Background: Rheumatic mitral valve disease is a common disease in India. Even in the presence of preserved global LV function as measured by EF, in some RHD patients, impairment in long-axis function can occur early. Recently impaired LV long-axis movement (by TDI) & myocardial strain is sensitive indicator of early myocardial dysfunction.

Aims and objectives: (1) To study TDI and its application in patients of Rheumatic MV disease. (2) To study the tissue Doppler velocities at the MV annulus. (3) To evaluate LV function in patients of rheumatic MV disease using strain & strain rate imaging.

Material and methods: This is single center prospective observational study conducted in 50 patients (pts) with rheumatic MS with or without MR with sinus rhythm in addition to 50 controls (cls). Pts with predecided inclusion & exclusion criteria were enrolled in this study & echo was done to measure MPI (tie) index, TD velocities (at the lateral MV annulus), Peak systolic myocardial velocity (Sm), Early diastolic velocity (Em) & strain imaging (SI) measured from averaged value of 6 LV segments from apical 4-C view.

Results: In this study mean age was 29.94 ± 8.02 in pts and 33.18 ± 9.0 in cls. Mean EF was 57.98 ± 5.257 in pts & 58.74 ± 2.783 in cls (p < .15). Mean MPI by TDI was 4560 ± .02740 in pts & 3934 ± .03543 in cls (p < .0001). Mean Sm was 7.98 ± 1.152 in pts & 13.44 ± 1.740 in cls (p < .0001). Mean Em was 8.70 ± 1.689 in pts & 16.18 ± 1.945 in cls (p < .0001). Mean averaged peak systic velocity was 17.7820 ± 1.1182 in pts & 21.9384 ± 1.431 in cls (p < .0001). Mean averaged strain rate was 0.838 ± 0.855 in pts & 1.455 ± 0.142 in cls (p < .0001).

Conclusion: Using TDI, our study revealed that the MPI was significantly increased in pts compared to control group. Moreover Sm, Em velocity and peak strain and strain rate was significantly lower in patients compared to control group. Thus MPI, Sm & Em velocity and strain imaging can be useful in the detection of subclinical LV dysfunction in patients of rheumatic MV disease.

Use of tenecteplase in treatment of thrombotic prosthetic mitral valves

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Introduction: Prosthetic valve thrombosis is defined as any obstruction of the prosthesis by non-infective thrombotic material. The diagnosis of PVT is made by a combination of clinical data (sudden worsening of heart failure, absence of prosthetic valve sounds, pulmonary edema, cardiogenic shock) and echocardiography. PVT is a life threatening emergency and requires treatment at the earliest. Reported incidence of thrombosis of prosthetic mitral valve is 0.05–4.3% per year. Thrombectomy or valve replacement is the conventional treatment with an associated mortality rate of 4.7–20%.

Thrombolysis is emerging as a promising alternative to surgery with a success rate ranging from 75% to 83%. In recent years, thrombolytic therapy has evolved as a substitute to surgery. Various thrombolytic treatments have been reported with variable success rates including streptokinase, urokinase and recombinant tissue plasminogen activators. Most patients in published series have been treated with infusions of streptokinase or urokinase for 12–24 hours. However, the data on the use of tenecteplase (a synthetic tissue plasminogen activator) is limited.

It has been used extensively in acute myocardial infarction (including in our institute) but its use in PVT treatment has rarely been reported.

Aims and objectives: To study the efficacy and safety of single intravenous bolus administration of tenecteplase in the management of patients presenting with thrombosis of prosthetic mitral valves in comparison to the use of streptokinase.

Materials and methods: All patients who presented with symptoms, clinical findings and radiological reports suggestive of thrombosis of prosthetic mitral valves and who were not willing to undergo emergency mitral valve replacement were included in this study. Patients who had contraindications to thrombolysis were excluded. Patients were offered both streptokinase and tenecteplase, choices differed as per financial constraints.

All consecutive patients presenting with thrombosis of prosthetic mitral valve, in NYHA Class III/IV were included in study if they were not willing to undergo emergency mitral valve replacement. The diagnosis of thrombosis of prosthetic mitral valves was established mainly by trans-thoracic or trans-esophageal echocardiography and/or fluoroscopy.

Patients with contraindication for thrombolytic therapy were excluded from the study (i.e., previous haemorrhagic stroke or other stroke within one year or known intracranial neoplasm or Active internal bleeding -excluding menses or suspected aortic dissection).

The fibrinolytic agent used was tenecteplase given in weight adjusted dose. Fibrinolytic regimens used were streptokinase and tenecteplase, 0.5 mg/kg IV bolus administered over 5 s. Heparin infusion was usually introduced after fibrinolytic therapy. Heparin infusion to obtain a partial thromboplastin time between 70 and 90 s was continued for one week and then replaced by warfarin treatment adjusted to obtain optimal prothrombin time and international normalized ratio. The efficacy of fibrinolytic therapy was assessed from hemodynamic parameters derived from echographic examinations as well as on clinical grounds.

We defined success as:

- Full: hemodynamic normalization confirmed by cinefluoroscopy (normal mobility of tilting disks) or TTE/TEE data (normalization of transprosthetic gradient and valve area, normal mobility of leaflet, reappearance of click).
- Incomplete: significant clinical improvement without complete recovery of disc or leaflet motion on fluoroscopy and/or TTE.
- Failure: no clinical improvement, in many cases associated with death or complications.

Results: Complete resolution of hemodynamic abnormalities was seen in 13/13 patients who received tenecteplase while 16 out of eighteen patients had complete resolution with streptokinase. One patient had failure of therapy and succumbed to pulmonary edema and another had partial resolution.

In patients who received tenecteplase, symptoms improved markedly, and prosthetic-valve clicks reappeared after a mean (±SD) period of 67.5 ± 29 minutes (within 30 min in two patients). No hemorrhagic complications were observed in any patients. Two documented embolic event occurred during fibrinolytic therapy. (Brachial artery embolism, requiring embolectomy and stroke due to embolism.)

None of the patients required subsequent surgery, and all were alive and well after treatment and discharged in a stable condition on warfarin in 7–10 days.

Treatment of patients with thrombosis of a prosthetic valve remains controversial.

Our results in thirteen consecutive patients suggest that tenecteplase can be used safely in patients with prosthetic-valve thrombosis, resulting in rapid restoration of valve function as compared the conventionally used streptokinase, beside ease of administration, i.e., bolus verses prolonged infusion.