AMERICAN COLLEGE OF EMERGENCY PHYSICIANS
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Managing Valvular Emergencies

The presentation of a patient with prosthetic valve failure can dilate the sphincters of the most seasoned emergency physician. This course reviews the presentation, diagnosis (including audio of the auscultatory findings), and treatment of acute valvular emergencies, including endocarditis and a clotted prosthetic valve. How and when to reverse anticoagulation in these patients and when it can be restarted will also be discussed.

- Recognize the presentation of common valvular emergencies, including endocarditis and clotted prosthetic valves.
- Review the diagnostic approach to these problems.
- Discuss the treatment options available in the emergency department, including the reversal of anticoagulation.

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FACULTY

David FM Brown, MD, FACEP

Instructor, Division of Emergency Medicine, Harvard Medical School; Core Faculty, Harvard Affiliated Emergency Medicine Residency; Assistant Chief, Emergency Medicine, Massachusetts General Hospital, Boston, Massachusetts

American College of Emergency Physicians
1999 Scientific Assembly
Managing Valvular Emergencies

Instructor: David F. M. Brown, MD, FACEP

MANAGING VALVULAR EMERGENCIES

I. Critical Aortic Stenosis

A. Definition/Pathophysiology
   1. "critical" AS: valve area < 1 cm^2 or pressure gradient >50 mm Hg
   2. average rate of progression is valve area reduction of ~0.1 cm^2/yr
   3. symptoms occur late as compensatory LVH maintains stroke volume and cardiac output

B. Etiology
   1. age > 65: calcific degeneration most common cause
   2. age < 65: bicuspid aorta > rheumatic heart disease

C. Clinical findings
   1. harsh crescendo-decrescendo systolic murmur heard best at the base with radiation to the neck
   2. paradoxical splitting of S2 (widening of the split on expiration)
   3. hyperdynamic LV with palpable heave
   4. slow delayed carotid upstrokes (pulsus parvus et brevis)
   5. narrow pulse pressure
   6. ECG with LVH
   7. CXR with LV enlargement (late finding)
   8. Transthoracic echo (TTE) may be needed to confirm diagnosis

D. Complications
   1. syncope
      a. due to cerebral hypoperfusion, esp. during exertion
      b. less commonly due to dysrhythmia
      c. mean survival (unoperated) = 3-5 yrs
   2. angina
      a. due to hypoperfusion of coronary arteries (epicardial and subendocardial) rather than atherosclerosis
      b. mean survival (unoperated) = 3 yrs
   3. CHF
      a. occurs when hypertrophic LV begins to dilate and cannot maintain cardiac output
      b. poorest prognostic indicator
      c. mean survival time (unoperated) = 1-2 yrs
   4. sudden death
      a. accounts for 20% of death from AS
      b. pre-existing angina/CHF/syncope in ~75%

E. ED management
   1. IV, oxygen, cardiac monitoring, pulse oximetry
   2. ECG, chest radiograph, and cardiac enzymes
   3. manage hypotension with judicious IV crystalloid
   4. cautious treatment of chest pain and CHF
      a. venodilators can be very dangerous – may lead to relative hypotension and therefore larger gradient across the aortic valve

b. coronary hypoperfusion may result leading to worsening myocardial ischemia or dysfunction
5. for ongoing symptoms, cardiology and cardiac surgery consultation and CCU admission necessary
6. definitive therapy is AVR

II. Acute Mitral Regurgitation

A. Etiology
1. ruptured papillary muscle (secondary to recent AMI)
2. may occasionally be due to reversible ischemic papillary muscle dysfunction
3. ruptured chordae tendinae (usually due to RHD)
4. valvular perforation (usually due to endocarditis)

B. Pathophysiology
1. regurgitant volume can be 3-4x the volume of forward flow
2. results in severe pulmonary edema and cardiovascular collapse

C. Clinical findings
1. fulminant CHF, often with chest pain and evidence of myocardial ischemia
2. loud systolic murmur that obscures a soft s1
3. s3 gallop
4. no evidence of LAE or LVH seen with chronic MR
5. CXR with pulmonary edema with normal heart size

D. Diagnosis
1. transthoracic echocardiography will make the diagnosis
2. cardiac catheterization is generally necessary, especially in cases of suspected AMI

E. Management
1. airway management
2. afterload reduction (nitrates, nitroprusside, morphine, diuretics)
3. IABP may help stabilize the patient prior to surgery
4. emergent MVR unless reversible papillary muscle dysfunction is present and responds to treatment

III. Acute Aortic Regurgitation

A. Causes
1. Proximal aortic dissection
2. Endocarditis
   a. bacterial
   b. fungal
   c. noninfectious (marantic)
3. Aortitis (Takayasu, syphilitic, CTD's, others)
4. S/p valvuloplasty
5. Prosthetic valve dysfunction
6. Trauma

B. Pathophysiology

1. acute AR causes LV diastolic pressure to rise rapidly
2. in response, the LV dilates and fails
3. this causes fulminant pulmonary edema

C. Clinical findings
1. dyspnea, tachypnea, tachycardia
2. hypotension with cool extremities
3. s1 diminished or absent
4. diastolic blowing decrescendo murmur heard best with patient leaning forward
5. systolic flow murmur may be present
6. rales
7. CXR with pulmonary edema
8. note that a widened PP, water-hammer pulses, Quincke's nail pulsations, and Duroziez's to-and-fro murmur over the femoral artery are features of *chronic* AR and are not seen in acute AR

D. Diagnosis of acute AR and its cause
1. echocardiography
   a. TTE will diagnose AR
   b. TEE may determine its cause (AD, endocarditis, etc)
2. cardiac catheterization
   a. may be necessary for diagnosis
   b. also allows placement of IABP as necessary

E. Management
1. airway management
2. surgical emergency - immediate AVR (may be delayed if cause is endocarditis)
3. pre-operative stabilization with vasodilators (e.g. nitroprusside) and diuretics
4. IABP may be necessary while awaiting surgery

IV. Infective Endocarditis

A. Subacute endocarditis
1. epidemiology
   a. most patients have diseased valves (rheumatic/congenital most common)
   b. mitral valve most common site
   c. mean age range 55-65 years old
   d. recurrence rate 3-17%
2. pathophysiology
   a. sterile thrombus on a diseased valve
   b. subclinical bacteremia (dental procedures, GU/GI procedures, vaginal delivery, bronchoscopy)
   c. Strep species (esp. viridans) most common organism (65-75%), followed by staph aureus
   d. Strep bovis associated with GI malignancy/microperforation
   e. fungal endocarditis (candida, aspergillus) common in patients with long-term indwelling venous catheters and immunosuppressed hosts

3. clinical presentation
    a. fever, malaise, weakness, confusion
    b. anorexia, night sweats, weight loss
    c. regurgitant heart murmur
    d. vasculitic findings in >50% (Osler's nodes, splinter hemorrhages, Janeway lesions, petechiae)
    e. splenomegaly in 1/3 of patients
    f. retinal changes including flame hemorrhages or Roth spots

B. Acute endocarditis
    1. epidemiology/pathophysiology
        a. generally found on previously normal heart valves
        b. principal risk factor is injection drug use
        c. tricuspid valve most common site
        d. Staph aureus most common organism, followed by strep species and GNR's.
    2. clinical presentation
        a. patients look **toxic**
        b. tachycardic, tachypneic, fever
        c. regurgitant murmur in <35%

C. Prosthetic valve endocarditis
    1. epidemiology
        a. when occurring within 3-6 mos post-op, most likely organism is Staph epi (coag neg staph) followed by GNR's
        b. thereafter, strep viridans is most common, followed by staph epi and others
        c. incidence is 0.5-4% per year
    2. clinical presentation is similar to that of SBE

D. Diagnosis
    1. empiric for the EP
    2. blood cultures (at least three sets from different locations separated by time)
    3. echocardiography
        a. TTE 98% specific, <60% sensitive for vegetations
        b. TEE equivalent specificity with sensitivity ranging from 75-98%

E. Empiric antibiotic therapy
    1. subacute course:
        a. pencillin G *and* gentamicin
        b. vancomycin *and* gentamicin is an acceptable alternative regimen
    2. acute course
        a. nafcillin *and* gentamicin *and* ampicillin
        b. vancomycin *and* gentamicin

F. Complications
    1. embolic phenomenom
        a. seen in 25-50% of cases
        b. most cases occur within first 2 weeks of treatment
        c. staph and fungus emboli most frequently
        d. CNS most common site of emboli

2. Periannular infection
    a. very common with prosthetic valve IE (56-100%)
    b. 10-40% incidence in native valve IE
    c. AV conduction block may result
    d. prosthetic valve dehiscence leading to CHF
    e. rupture into the pericardium also a concern
3. acute valvular insufficiency with acute CHF
    a. perforation of valve
    b. rupture of chordae tendinae
    c. dehiscence of prosthetic valve
    d. paravalvular leak
4. valve obstruction from bulky vegetation
    a. fungus is frequent pathogen in this setting
    b. less common complication

G. Prognosis
1. with appropriate antibiotic therapy, symptoms begin to improve after 7-10 d
2. 5 year mortality is 20%

H. Prophylaxis
1. who:
    a. prosthetic heart valve
    b. h/o endocarditis
    c. RHD
    d. congenital heart disease
    e. MVP with regurgitation
    f. other valvular heart disease
2. for which ED procedures:
    a. dental procedure causing gingival bleeding
    b. insertion of FC when UTI is present
    c. I&D of abscess
    d. anoscopy/proctoscopy
3. which antibiotics:
    a. low risk patients get amoxacillin 3 g po one hr pre-procedure followed by 1.5 g po 6hr post-procedure (alternate therapy is erythromycin 1 g pre- and 0.5 g post-procedure)
    b. high risk patients get ampicillin 2g IV and gentamicin 1.5 mg/kg IV 30' pre-procedure (alternate therapy is vancomycin 1 g IV)

V. Prosthetic Valves Thrombosis

A. Epidemiology
1. mechanical prosthesis much more common
    a. tricuspid position most common
        i. 4-20% per patient-year
        ii. low pressure system facilitates thrombosis
    b. mitral position 0.5-0.6% per patient-year
    c. aortic position 0.1% per patient-year

2. incidence of thrombotic stenosis of bioprosthesis 0.03%/yr
3. prosthetic valve endocarditis is associated with a 13-40% incidence of thrombotic complications

B. clinical presentation
1. CHF
2. systemic emboli

C. diagnosis
1. clinically
   a. CHF
   b. change in heart murmur or mechanical sound
2. echocardiography (esp. TEE which images prosthetic valves better than TTE)
3. cineradiography – immobile disk/ball/leaflets

D. management
1. surgery used to be the standard of care
   a. redo valve replacement and/or thromboembolectomy
   b. in-hospital mortality 11-16% (much higher for patients with class IV hear t failure
2. thrombolysis
   a. first used in late 1970's, now increasingly first line therapy
   b. success rate 72-82%
      i. those with less severe heart failure did better
      ii. aortic did better than mitral
      iii. success unrelated to duration of symptoms, time since valve replacement or type of valve
   c. mortality is 10%
   d. rethrombosis in ~20% (can be re-lysed)
   e. systemic embolization is 12-20%
      i. neuro events are generally limited and transient
      ii. 4% required surgery or left permanent sequelae
   f. duration of therapy unclear
      i. older studies used prolonged infusion of SK (up to 3-4 days)
      ii. newer studies used SK and tPA in the manner currently used for STE-MI
   g. monitor response to therapy with TEE and/or cineradiography

SELECTED REFERENCES

I. Aortic Stenosis

1. Lester SJ, Heilbron B, et al: The natural history and rate of progression of aortic stenosis. *Chest* 1998;113:1109.
2. Sorgato A, Faggiano P, et al: Ventricular arrhythmias in adult aortic stenosis: prevalence, mechanisms, and clinical relevance. *Chest* 1998;113:482.
3. Gould KL: Why angina pectoris in aortic stenosis. *Circulation* 1997;95:790.
4. Faggiano P, Aurigemma GP, et al: Progression of valvular aortic stenosis in adults: literature review and clinical implications. *Am Heart J* 1996;132:408.
5. Selzer A: Changing aspects of the natural history of valvular aortic stenosis. *New Engl J Med* 1987;317:91.

II. Acute Mitral Regurgitation

6. Van Dantzig JM, Delemarre BJ, et al: Pathogenesis of mitral regurgitation in acute myocardial infarction: importance of changes in left ventricular shape and regional function. *Am Heart J* 1996;131:865.
7. Kono T, Sabbah HN, et al: Mechanism of functional mitral regurgitation during acute myocardial ischemia. *J Amer Coll Cardiol* 1992;19:1101.
8. Becker AE, Anderson RH: Mitral insufficiency complicating acute myocardial infarction. *Eur J Cardiol* 1975;2:351.
9. Gahl K, Sutton R, et al: Mitral regurgitation in coronary heart disease. *Brit Heart J* 1977;39:13.

III. Acute Aortic Insufficiency

10. Cohn LH, Birjiniuk V: Therapy of acute aortic regurgitation. *Cardiol Clin* 1991;9:339.
11. Rahimtoola SH: Recognition and management of acute aortic regurgitation. *Heart Dis Stroke* 1993;2:217.
12. Dervan J, Goldberg S: Acute aortic regurgitation: pathophysiology and management. *Cardiovasc Clin* 1986;16:281.

IV. Infective Endocarditis

13. Wilson WR, Karchner AW, Dajani AS, et al: Antibiotic treatment of adults with infective endocarditis due to steptococci, enterococci, staphylococci, and HACEK microorganisms: American Heart Association. *JAMA* 1995;274:1706.
14. Bayer AS, Bolger AF, Taubert KA, et al: Diagnosis and management of infective endocaridits. *Circulation* 1998;98:2936.
15. Dajani AS, Taubert KA, Wilson W, et al: Prevention of bacterial endocarditis: recommendations by the American Heart Association. *JAMA* 1997;277:1794.
16. Bayer AS: Infective endocarditis. *Clin Infect Dis* 1993;17:313.

VI. Thromboembolic Complications of Prosthetic Heart Valves

17. Edmonds LH Jr.: Thrombotic and bleeding complications of prosthetic heart valves. *Ann Thorac Surg* 1987;44:430.

18. Cannegieter, S.C., Rosendaal, F.R., Briet, E.: Platelets/Thromboembolism: thromboembolic and bleeding complications in patients with mechanical heart valve protheses. *Circulation* 1994;89(2):635.
19. Koller, Patrick T. MD, Arom, Kit V. MD, PhD: Thrombolytic therapy of left-sided prosthetic valve thrombosis. *Chest* 1995;108(6):1683.
20. Reddy NK, Padmanabhan TN, et al: Thrombolysis in left-sided prosthetic valve occlusion: immediate and follow-up results. *Ann Thorac Surg* 1994;58:462.
21. Vitale N, Renzulli A, Cerasuolo F, et al: Prosthetic valve obstruction; thrombolysis versus operation. *Ann Thorac Surg* 1994;57:365.
22. Graver LM, Gelber PM, Tyras DH: The risks and benefits of thrombolytic therapy in acute aortic and mitral prosthetic valve dysfunction: report of a case and review of the literature. *Ann Thorac Surg* 1988;46:85.
23. Kurzrok S, Singh AK, Most AS, Williams DO: Thrombolytic therapy for prosthetic cardiac valve thrombosis. *J Am Coll Cardiol* 1987;9:592.
24. Witchitz S, Veyrat C, Moisson P, et al: Fibrinolytic treatment of thrombus on prosthetic heart valves. *Brit Heart J* 1980;44:545.
25. Manteiga, Rosa MD, Souto Juan Carlos MD, et al: Short-course thrombolysis as the first line of therapy for cardiac valve thrombosis. *J Thorac Cardiovasc Surg* 1998;115:780.