Review article

Traumatic mitral valve regurgitation: a case report and state-of-the-art review

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Traumatic mitral valve regurgitation is a rare and often insidious condition. Clinical presentation is variable and influenced by the anatomic structures injured; when papillary muscles are damaged, the clinical presentation is often acute, whereas, in the case of involvement of other anatomic structures of the valvular apparatus (e.g. chordae tendinae), the onset of symptoms may be delayed (days, weeks, or months). Therefore, diagnosis may be belated because of the heterogeneous clinical presentation. Traumatic mitral valve injury should be excluded in patients admitted to the emergency services with blunt chest trauma, in particular when signs or symptoms of acute heart failure occur. Echocardiography, particularly with the transoesophageal approach, may play a pivotal role in this setting. Herein, we present a case of severe mitral regurgitation because of blunt chest trauma and a systematic review of the literature. We examined 192 described cases, classified according to epidemiology, aetiology, anatomic features, clinical presentation, diagnosis, surgical/clinical management and prognosis.

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Introduction

Traumatic mitral valve injury after a blunt chest trauma (BCT) is an uncommon occurrence. Despite this, its relevance in clinical practice is mainly because of the variable clinical presentation, often hampering prompt diagnosis.

The most common cardiac injury observed after BCT is myocardial contusion, occurring in 16–76% of patients.[1] Different patterns of traumatic cardiac injury have been described (e.g. myocardial contusion, free wall rupture, atrial septal defect, valve injury, ventricular septal defects, coronary arteries injury).[2] Concerning valve involvement, the aortic valve is most commonly affected, followed by the mitral and tricuspid. Among mitral valve injuries, ruptures of the chordae tendineae and papillary muscles are the most common features, followed by tears of the posterior and/or anterior leaflets.[3]

BCT usually occurs after direct impact on the anterior chest caused by high-speed sudden deceleration or compression of the thorax, which is a typical result of motor vehicle accidents, the most frequent cause of chest trauma.[4]

In 1936, Glendy and White[5] reported the first mitral traumatic rupture after closed thoracic trauma.

Although the real prevalence of traumatic mitral valve injuries is probably underestimated, there has been an increase in their incidence probably because of the raising number of motor vehicle accidents[4] and the improvement of diagnostic accuracy. The peculiarity of this condition is its variable clinical presentation, often masked in the context of a polytrauma. A correct and prompt diagnosis in this context may be difficult to achieve, with remarkable clinical consequences.

The aim of the present article is to give an update of this often neglected clinical condition.

Clinical case

A 49-year-old man experienced a workplace accident because of an industrial tyre explosion, which caused BCT and multiple fractures (facial, cranial, thoracic and pelvic area). The patient was urgently referred to a local medical center. Urgently after hospital admission, the patient underwent orthopaedic surgery to stabilize bone injuries. His medical history before the chest trauma was unremarkable. The only cardiovascular risk factors were arterial hypertension and former cigarette smoking. Physical examination showed no cardiac murmurs and transthoracic echocardiography pointed out a prolapse of the posterior leaflet of the mitral valve with mild-to-moderate regurgitation. Due to these preliminary results, further assessment with transoesophageal echocardiography was deemed unnecessary.

After a 2-month hospitalization, when the bone fractures were healing, the patient was discharged.

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However, as soon as the patient started standing and walking, he experienced dyspnoea for mild efforts (NYHA functional class III) and was, therefore, re-admitted to the hospital. Clinical examination showed a new holosystolic murmur at apex.

Transthoracic and transoesophageal echocardiography revealed severe mitral regurgitation because of a flail of a mildly myxomatous posterior leaflet (P2 and P3 scallops). Therefore, the patient was referred to our hospital for further assessment of the valvular disease and surgical correction. Echocardiographic findings were confirmed (flail of the posterior leaflet of the mitral valve – P2 scallop, because of chordal rupture) [Fig. 1a–c].

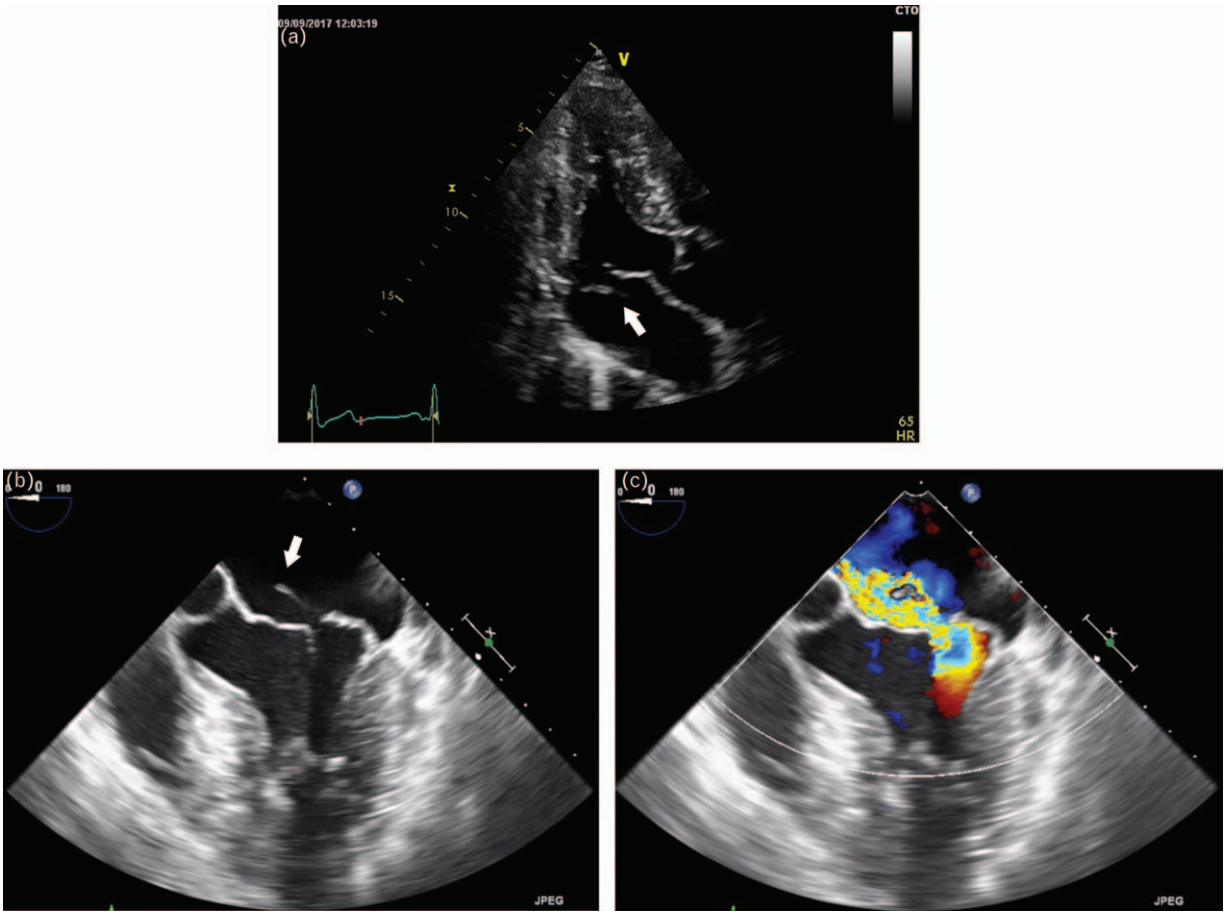
The patient underwent surgical mitral valve repair with triangular resection of the P2 scallop, sliding of the P2-P3 scallops, transposition of two II degree chordae, artificial chordae implantation and annuloplasty. A subsequent echocardiogram showed no residual regurgitation. The hospital stay was uneventful and the patient was discharged in good clinical condition.

The present case deserves some considerations. First, it emphasizes the importance of a comprehensive cardiological evaluation including repeated echocardiograms in patients with polytrauma, because of the insidious and often delayed presentation of the mitral valve injury. Second, a preexistent degenerative mitral valve disease may predispose to serious damage following a thoracic trauma. Third, the effects of mitral injury may be subacute. Finally, in patients with polytrauma, the prolonged hospitalization and being bedridden may contribute to the masking of symptoms, further delaying the diagnosis.

Methods

All cases described in the literature until July 2018 (without restriction for date or language) were collected using the PubMed search engine, with the keywords 'traumatic mitral regurgitation', 'traumatic mitral insufficiency' and 'traumatic mitral valve rupture'. Reference lists were examined for additional articles. Both reviews and case reports of mitral valve regurgitation after CBT were included. Iatrogenic and penetrating mitral valve

Fig. 1



(a) Transthoracic echocardiography (apical three-chamber view) showing a flail of the posterior leaflet of the mitral valve (white arrow). (b and c) Transoesophageal echocardiography (mid-oesophageal view) confirming the presence of a flail leaflet and severe mitral regurgitation with color Doppler examination.

injuries were also collected for completeness, but not analyzed.

One hundred and ninety-two cases of traumatic mitral valve injury after blunt trauma were collected,[4–110] including 111 cases with available information and another 51 cases without any accessible data (33 autoptic findings reported by Parmley *et al.*[111] and 48 cases described by Reardon *et al.*[112]). Ten more cases of valve damage because of a penetrating trauma and 56 cases of iatrogenic mitral valve injury were reported. Data were classified according to the features of population, clinical presentation, symptoms onset and timing of diagnosis, mechanism of trauma, types of mitral valve injury and of surgical intervention, as reported in Table 1.

Table 1 Traumatic mitral valve regurgitation

Cause (number of cases)	
Blunt chest trauma	192
Penetrating injuries	10
Iatrogenic	56
Blunt chest trauma cases	
Sex [*n* (% of 111)]	
Male	70 (63%)
Female	14 (13%)
Not specified	27 (24%)
Age at diagnosis (years)	
Mean	32
SD	16
Range	2–75
Time to symptoms onset (days)	
Mean	119
SD	±428
Range	0–7 years
Time to diagnosis (days)	
Mean	136
SD	±448
Range	0–7 years
Mechanism of blunt trauma [*n* (% of 111)]	
Road accident	57 (51%)
Motorcycle accident	15 (14%)
Fall from great heights	9 (8%)
Object impact	15 (14%)
Not specified	15 (14%)
Clinical presentation [*n* (% of 111)]	
Shock	31 (28%)
Dyspnea	27 (24%)
Pulmonary oedema	11 (10%)
Asymptomatic/new murmur	8 (7%)
Cough	1 (1%)
Not specified	33 (30%)
Valve lesion [*n* (% of 111)]	
Papillary muscles	51 (46%)
Anterolateral	22/51
Posteromedial	15/51
Both	3/51
Unknown	10/51
Complete rupture	22/51
Partial rupture	3/51
Necrosis	2/51
Chordae tendineae	26 (23%)
Direct damaged leaflets	12 (11%)
LMA	2/12
LMP	5/12
Not specified	5/12
Annulus	8 (7%)
Other valves	13 (12%)
Coronary lesion	6 (5%)
Mitral valve regurgitation entity [*n* (% of 111)]	
Mild	0 (%)
Moderate	10 (9%)
Severe	63 (57%)
Not specified	38 (34%)
Type of surgery [*n* (% of 111)]	
Mitral valve repair	36 (32%)
Annuloplasty	9 (%)
Mitral valve replacement	36 (32%)
Mechanical valve	20/36
Bioprosthesis	11/36
Not specified	5/36
Not specified treatment	37 (33%)
Death [*n* (% of 111)]	5 (4%)

Results

Among a total of 111 cases analyzed, sex was available in 84 patients, with 83% being men. The average age at diagnosis was 32 ± 16 years. The most common cause of traumatic mitral valve injury was a traffic accident (57 cases; 51%), often involving a motorcycle (15 cases; 14%). Nine (8%) cases were because of falls from great heights and another 15 (14%) cases were secondary to high-speed impact. The time interval between trauma and mitral valve disease diagnosis was variable (ranging from 24 h to 7 years) and generally remarkable (mean 136 ± 448 days). The most common clinical presentation was cardiogenic shock (31 cases; 28%), followed by dyspnoea and progressive heart failure (27 cases; 24%). In 11 (10%) cases, the mitral injury was associated with acute pulmonary oedema, whereas, in 8 (7%) cases, patients were completely asymptomatic, and the diagnosis was suspected by a new cardiac murmur detection. In one case, the only symptom was cough.

With regard to the entity of valvular dysfunction, in 63 (57%) cases, the grade of regurgitation was severe and in 10 (9%) cases moderate. The most common injuries were represented by papillary muscles ruptures (51 cases; 46%), causing a valve prolapse in 17 cases and a flail leaflet in 23 cases (both classified as Carpentier type 2 because of increased mobility of the leaflets). Of the 51 (46%) reported papillary muscle lesions, 22 involved the anterolateral, 15 the posterolateral, and in 3 cases both of them. The papillary muscle rupture was complete in the majority of cases (22) and rarely partial (3), involving the head of the muscle, whereas, in 2 cases, necrosis of the papillary muscle was reported. Damaged chordae tendineae occurred in 26 (23%) cases. In 12 (11%) cases, clefts and tears of valve leaflets have been reported, involving mainly the posterior leaflet (5 cases), and less frequently the anterior leaflet (2 cases), whereas, in 5 cases the injured leaflet was unspecified. In eight (7%) cases, the annulus was the only damaged structure.

In 36 (32%) cases, other concomitant cardiac lesions were described. Indeed, in 15 (13%) patients, other valves were injured, namely the aorta in four cases and tricuspid valve in nine cases. Six concurrent coronary lesions have been described. Other concomitant cardiac injuries were myocardial contusion, ventricular free wall rupture, pericardial lesions, ventricular and atrial septal defects, aortic dissection. In two cases, the traumatic rupture involved a mitral valve with previous annuloplasty.

Concerning the surgical treatment, 27 (24%) patients underwent valve repair. Out of 36 (32%) cases of valve replacement, 31 mechanical and 20 biological prosthesis have been reported. In five cases, valve prosthesis was unspecified. Death occurred in five (4%) patients.

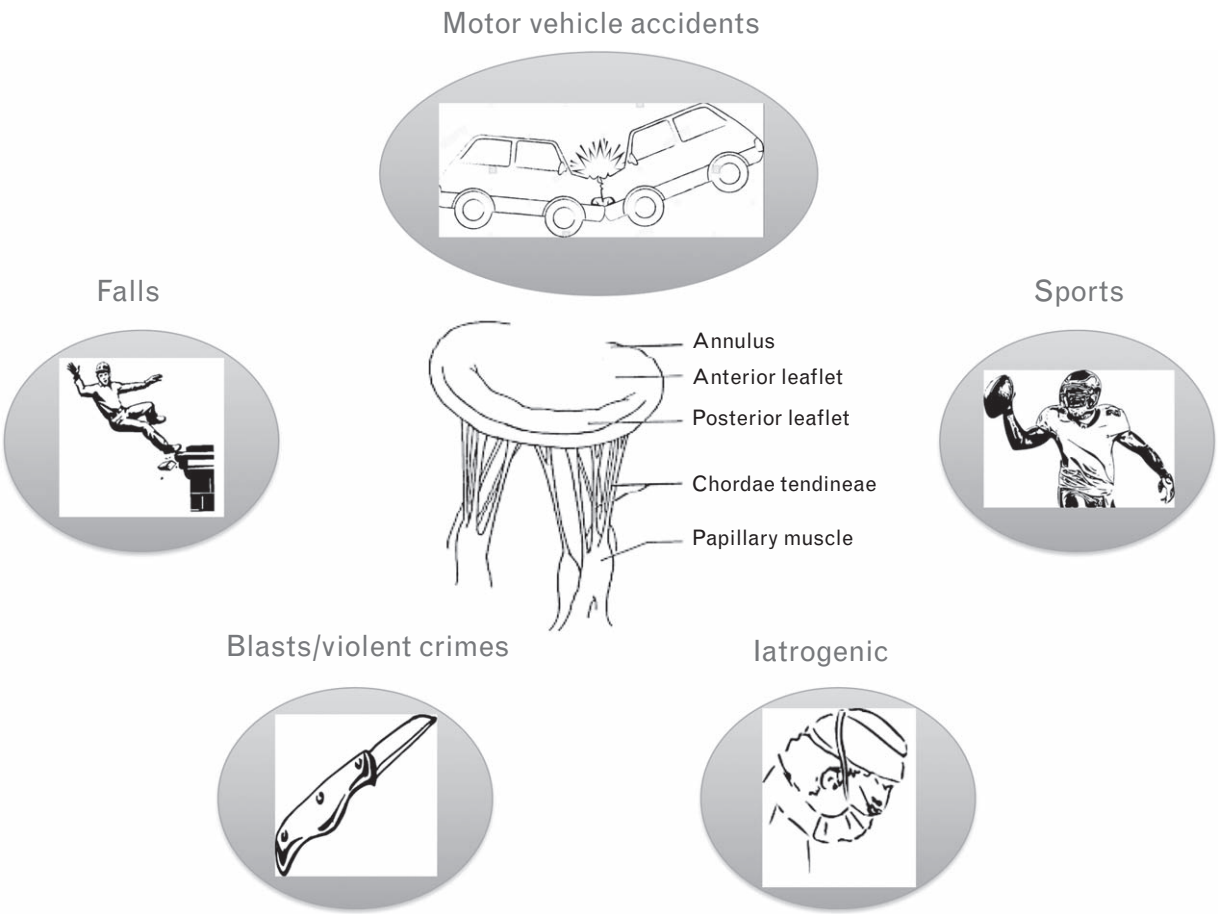
Epidemiology and cause of traumatic mitral regurgitation after blunt chest trauma

BCT involving mitral valve usually occurs after a traffic accident, most commonly car crashes, followed by motorcycle accidents. Other mechanisms less frequently described are falls from heights, work accidents, and animal kicks.[113] According to our literature review, traffic accident was the most common finding, but several other scenarios including a variety of falls and high-speed impacts have been reported. For example, blast injuries should be taken into account in people living in war scenarios. Finally, athletes engaged in sports with potential bodily contact could be at risk for accidental mitral valve injuries (Fig. 2). Sometimes mitral valve regurgitation could be the result of a Takotsubo stress syndrome following the chest blunt trauma. In these circumstances, there is not a direct injury of the mitral valve apparatus and the functional regurgitation recovers within a short time.[114]

Mechanisms and pathophysiology of mitral regurgitation secondary to blunt chest trauma

Traumatic mitral valve rupture secondary to BCT usually occurs as a result of high energy transmission, such as sudden deceleration or compression of the heart and subsequent abrupt increase of intracardiac pressure. The most vulnerable phases of the cardiac cycle with respect to the mitral valve are end-diastole and early systole, when atrioventricular valves are closed. The occurrence of this phenomenon during these phases may lead to compression of the distended ventricles in the former and further pressure during isovolumetric contraction in the latter. The massive wall stress transmitted to the left ventricle may lead to damage of papillary muscles, chordae tendineae and mitral leaflets. Experimental studies have demonstrated that intraventricular pressures greater than 320 mmHg can cause rupture of any cardiac structure.[115] The contusion or necrosis

Fig. 2



Vignette showing the main causes of traumatic mitral valve regurgitation.

of a papillary muscle may be another responsible mechanism. Finally, mitral valve damage can be the result of a coronary artery trauma/dissection with subsequent ischemia of a papillary muscle, or the consequence of an initial limitation to papillary muscle blood supply (leading to) progressive ischemic necrosis and late rupture.[116]

Clinical presentation of traumatic mitral injury: the chameleon

Traumatic mitral valve injury because of BCT is characterized by a complex clinical scenario with variable presentation, ranging from sudden death to asymptomatic patients receiving a delayed diagnosis. In our review, we considered the latency between the BCT and the mitral valve disease diagnosis. The detection of mitral valve injuries is often belated, as shown in Table 1. This might be because of the chameleon clinical spectrum, which often misleads physicians. Indeed, acute and severe clinical presentation usually led to a more or less prompt detection of mitral damage, whereas mild or late symptoms often resulted in misdiagnoses or great diagnostic delays. The development of a cardiac trauma does not seem to be directly related to the intensity of the chest wall impact.[117] On the contrary, clinical presentation is influenced by the injured valve component. Symptoms and signs secondary to the rupture of a papillary muscle are dramatic; patients usually present hemodynamic instability, pulmonary oedema, or progressive congestive heart failure, whereas the rupture of chordae tendineae tends to present with delayed clinical manifestations. Symptoms may be absent for weeks or even years, as the degree of regurgitation tends to worsen over time. For instance, a patient may develop pulmonary oedema 1 or 2 weeks after a CBT, as an initially mild mitral regurgitation may progress into a severe symptomatic valve failure. In the literature, the most frequent clinical presentation was hemodynamic shock, followed by dyspnoea and pulmonary oedema. Critical conditions at admission were usually related to a complete papillary muscle rupture with acute severe mitral regurgitation. Asymptomatic murmurs (e.g. loud holosystolic murmur at the apex and radiating to the axilla) were detected in just a few cases, mostly in cases with delayed presentation and mainly related to chordae tendinae, leaflets or annulus damage.

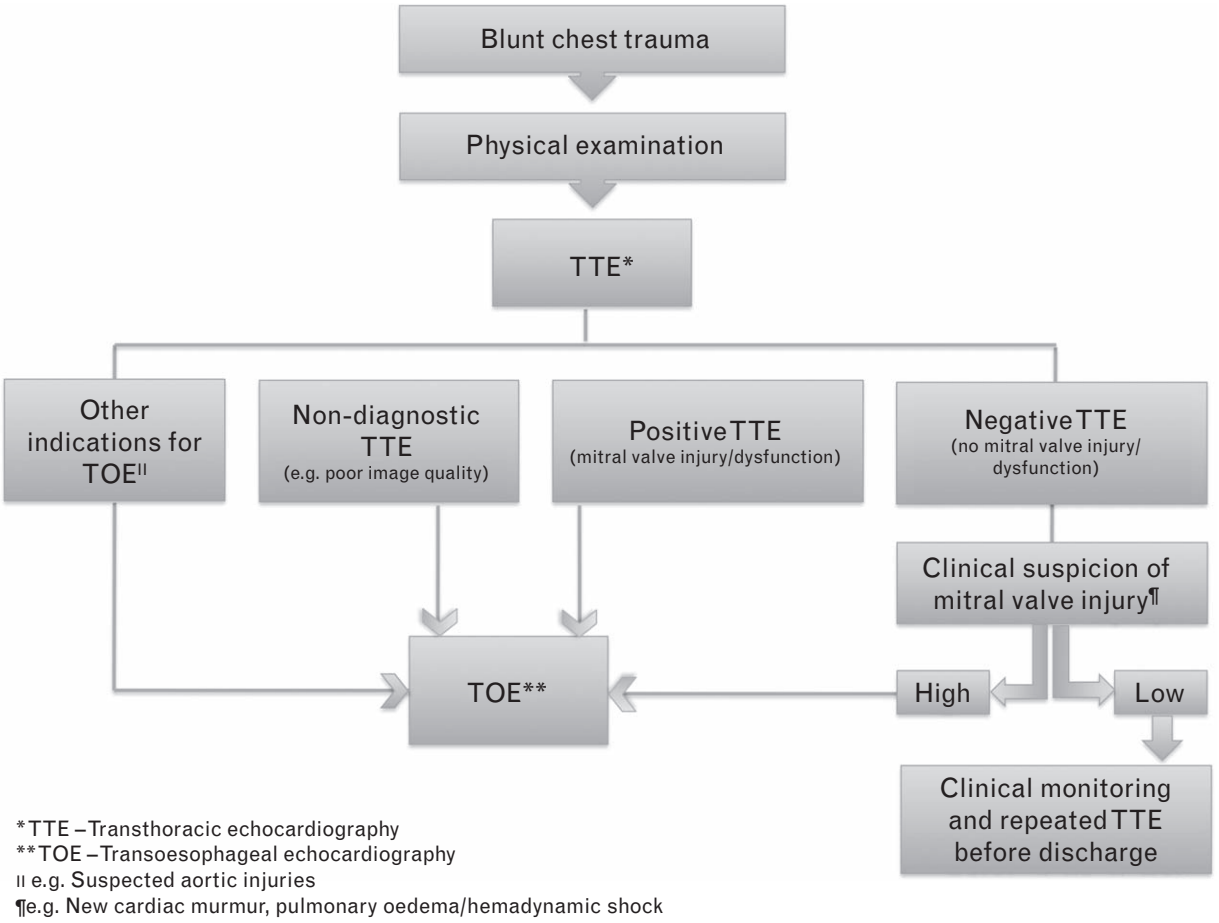
Diagnosis of traumatic mitral valve injuries: the pivotal role of echocardiography

There is currently no gold standard for the diagnostic process of mitral valve injuries following a BCT. A comprehensive clinical evaluation, including fast but accurate patient medical history (trauma dynamics, history of mitral valve disorders), physical examination (including cardiac auscultation), chest X-ray and a 12-lead ECG are advisable for all patients after a BCT. An increase in cardiac enzymes may suggest a cardiac damage, despite the lack of specificity of Troponins in this context. Echocardiography has a pivotal role in this context for the diagnosis of mitral valve injuries.[118] Transthoracic echocardiography (TTE) is the first line technique in this clinical scenario. The technique is fast, widely available, and it provides several parameters about the hemodynamics in patients after a thoracic trauma.

Transoesophageal echocardiography (TOE) allows a more accurate assessment of cardiac lesions.[119] In fact, patients who experience a BCT often present to the hospital emergency room or to the ICU in critical condition. In this context, an accurate TTE is not always feasible owing to thoracic wall bruising or fractures resulting in substantial pain.[16] Suboptimal TTE may also be because of high thoracic impedance in the context of pulmonary oedema, but sometimes related to pneumotorax or subcutaneous emphysema. When there is the clinical suspicion of valve damage, TOE is mandatory to provide a comprehensive diagnosis and define the anatomic features responsible for the mitral valve dysfunction and eventually other associated injuries (i.e. aortic dissection/rupture). TOE is usually feasible in this clinical scenario (patients often under general sedation and/or invasive ventilation), and it is of paramount importance for planning and guiding the surgical treatment. Three-dimensional transoesophageal echocardiography (3D-TOE) may have a pivotal role for assessment of mitral valve functional anatomy before cardiac surgery, especially in patients with prolapse-related lesions.[120] 3D-TOE is more accurate than two-dimensional (2D) TOE in this setting, and it correctly identifies the prolapsed segments in good agreement with surgical inspection.[17] In this regard, real time 3D-TOE may be even more sensitive than surgical inspection in recognizing secondary prolapsing lesions; in fact surgical inspection is performed with an arrested heart, and therefore, those dynamic mitral valve lesions assessed in awake patients might not be recognized.[120] Figure 3 shows a proposed diagnostic algorithm for the use of echocardiography in patients with BCT in order to exclude the presence of a mitral valve injury.

Among the other imaging techniques, cardiac magnetic resonance (CMR) seems to be promising in the assessment of mitral regurgitation. CMR has widely acknowledged advantages in evaluating left ventricular size and function. Furthermore, it has an invaluable role for tissue characterization. Finally, because of the potential inaccuracy of echocardiography in the quantification of the severity of mitral regurgitation (poor-quality imaging, potential over-estimation of nonholosystolic Doppler profiles, measurements errors with Doppler parameters, questionable reliability of flow-convergence methods), CMR should be more reliable in quantifying regurgitant volumes, and thus the severity of the disease. However, restricted spatial and temporal resolution remains a limiting factor in defining mitral apparatus anatomy with CMR. At the moment, there are no studies describing the

Fig. 3



Proposed diagnostic algorithm for the use of echocardiography in patients with blunt chest trauma in order to exclude the presence of a mitral valve injury.

use of CMR in traumatic mitral valve regurgitation. We may speculate that its role in this setting should be further limited because of the patients' characteristics (e.g. poor compliance and difficult achievement of breath-hold images and long examinations, medical/ orthopaedic supports after a polytrauma making patients unable to enter the MR scanner and staying in a supine immobile position).[121]

Surgical treatment

The timing of surgical intervention is often dictated by the patient's condition. Haemodynamic instability requires emergency intervention. On the contrary, stable patients may undergo elective surgery. Several factors including the damaged structures and the complexity of valve lesions influence surgical treatment (e.g. valvular repair vs. replacement). In this context, the choice between biological or mechanical prostheses, and even the surgical approach (open surgery vs. minimally invasive), depends on several factors, namely the age of the patient (generally very young), global clinical conditions, the need for other major noncardiac surgery, bleeding risk and the presence of concomitant coronary lesions.

Prognosis

When promptly recognized and surgically treated, traumatic mitral injury has a good prognosis. Death occurs in patients presenting in critical clinical conditions, and the decease is mostly related to other cardiac damage or concomitant disease.

Conclusion

Although being of rare occurrence, the clinical relevance of traumatic mitral valve injury is mainly related to its variable presentation, which can lead to a misdiagnosis or late diagnosis in several cases, with unfavorable consequences. The variable and often insidious presentation is strictly related to the involved structures of the mitral valve apparatus and to the presence of other concomitant cardiac lesions. A careful assessment is required in patients with BCT, often occurring in a context of polytrauma, when other obvious severe injuries may divert

the attention of the treating physician. An accurate and complete echocardiographic evaluation, in particular, with a transoesophageal approach, is of paramount importance in these patients, not only at the time of hospital admission but even during the follow-up period.

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Conflicts of interest

There are no conflicts of interest.

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