหัวใจไมตรัลรั่วร่วมกับภาวะ atrial fibrillation อภิปรายแนวทางการรักษาภาวะ atrial fibrillation และรายงานผลความสำเร็จของการผ่าตัดซ่อมลิ้นหัวใจไมตรัลร่วมกับการผ่าตัดแก้ไขภาวะ atrial fibrillation

INTRODUCTION

Atrial fibrillation (AF) is a common arrhythmia associated with both morbidity and mortality and is often found in association with mitral valve disease. There are several methods available for its management including medication and surgery. The Maze procedure is a surgical operation which is usually performed to eliminate AF in cases with mitral valve disease[1]. However, the Maze procedure is a complicated operation, needs sophisticated equipment and demands a high level of experience. Recently, Haissaguerre and coworkers reported a focal source of paroxysmal AF originating from the pulmonary vein[2], and suggested this to be a focal source in cases with paroxysmal and persistent AF. On this basis, we performed a simple pulmonary vein isolation during mitral valve surgery to eradicate chronic AF.

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CASE REPORT

A sixty three years old man presented with a history of dyspnea on exertion for 4 years with worsening over the previous 10 months. He occasionally had symptoms of chest pain and palpitation. Physical examination showed a totally irregular pulse rate at 110 beats/minute, a pansystolic murmur grade III at the apex which radiated to the axilla and left lower parasternal border, with an increased P2 sound, a right ventricular heave, normal breath sound in both lungs, a just palpable liver and mild pitting edema in both legs. Chest X-ray revealed cardiomegaly which could indicate left atrial and left ventricular enlargement. The ECG showed atrial fibrillation. Transthoracic and transesophageal echocardiography demonstrated severe mitral valve regurgitation due to prolapse of the posterior mitral leaflet and ruptured chordae of the PM1 and PM2 segments. The size of the left atrium was 77.5 mm. Coronary angiography was normal. In conclusion, this patient had both severe mitral valve regurgitation and atrial fibrillation.

The pathophysiology of atrial fibrillation[3,4]

The core mechanism of atrial fibrillation is multiple small re-entry circuits. In order for circular conduction circuits to be established, the action potential duration (APD), the effective refractory period (ERP) and conduction velocity are altered. A decrease in the APD, which contributes to a shorter ERP, makes cardiomyocytes available for activation by another action potential from adjacent tissue resulting in the formation of a re-entry loop. A slower conduction velocity is also responsible for the re-entry mechanism since it delays the action potential propagation until the action potential of the adjacent area is complete. The faster pathway that becomes available for reactivation at this time is stimulated, and conveys the conduction retrogradely to form a re-entry loop.

Recently acquired knowledge helps understand the progression of atrial fibrillation from occurring paroxysmally to becoming persistent. These studies lead us to the mechanisms of atrial fibrillation that involve triggers and substrates. The trigger initiates atrial fibrillation and conducts the changes (called substrates) in both the electrophysiological properties and the structure of the myocardial wall. It has been shown that the pulmonary vein could be the source for triggering atrial fibrillation. Furthermore, some clinical conditions, e.g. mitral regurgitation, may cause the late development of atrial fibrillation.

In this patient, mitral regurgitation causes volume overload, which produces a stress on the atrial wall. Due to the heterogeneous character of the atrium, the stress on its wall is not homogeneous, resulting in differences in the intracellular alterations of atrial cardiomyocytes. This stress activates the stress-operated channels on the cardiac sarcolemma, producing the ion influx. Beside activating the stress-operated channel, there are evidences in the atrial fibrillation-induced animal and the patient suffering from persistent atrial fibrillation that the kinetic properties of some ion channels are changed in AF. The L-type calcium channels and the transient outward potassium channels are the targets of this change, which is known as electrical remodeling. The decrease in the L-type calcium current provides faster repolarization, establishing a shorter action potential duration and effective refractory period. The transient outward potassium current is also decreased. Though this decrease actually increases the duration of the action potential, the heterogeneity of the effect in myocytes along the conduction pathway provides a non-homogeneous APD which increases the chance of developing atrial fibrillation. In the heart with chronic atrial fibrillation, the atrial wall shows a fibrotic pattern. The myocytes in the fibrotic heart establish slower conduction, probably due to the loss of continuity of the branching connections. Furthermore, the contraction generated by the fibrotic wall provides an uneven tension in the whole atrial wall, which in turn drives the myocardial wall into the electrical and structural remodeling as described above.

Though there is no evidence concerning apoptosis in the atrial fibrillation-induced animal, some small apoptotic lesions have been found in the human heart suffering from chronic atrial fibrillation. These focal apoptotic lesions develop uneven myocardial contraction and the uneven stress on the atrial wall as well. Since cardiomyocytes are cells that cannot be regenerated, the apoptotic lesions

contribute to the irreversible changes in cardiac structure.

Management of atrial fibrillation

In general, there are factors that need consideration when one encounters a patient with atrial fibrillation (AF). The urgency in managing the AF patient largely depends on the hemodynamic situation. With a stable AF patient, the factors influencing the treatment options are : -

1. The causes of AF
2. Prevention of thromboembolic complications by chronic anticoagulation or antiplatelet medication
3. Control of the heart rate which might be achieved by controlling the ventricular response to AF only, or by controlling the rhythm (try to maintain sinus rhythm).

Causes of atrial fibrillation

As we know, AF is the most common cardiac arrhythmia and there are a number of different causes of AF[5]. However, we can classify the causes of AF as cardiac and non-cardiac related as shown : -

Cardiac causes of atrial fibrillation

Hypertensive heart disease
Ischemic heart disease
Valvular heart disease
- Rheumatic: mitral stenosis
- Non – rheumatic: aortic stenosis, mitral regurgitation

Pericarditis
Cardiac tumor: atrial myxoma
Sick sinus syndrome
Cardiomyopathy
- Hypertrophic
- Idiopathic dilated (cause vs. effect?)

Post-coronary bypass surgery

Non-cardiac causes of atrial fibrillation

Pulmonary
- COPD
- Pneumonia
- Pulmonary embolism

Metabolic
- Thyroid disease: hyperthyroidism
- Electrolyte disorder
- Toxic:alcohol("holiday heart" syndrome)

The term "lone atrial fibrillation" is used when there is no identifiable cardiovascular, pulmonary, or associated systemic disease. Finding treatable causes of AF, such as hyperthyroidism, is crucial in the management of the AF patient.

In this patient, the obvious cause of AF is severe mitral regurgitation. However looking for signs and symptoms of a treatable cause of AF is still mandatory.

Prevention of thromboembolic complications in the patient with atrial fibrillation

The thromboembolic complications of AF are well known[6,7]. Studies have already shown the benefit of chronic anticoagulation to prevent thromboembolic complications in AF patients[8,9].

	SPAF		BAATAF	CAFA	AFASAK		SPINAF
Number of patients	1330		420	378	1007		571
Drug used	Warfarin (INR 2-4.5)	ASA 325 mg	Warfarin (PT 1.2-1.5 x Control)	Warfarin (INR 2-3)	Warfarin (INR 2-4.5)	ASA 75 mg	Warfarin (INR 1.2-1.5)
Embolic rate (%)							
Treatment	2.3	3.6	0.41	3.5	1.5	6.0	4.3
Control	7.4	6.3	2.98	5.2	6.2	6.2	20.9
Risk reduction (%) (95% confidence)	67	42	86	4.5	---	---	79
Major bleeding complications (%)							
Treatment	1.5	1.4	0.9	2.5	6.3	0.6	1.5
Control	1.6	1.9	0.5	0.5	0.0	0.0	0.9

It is recommended that AF patients who are at risk of thromboembolic complications should be put on chronic anticoagulation unless there is a contraindication. In these cases antiplatelet medication such as aspirin should be considered.

In this patient, the type of mitral valve surgery would influence the decision to anticoagulate. If metallic mitral valve replacement is planned, chronic anticoagulation is mandatory. If mitral valve repair or tissue valve replacement is planned, long term anticoagulation can be avoided. This factor would influence the AF treatment in that one would try to obtain normal sinus rhythm and avoid the need for long term anticoagulation.

Heart rate control in atrial fibrillation

Most AF patients are presented with a rapid ventricular response. In a hemodynamically stable situation, heart rate control in AF may be produced with control of the ventricular response and both atria remain fibrillating. The other approach is to produce and maintain normal sinus rhythm.

The most widely used method to control the ventricular response in AF is by medication[10,11]. In a refractory situation, atrioventricular nodal modification with radiofrequency ablation and atrioventricular nodal ablation with permanent pacemaker can be considered[12-14].

To obtain and maintain normal sinus rhythm in AF patients is challenging. This goal may be achieved with chronic antiarrhythmic medication and sometimes combined with direct current cardioversion. This approach is well accepted. However the high recurrence rate and side effects are the main limitation. Recently, the use of radiofrequency catheter ablation and surgery in AF patients are evolving and promising[15-17].

In the patient needing mitral valve surgery and in whom we need to avoid chronic anticoagulation, AF surgery would be the treatment option.

Surgical approach to atrial fibrillation

In this case, we are dealing with two problems. The first is severe mitral regurgitation and the second is chronic AF. The treatment of choice for severe mitral regurgitation is mitral valve repair because left ventricular function is preserved better than in mitral valve replacement, and long-term anticoagulant can be avoided.

We were also determined to eliminate AF in order to decrease the risk of postoperative thromboembolism and to restore atrial function. Therefore, we performed a simple pulmonary vein isolation with mitral valve repair in this patient.

The operative technique

We approached via a median sternotomy. The patient was placed on bicaval aortic bypass and cold blood cardioplegia was given after aortic clamping. Left atriotomy was performed and the superior vena cava was transected for good exposure. We extended the left atriotomy incision on the left side and completely divided it from the pulmonary veins. The mitral valve was repaired using quadrangular resection and a sliding plasty technique. Mitral annuloplasty was done using a 34 mm Carpentier-Edward ring. The free edge of the left atrium was anastomosed to the isolated posterior veins using running sutures. The patient came off cardiopulmonary bypass uneventfully. Sinus rhythm resumed immediately after surgery.

RESULTS

The patient's condition was gradually improved. A week after operation, transthoracic echocardiography revealed trivial MR, mild TR, mild pulmonary hypertension and good left ventricular contraction. The left atrial size had decreased to 49 mm, but left atrial contraction could not be demonstrated. The postoperative ECG showed sinus rhythm without recurrence of atrial fibrillation. The patient remained in sinus rhythm at the 4-month follow-up and had a functional grade class I.

DISCUSSION

Atrial fibrillation is one of the most prevalent of all arrhythmias, being present in 30% of patients at the time of mitral valve repair[18]. In this patient, we were concerned with two problems, mitral regurgitation and atrial fibrillation. Mitral valve repair is the preferred procedure for most patients

with mitral regurgitation as a result of degenerative disease. There are many advantages over valve replacement as a result of valve preservation including preserved left ventricular function, reduced risk of thromboembolism and avoidance of anticoagulation. However, 80% of patients with preoperative chronic atrial fibrillation in whom mitral valve repair is performed still have atrial fibrillation at the 5-year follow-up[18]. To eliminate atrial fibrillation, we performed a combined procedure in this patient. The various surgical procedures that have been described include the maze procedure[19] and pulmonary vein isolation. The maze procedure has cured atrial fibrillation, restored atrioventricular synchrony and preserved atrial transportation in 98% of patients[20], but the procedure is complicated, technically demanding and needs special equipment. A simple and effective procedure is pulmonary vein isolation which has demonstrated good results in restoring sinus rhythm in 92.3% of patients at 6-month follow-up[21]. Another report presented a successful result in 75% of chronic AF patients treated by a simple surgical isolation of the pulmonary vein orifices[22].

We report a successful combined procedure of mitral valve repair and pulmonary vein isolation in the management of a patient with severe mitral regurgitation and atrial fibrillation. We believe that pulmonary vein isolation is a simple and effective approach to eliminate atrial fibrillation in the patient with mitral valve disease.

COMMENT

Atrial fibrillation is a common arrhythmia often found in mitral valvular disease which causes many problems especially cerebral thromboembolism which results in death or permanent severe neurological deficit. There are many methods of managing atrial fibrillation including medication, intervention and surgery. Appropriate treatment should depend on many factors such as the patient's condition, associated disease and available resources. In this case, the severe mitral regurgitation is an indication for surgery and the pulmonary vein isolation is a combined procedure to eradicate atrial fibrillation. This surgical option is a simple procedure, does not demand sophisticated instruments and shows a good result. However, this is only an early follow-up (4 months) and so long-term follow-up is needed.

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