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Post Myocardial Infarction VSD

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Post MI-VSD [VSR] Incidence

- 0.2% in the thrombolytic era [GUSTO-I] vs 1-2% in pre-thrombolytic era.
- In the SHOCK Trial Registry, post-infarction VSR was present in 3.9% of patients, with cardiogenic shock complicating AMI
- The time interval from onset of AMI to the manifestation of VSR has a bimodal distribution
  - Higher incidence in the 24h after the onset of infarction
  - Later between the third and fifth day
  - Rarely develops > 2wks after AMI

Post MI-VSD [VSR]

- VSR is usually associated with a lesion in a SVD [Total occlusion]. A minority of patients presents with MVD
- 60% complicates anterior MI
  - Simple VSD in the apical portion of the septum
- 40% complicates inferior MI
  - VSD in basal septum
  - Complex with extensive intramural rupture with propagation in different directions
  - Associated with significant damage of the right ventricle and MR [PM dysfunction] → worse prognosis

Post MI-VSD [VSR] Mechanism

- First 24 hours →
  - intramural haematoma dissection or haemorrhage into ischemic myocardial tissue
- 3-5dys →
  - coagulation necrosis of the myocardium with neutrophilic infiltration
- Subsequent retraction of surrounding tissue → enlargement of the defect, followed by a progressive fibrosis and mechanical reinforcement of tissue in the following weeks

Post MI-VSD [VSR] Risk factors

- First MI → absence of collaterals in the coronary circulation
- Old age
- Hypertension
- Female gender

Post MI-VSR

- Acute ischemic insult with loss of structural integrity of IVS.
  - Essential for partitioning and providing mechanical support for both RV and LV.
  - Actively participates in intracardiac conduction system.

Post MI-VSD [VSR] Hemodynamics

- L to R shunt →RV pressure and volume overload + LV volume overload
- Degree of shunt
  - Size of VSR
  - SVR/PVR ratio
- The decrease in COP → compensatory increase in SVR →increased L to R shunt.

Post MI-VSD [VSR] Clinical picture

- Severe dyspnea
- Hypotension
- Biventricular failure
- New systolic murmur – thrill
- Loud P2
- AV block
- Cardiogenic shock → multi system failure [shock liver – renal failure…..]

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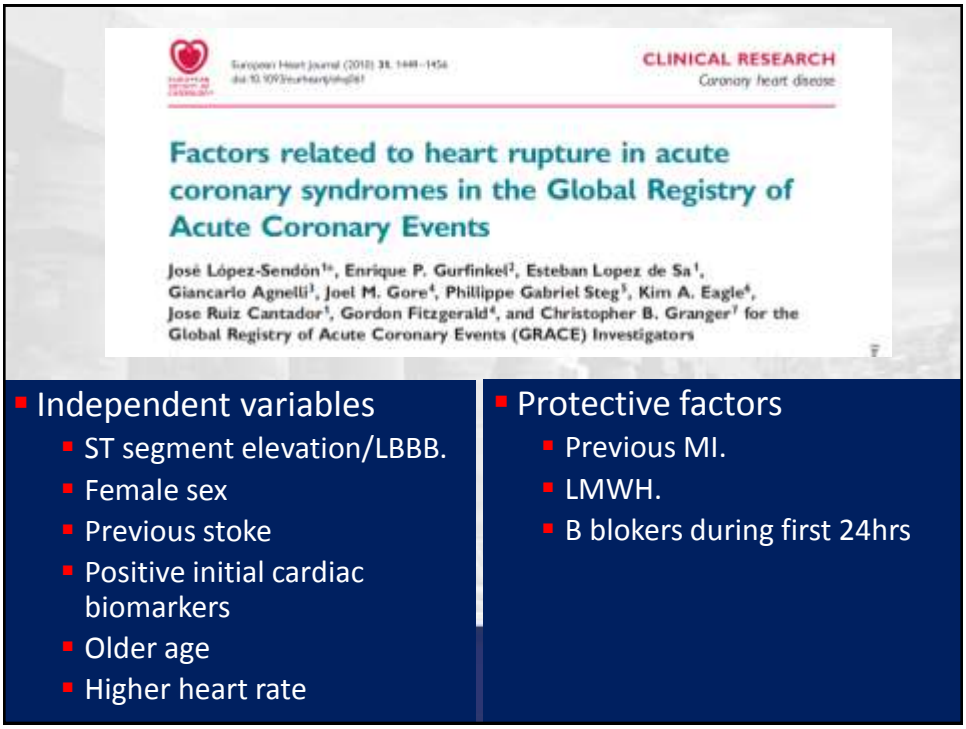

CLINICAL RESEARCH
Coronary heart disease

Factors related to heart rupture in acute coronary syndromes in the Global Registry of Acute Coronary Events

José López-Sendón[1*], Enrique P. Gurfinkel[2], Esteban Lopez de Sa[1], Giancarlo Agnelli[3], Joel M. Gore[4], Phillippe Gabriel Steg[5], Kim A. Eagle[6], Jose Ruiz Cantador[1], Gordon Fitzgerald[4], and Christopher B. Granger[7] for the Global Registry of Acute Coronary Events (GRACE) Investigators

- Independent variables
  - ST segment elevation/LBBB.
  - Female sex
  - Previous stoke
  - Positive initial cardiac biomarkers
  - Older age
  - Higher heart rate
- Protective factors
  - Previous MI.
  - LMWH.
  - B blokers during first 24hrs

Post MI - VSD

- ACC/AHA guidelines:-
  - Class I : patients with STEMI complicated by VSR should be considered for urgent surgical repair.
- If patient hemodynamic status is stable
  - Survival improves significantly if closure is preformed after 3 wks [Recession of necrotic ventricular septum is almost complete by 3-4 weeks]

Management of cardiogenic shock in ST-elevation myocardial infarction

ESC European Society of Cardiology

Recommendations	Class	Level
Immediate PCI is indicated for patients with cardiogenic shock if coronary anatomy is suitable. If coronary anatomy is not suitable for PCI, or PCI has failed, emergency CABG is recommended.	I	B
Invasive blood pressure monitoring with an arterial line is recommended.	I	C
Immediate Doppler echocardiography is indicated to assess ventricular and valvular functions, loading conditions, and to detect mechanical complications.	I	C
It is indicated that mechanical complications are treated as early as possible after discussion by the Heart Team.	I	C
Oxygen/mechanical respiratory support is indicated according to blood gases.	I	C

www.escardio.org/guidelines 2017 ESC Guidelines for the Management of AMI-STEMI (European Heart Journal 2017 - doi:10.1093/eurheartj/ehx095)

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ESC European Heart Journal (2017) 00, 1–0 doi:10.1093/eurheartj/ehx133

ESC GUIDELINES

2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation – Web Addenda

The Task Force for the management of acute myocardial infar
in patients presenting with ST-segment elevation of the Europ
Society of Cardiology (ESC)

- IABP may be used to stabilize patient
- Diuretics , vasodilators may be used with caution
- No consensus on optimal time for surgery
- Early surgery→ 20-40% mortality
- Delayed surgery → easier but carries the risk of rupture extension and death → early surgery should be performed in all pts with severe HF not responding to aggressive therapy
- Trans- catheter closure may soon become an alternative to surgery

8.4.2 Ventricular septal rupture

Ventricular septal rupture usually presents as rapid-onset clinical deterioration with acute heart failure or cardiogenic shock, with a loud systolic murmur occurring during the subacute phase. It may occur within 24 h to several days after MI and with equal frequency in anterior and posterolateral MI. The diagnosis is confirmed by echocardiography and Doppler, which will differentiate this from acute mitral regurgitation, and define the rupture and its size, and quantify the left to right shunt,[50] which can be more precisely confirmed by a Swan–Ganz catheter. The shunt may result in signs and symptoms of acute, new-onset right heart failure. IABP may stabilize patients in preparation for angiography and surgery. Intravenous diuretics and vasodilators should be used with caution in hypotensive patients. Surgical repair may be required urgently, but there is no consensus on the optimal timing for surgery.[51] Early surgery is associated with a high mortality rate, reported as 20–40%, and a high risk of recurrent ventricular rupture, while delayed surgery allows easier septal repair in scarring tissue but carries the risk of rupture extension and death while waiting for surgery. For this reason, early surgery should be performed in all patients with severe heart failure that does not respond rapidly to aggressive therapy, but delayed elective surgical repair may be considered in patients who respond well to aggressive heart failure therapy. Percutaneous closure of the defect with appropriately designed devices may soon become an alternative to surgery.[52]

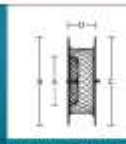
- 1957, cooley et al. Performed first surgical repair of PMIVSR.
- In 1988, lock et al. Reported first the transcatheter closure of VSR using cardioseal device
- Approximately 300 cases of TCC have been reported worldwide in the form of case reports, and small series with success rate of approximately 80–90%
- The periprocedural use of hemodynamic support like IABP, ECMO, and impela device provides stability
- Delay in intervention is supported by consensus

Patient Selection

- All patients with VSR should be assessed in consideration for device closure
- Echocardiography is the mainstay of imaging [site, size , no]
- The VSD must be remote enough to avoid device impingement of the TV,MV and AoV.
- The distance of the defect from the free wall of the ventricles has not been a factor but caution is required

Devices

AMPLATZER®
Muscular VSD PI Occluder

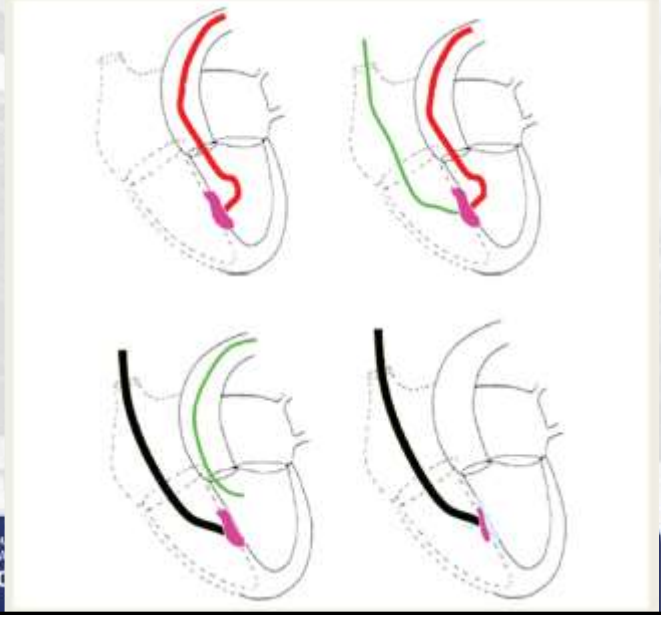


CONFIGURATION
A = Waist
B = RV Disc
C = LV Disc
D = Waist Length

Occluder Size (Waist mm)	TempVue® Delivery System Size (80 cm length)	Occluder Critical Dimensions (mm)
16 to 18	9 Fr	RV Disc (Waist + 10) LV Disc (Waist + 10) Waist Length (10)
20 to 24	10 Fr	RV Disc (Waist + 10) LV Disc (Waist + 10) Waist Length (10)

Amplatzer ASD used on an off –label basis

Technique

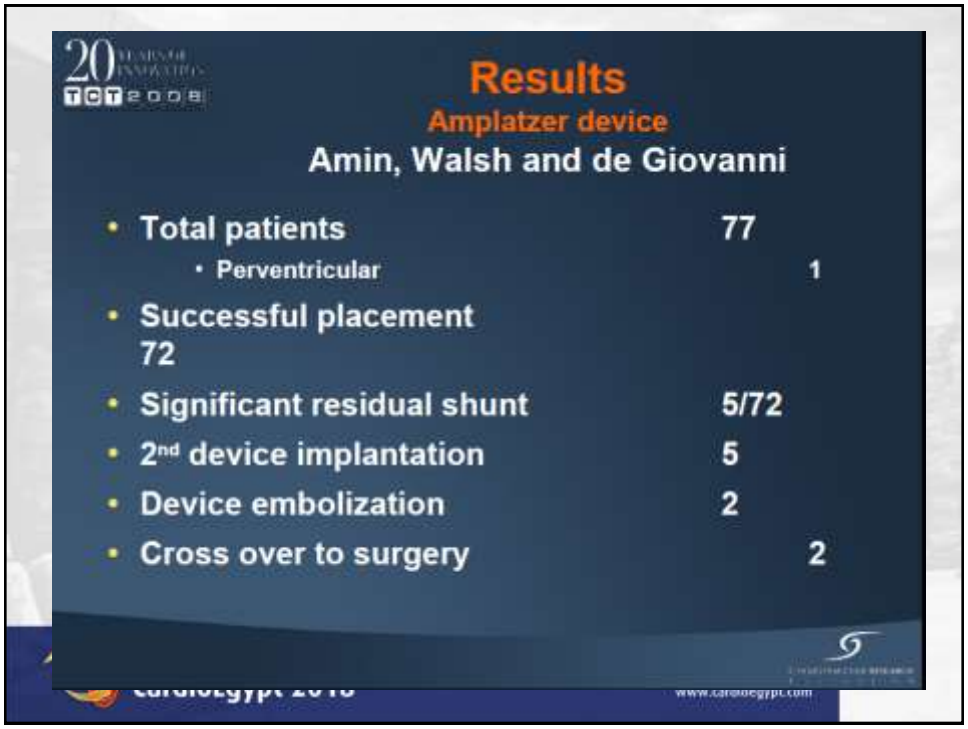


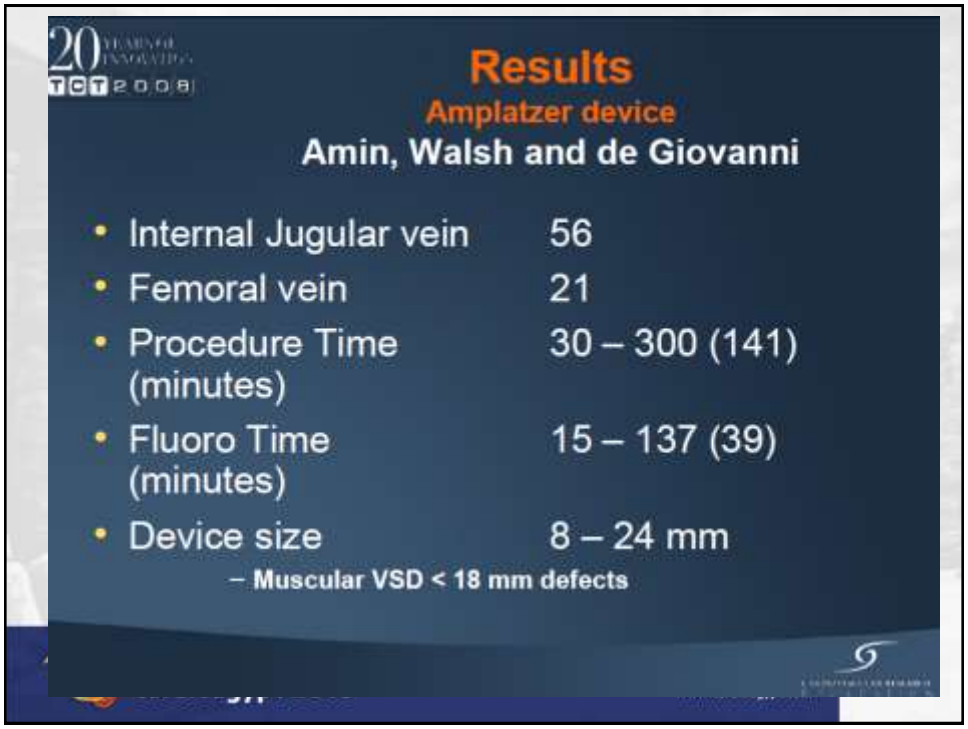
Catheterization and Cardiovascular Interventions 61:196–201 (2004)

Transcatheter Closure of Postinfarction Ventricular Septal Defects Using the New Amplatzer Muscular VSD Occluder: Results of a U.S. Registry

Ralf Holzer,[1] MD, David Balzer,[2] MD, Zahid Amin,[3] MD, Carlos E. Ruiz,[4] MD, Jeffrey Feinstein,[5] MD, John Bass,[6] MD, Michael Vance,[7] MD, Qi-Ling Cao,[1] MD, and Ziyad M. Hijazi,[1*] MD

N=18, early survival 40%


Results
Amplatzer device
Amin, Walsh and de Giovanni
Total patients 77
Perventricular 1
Successful placement 72
Significant residual shunt 5/72
2nd device implantation 5
Device embolization 2
Cross over to surgery 2


Results
Amplatzer device
Amin, Walsh and de Giovanni
Internal Jugular vein 56
Femoral vein 21
Procedure Time (minutes) 30 – 300 (141)
Fluoro Time (minutes) 15 – 137 (39)
Device size 8 – 24 mm
– Muscular VSD < 18 mm defects

20 YEARS OF INNOVATING TCT 2008

Results

Amplatzer Device

Amin, Walsh and de Giovanni

- Tricuspid valve damage 3
  - 1 died, 1 surgery, 1 RV failure
- Hemolysis 2
- Mortality 24/77 31 %

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European Heart Journal (2009) 30, 81–88
doi:10.1093/eurheartj/ehn524

CLINICAL RESEARCH
Valvular and congenital heart disease

Immediate primary transcatheter closure of postinfarction ventricular septal defects

Holger Thiele[1]*, Carl Kaulfersch[1], Ingo Daehnert[2], Martin Schoenauer[1], Ingo Eitel[1], Michael Borger[4], and Gerhard Schuler[1]

Aims Immediate surgical repair of ventricular septal defect (VSD) complicating acute myocardial infarction is associated with high mortality. Percutaneous device closure appears to be safe and effective in patients treated for a residual shunt after initial surgical closure, as well as in patients with a chronic post-infarct VSD. Primary transcatheter VSD closure in the acute setting may also offer advantages over surgery.

Methods and results Between September 2003 and February 2008, 29 consecutive patients underwent primary transcatheter VSD closure. Clinical, procedural, and outcome data were collected. Patients were divided into those with and those without cardiogenic shock at presentation for risk stratification. The median follow-up time of surviving patients was 730 days. The median time between VSD occurrence and closure was 1 day [interquartile range (IQR) 1–3] and the initial procedural success rate was 86%. The shunt (Qp:Qs) could be reduced from 3.3 (IQR 2.3–3.8) to 1.4 (IQR 1.2–1.7; $P < 0.001$). Procedure-related complications such as major residual shunting, left ventricular rupture, and device embolization occurred in 41%. The overall 30-day survival rate was 35%. Mortality was higher for cardiogenic shock in comparison to non-shock patients (88 vs. 38%, $P < 0.001$).

Conclusion Interventional acute VSD closure is a promising technique that can be performed with a high procedural success rate and may offer an alternative to surgery. Despite the less invasive technique, mortality of postinfarction VSD remains high, particularly in patients with cardiogenic shock. Further developments in devices and delivery techniques are required.

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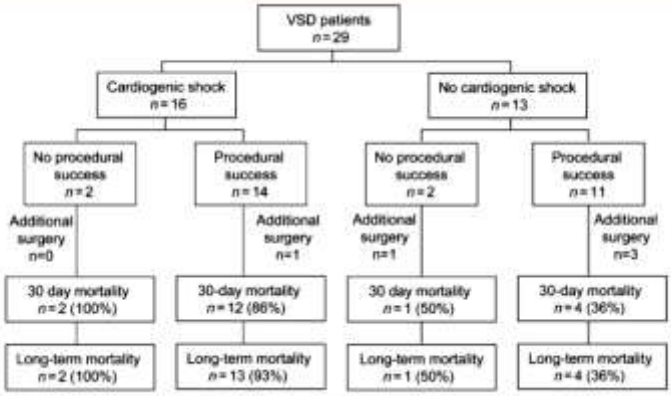


Percutaneous Closure of Post-MI Ventricular Septal Defect (VSD)

395-day follow-up of 53 post-MI percutaneous VSD closure attempts at 11 British centers, 1997 to 2011.

- **Procedural success was 89%, with more than half of patients (58%) surviving to discharge**
- **Only 4 additional patients died over 395 days of follow-up**
- **Prior surgical closure of the VSD, immediate shunt reduction associated with reduced long-term mortality**

Implications: Percutaneous closure of VSD is associated with high in-hospital mortality, but patients who survive to discharge have generally favorable long-term outcomes.

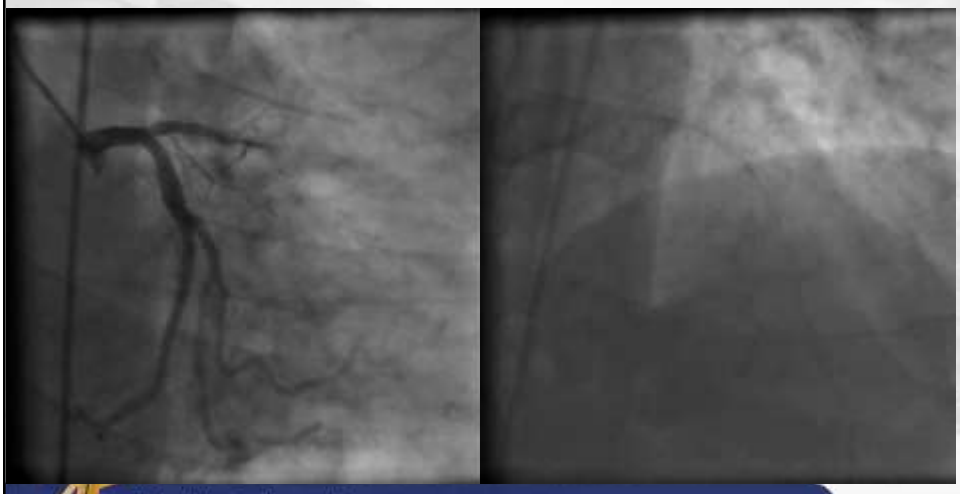
Calvert P, et al. *Circulation*. 2014;Epub ahead of print.

tctmd The Source for Interventional Cardiovascular News and Education

Case 1

- 77 years old male.
- Ex smoker , DM, HTN.
- Presented with Chest pain 7 hours duration.
- ECG :- acute anterior STEMI.
- Coronary angio – total occlusion LAD
- Primary PCI.

PCI



- Few hours after PCI developed a systolic murmur.
- Stable at first then rapid deterioration with congestive heart failure
- Echo:- VSR – EF 45% -↑RVSP.
- Shock liver with marked elevation of SGOT and SGPT
- ↑ BUN and creatinine .
- On support – IAB for 5 days.
- Surgery was considered very risky and trans-catheter closure was planned.

Case 2

- 62 years old male.
- No risk factors.
- Presented to hospital with progressive dyspnoea.
- Had a completed anterior STEMI 14 days earlier.
- Did not receive thrombolytic therapy and was managed at home with medical treatment.

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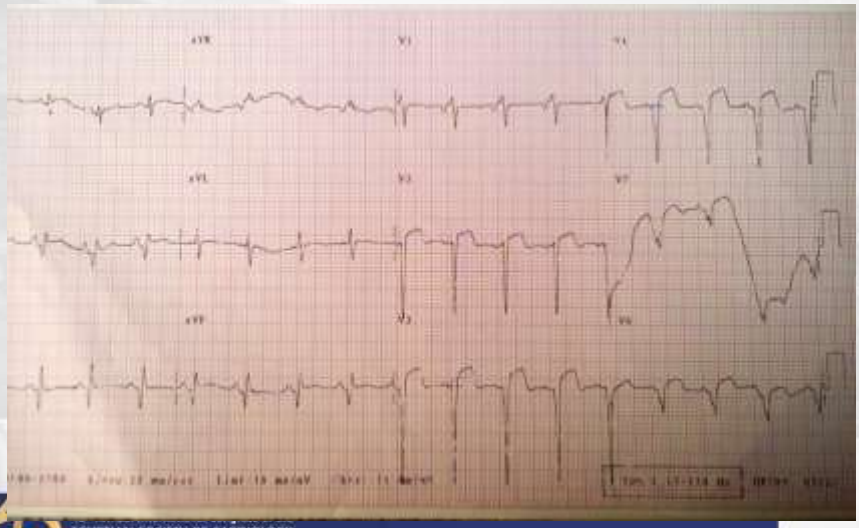
Examination

- Pale , orthopnic .
- BP = 90/60, HR =110b/min, Resp rate 25/min.
- Congested pulsating neck veins.
- Oedema of lower limbs.
- Normal S1 , Loud P2, S4.
- Harsh pan systolic murmur.
- Bilateral sibilant ronchi , scattered coarse crepitations together with fine basal crepitations.

ECG



Lab

- Random blood sugar : 95 mg/dl
- Serum creatinine : 1.8 mg/dl
- SGPT : 750 IU/L
- SGOT : 1050 IU/L
- INR : 1.2
- Albumin: 3.6
- Na: 125 K: 4.1 Ca: 8
- CK total: 42 - CK MB: 18
- WBCs: 15,000 HB: 9.8 Platelets: 142,000

Outcome

- Patient improved and was weaned off support.
- Echo showed good device position and improvement in his LV functions EF=45%.
- One week later he required ventilation for severe chest infection and died

Conclusion

- Device closure of VSD is feasible and offers a good alternative /bridge to surgery.
- Appropriate patient selection improves survival. ? Early intervention if needed.
- Post procedural ICU
- Ultimate outcome remains dismal
  - Co-morbidities and pre-procedure status of patient directly reflects outcome.
  - Technical success is dependent upon experience and team approach to VSD closure.


Thank You
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