Vol.13 July'15

MAX HEALTHCARE
Super Speciality Hospital

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Professional Writings by Medical Practitioners, Max Super Speciality Hospital, Saket

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Fit & Proper
– Don't ignore the injured muscle

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Sunil Bagga, 32, was rushing down the stairs of his office to make it in time for a meeting when he twisted his foot. He felt a searing pain, but limped through it. The pain persisted and the foot became red and swollen, but all he did was apply balm . A few days later, the pain became so severe that he was unable to walk. The doctor found Bagga had injured a muscle. The injury had become worse due to neglect. Recovery, he was told, would be a long process.

Broken bones, if set and fixed properly, heal so well that they are as strong as before the fracture. The same is sadly not true for the other component of our musculoskeletal system - the muscle. Muscles are responsible for all the movements of our body. Bones cannot move by themselves. It's the muscles that propel them and enable our body to move and function.

Bones heal by a regenerative process and the healing tissue is identical to the one that existed previously. Muscles heal differently. A muscle tear is often unpredictable and usually happens when the muscle is stretched too quickly. Usually this occurs while the muscle is in motion, such as when running, working or participating in some form of physical activity.

Muscle injuries are also often poorly diagnosed and inadequately managed.

The repair of muscle injury involves two processes. One is regeneration of the disrupted muscle fibres (myofibres) and the other is formation of connective tissue scar during the process of repair. A balanced progression is a prerequisite for optimal recovery of the contractile function of the muscle.

Muscles do not actually heal with muscle tissue but with "foreign" substances, including collagen. The resulting scar tissue is weaker, less elastic, and highly prone to re-injury. (Scar tissue is formed as part of the normal healing process when we injure our muscles, ligaments and tendons. It binds and ties down tissues that need to move freely and as the scar tissue builds up, muscles become shorter and weaker).

Although a majority of skeletal muscle injuries heal without formation of a disabling functional scar, sometimes this scar may be excessive within the injured muscle. This would lead to the muscle function being less than before the injury. This might also lead to chronic pain which persist for months - or even years. Once a muscle is damaged, it can become the source of a great deal of pain.

In a complete muscle tear, the torn muscle ends are not in contact and even repair by scarring might not happen, leading to a complete loss of muscle function unless treated properly.

QUICK TIPS

- Although diagnosis of muscle injury is usually through clinical examination, MRI or ultrasound might be required for a more detailed characterisation of the injury.
- When recovering from a muscle tear, the first thing you need to do is cease the activity that caused it.
- The amount of swelling can best be managed early by applying ice packs and maintaining the strained muscle in a stretched position.
- Compression can be gently applied with an elastic bandage, which can both provide support and decrease swelling.
- You need to give your body adequate rest and proper recuperation time to heal it. However, remaining completely sedentary is also not the answer.
- You should begin to move the joints and the muscle as soon as possible to prevent stiffness, atrophy, and weakness. Once you can do these without pain, you can begin some light activity, warming up properly beforehand.
- If you hear a "popping" sound with the injury, cannot walk or there is significant swelling, pain, fever or open cuts, you should be examined in a hospital's emergency department.
- Finally, sometimes surgery could be required to remove large blood clots or repair complete tear of muscles.

Digging deep – Unusual Case of FUO

– A diagnostic dilemma

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ABSTRACT

Recurrent episodic fever of unknown origin (FUO) arising from tuberculosis of the hepatobiliary system is rare and accounts for less than 1% of all tuberculous infections. We report an otherwise healthy 44-year-old man with recurrent circumscribed bouts of fever and raised CRP for 2 months who has remained well and fever-free in between. Occult tuberculosis should be considered and sought when routine investigations for FUO are negative.

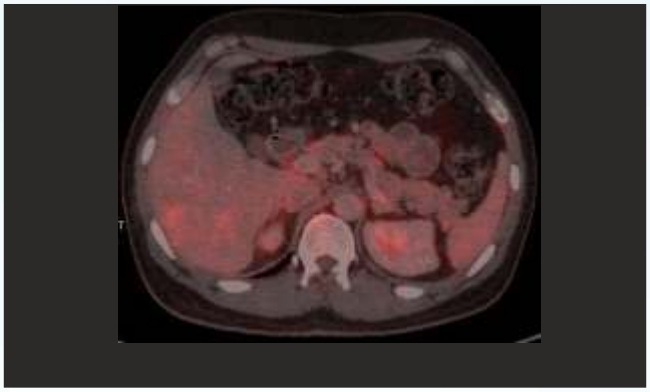
CASE REPORT

A 44 year old gentleman, with no such significant past history, founded with fever since 2 months. Moderate grade, irregular in nature with evening rise pattern, he also had low backache, easy fatigability, lethargy and generalised weakness. Risk factors for brucellosis, endocarditis, tuberculosis, travel to tropical countries, and retrovirus infection were negative. The patient denied anorexia and weight loss, and there was no family history of fevers, lymphoproliferative disease, or cancer. A complete physical examination was normal and except palpable right supraclavicular lymphnode, solitary, mobile, non-tender, 1x1 cm in dimension with soft in consistency. There were no heart murmurs, clubbing of the nails, skin rashes, joint effusions, and synovitis.

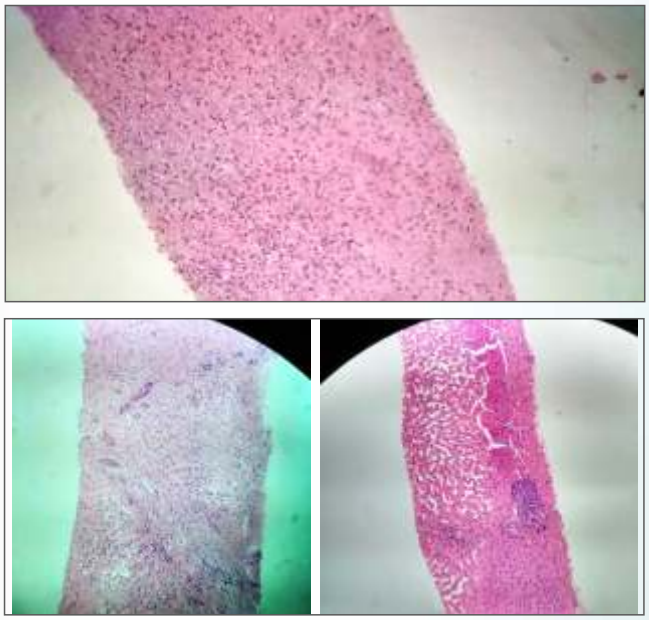
Patient was managed with broad spectrum IV antibiotics other supportive treatment. He responded only partially to the given treatment and continued to have intermittent low grade fever.

Numerous blood tests were returned as normal or negative. These included full blood count and blood film analysis, several blood cultures, creatine kinase, and lymphocyte subset analysis, as well as tests of organ-specific autoimmunity on a liver, kidney, stomach tissue block. There was also no evidence of antinuclear antibodies, rheumatoid factor, and anti neutrophil cytoplasmic antibodies. Serum immunoglobulins were normal, and there was no abnormality on serum immunoelectrophoresis. The kappa and lambda free light chain estimation, serum-angiotensin-converting enzyme and tests of liver and renal function as well as lactate dehydrogenase were also either normal or negative.

Quantiferon TB gold and PPD test- Positive, markedly raised ESR and CRP.
FNAC right supraclavicular lymph node showed features of reactive lymphadenitis, bone marrow aspiration and biopsy -- normal. CT (PET) whole body was done which yield nothing but multiple (at least 10 in number) FDG avid and non-avid hypo dense lesions in both lobe of liver.



After all above non-invasive investigation, further FNAC from Liver lesion were done --showed occasional atypical cells, not further characterizable.



CT guided biopsy of the liver lesion also left us in diagnostic dilemma (nonspecific hepatitis with focal fibrosis).

In view of positive quantiferon and PPD test, markedly Raised ESR and CRP and absence of any obvious focus of infection, it was decided to start the patient on empirical ATT. The patient is being started on ATT in view of suspected disseminated/ occult tuberculosis.

Further investigation on OPD basis:

- Anti LKM & ASMA were negative.
- IHC (Immunohistochemistry) which showed mixed lymphocytic infiltrates, not conclusive for a lymphoproliferative disorder. The patient made an uneventful recovery and has remained free of further bouts of fever after 2 weeks of starting ATT.

DISCUSSION

Pyrexia or fever of unknown origin (FUO) is often defined as fever of more than 38.3°C for 3-week duration and without an identifiable cause despite intensive investigations for more than one week. While fever can frequently be traced to infection (pyogenic or tubercular), systemic autoimmunity and inflammatory disease, malignancy involving the lymphoreticular system, solid organ tumours such as those of the kidneys and liver are not infrequently implicated. If we talk about solid organ liver which is an inhospitable place for tubercle bacillus owing to its low tissue oxygen tension? If the organism reaches the hepatobiliary tract via the hepatic artery from a tuberculous infection of the lungs (which may be active or inactive), it results in miliary tuberculosis. In some cases, however, infection could reach the liver via the portal vein especially if there is a concomitant involvement of the gastrointestinal tract. The tubercle bacilli may also reach the liver by lymphatic spread or due to rupture of a tuberculous lymph node in the portal tract. These latter cases result in localized hepatobiliary tuberculosis. This difference in pathogenesis may explain the observation that in miliary tuberculosis, lesions are concentrated near the hepatic veins, although in the local form, they are usually found periportally.

Histoplasmosis is differentiated from tuberculosis by the presence of small, discrete, scattered calcifications. Benign tumors of liver may show popcorn calcification. Liver cysts show marginal calcification. Primary hepatocellular carcinoma are usually solitary and show irregular calcification if any. On the other hand calcification is unusual in hepatic metastasis.

The mechanism of the intermittent fever and raised parameters of inflammation is unclear. However, cytokines such as IL1, IL6, IL18, and tumour necrosis factor are very likely involved.

CONCLUSION

The term hepatobiliary tuberculosis refers to the localized form of hepatic tuberculosis as a distinct clinical entity, with signs and symptoms related to the hepatobiliary tract. Presentations are often delayed, and manifestations can be nonspecific. The diagnosis of hepatobiliary tuberculosis in endemic countries should be considered in any patient with prolonged fever and chronic right upper quadrant pain associated with hepatomegaly, especially if accompanied by weight loss. The presence of associated pulmonary lesions (active or inactive) by chest radiography along with scattered hepatic calcifications by plain abdominal radiograph will aid in the diagnosis. Liver biopsy done either during laparoscopy, ultrasound- or CT-guided biopsies will establish the diagnosis if the caseating granuloma is seen. To confirm the diagnosis for a non-caseating granuloma, a positive AFB and/or culture for Mycobacterium tuberculosis would be needed.

Identification of Mycobacterium tuberculosis by PCR is more successful than by the conventional method of AFB and culture. In patients with chronic recurrent obstructive jaundice, especially when associated with an enlarged nodular liver and in those who have had the condition for more than one year, diagnosis of hepatobiliary tuberculosis should be highly entertained. Nearly two-third of patients responds to standard ATT given for one year. With the emergence of AIDS epidemic, physicians are likely to experience more aggressive, unusual forms of hepatobiliary tuberculosis with a higher rate of drug resistance and fatal outcome.

LEARNING POINTS

Infection (pyogenic or tubercular), systemic autoimmunity, inflammatory disease, malignancy involving the lymphoreticular system, solid tumor, isolated infection of the liver and kidney are common causes of FUO. Inspite of addition of medical nanotechnology to existing knowledge of molecular and cellular biology and sophisticated medical technology sometime fell short to make a diagnosis, in that scenario clinicians have to take step forward to take a decision taking in consideration the epidemiological nature of diseases.

Triple valve replacement

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INTRODUCTION

Rheumatic Heart Disease (RHD) is a significant cause of cardiac operations in developing countries. Tricuspid valve lesions associated with mitral and/or aortic valve replacement are most often repaired. Triple Valve Replacement is a complex operation and has an in-hospital mortality rate of about 10–12%[1,2]. Triple valve replacement (TVR) is challenging for most cardiac surgeons due to its prolonged periods of cardiopulmonary bypass (CPB) and aortic cross-clamping. Patients are commonly at late stages of RHD[3,4]. Despite substantial improvements in myocardial protection and CPB techniques, Triple Valve Replacement for advanced RHD. We report a case of a woman with Anaemia, Chronic Kidney Disease and stenotic lesions an all the three heart valves and who had Triple Valve Replacement with bio prostheses in Tricuspid position and mechanical valves in aortic and mitral position.

CASE REPORT

A 45-year-old female patient, known case of chronic renal failure complained of shortness of breath for 6 months. 2D Echo revealed thickened aortic valve, severe aortic stenosis with mild re-gurgitation, mean PG of 36mmHg across aortic valve, mitral leaflets thickened with fixed PML, severe sub-valvular fusion, severe mitral stenosis with mild regurgitation, mean PG of 11mmHg across mitral valve. Tricuspid leaflets thickened, severe tricuspid stenosis with regurgitation, mean PG of 6mmHg across tricuspid valve, Tricuspid annulus of 34mm, RVSP 70mmHg, RV systolic dysfunction present with LVEF 55%. Other investigation revealed atrial fibrillation (AF) with raised serum creatinine levels.

The patient underwent Triple Valve Replacement with 19mm SJM Regent Mechanical valve at aortic position, 25mm ATS Mechanical valve at the mitral position and 27mm SJM Epic Bio prosthetic at Tricuspid position. Surgery was performed under mild hypothermia at 34°C with cold blood cardioplegia and topical cooling with local ice slush. The patient was weaned from cardiopulmonary bypass without difficulty with mild inotropic support. Intraoperative hemofiltration was done in view of raised creatinine levels in the setting of CRF. The total cardiopulmonary bypass time was 274 minutes and aortic cross clamp time was 238 minutes. The postoperative course was uneventful. At 6months follow up, patient is presently asymptomatic, still in rate controlled AF with normalized cardiac silhouette on Chest X-Ray. Her 2D Echo shows normal functioning prostheses in situ with mean gradients of 4mmHg across mitral and tricuspid valve and 12mmHg across aortic valve.

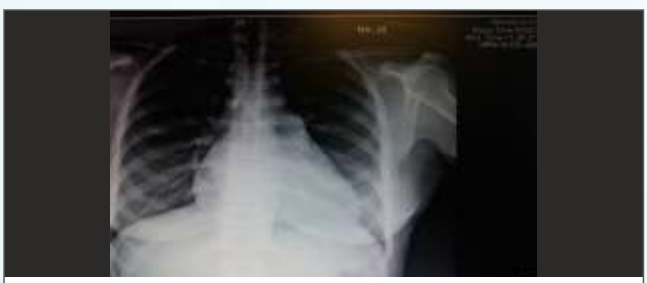

Figure 1: Pre-op CXR

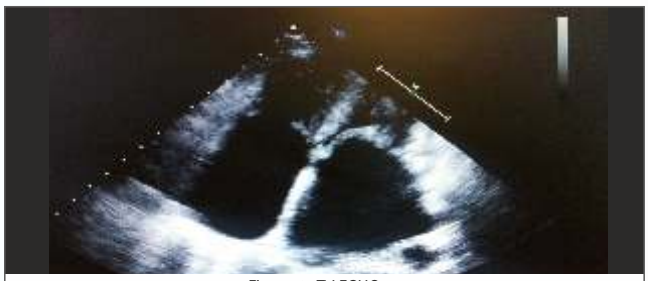

Figure 2: TV ECHO

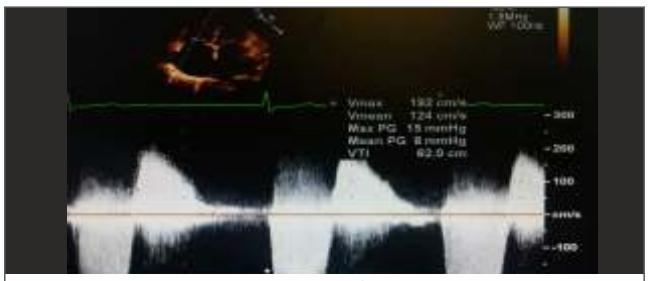

Figure 3: TV Peak Gradient

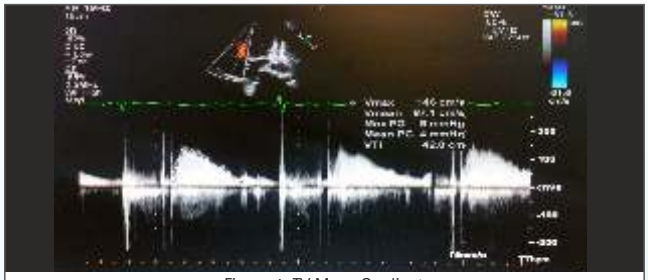

Figure 4: TV Mean Gradient

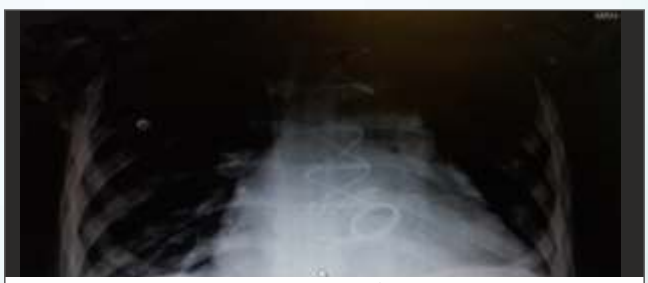

Figure 5: Post-op CXR

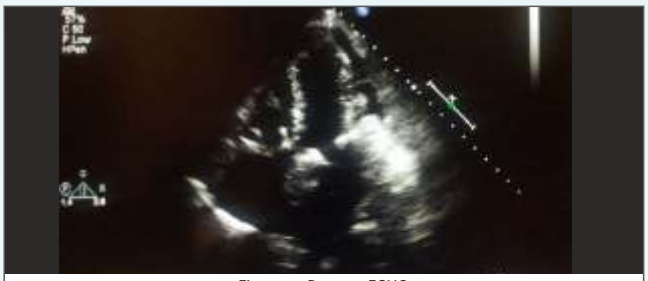

Figure 6: Post-op ECHO

DISCUSSION

TVR is an uncommon procedure and is infrequently performed; on the other hand, aortic and mi-tral valve replacement with tricuspid valve repair is commonly performed as the outcome of TVR is not as satisfactory as single or double valve replacement. Also TVR is associated with higher incidence of heart block as compared to TV repair[5]. Few cases of TVR using a combination of different types of valves have been reported[6]. It is well established that bio prosthetic valves in the aortic and mitral positions are associated with early degeneration and thus require early re-operation[6,7]. On the other hand, experience with mechanical valves implanted in the tricuspid position has not been encouraging[8,9]. Valve replacement remains the last resort in patients with severe organic tricuspid regurgitation and in the rare occurrence of tricuspid stenosis in which repair is not feasible or has failed[10]. Similarly in our patient we chose mechanical valve at mitral and aortic position along with Bio prosthetic valve at Tricuspid position due to severe commissural fusion and tethered chordae.

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Dual heart surgeries in Delhi hospital

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A 77-year-old woman suffering from heart disease with a damaged valve underwent a keyhole valve replacement surgery along with stent implantation surgery, to open up a blocked artery.

Doctors at Max Hospital, Saket, who conducted the procedure on Indira Narayan, 77, claimed that this was the first reported case of hybrid procedure involving both stenting and key-hole valve replacement.

"She was admitted in our hospital due to heart failure because of a damaged valve. She was treated with drugs initially and then advised a mitral valve replacement," said Dr Viveka Kumar, director, Cath Lab at Max Heart and Vascular Institute, Saket. He conducted the procedure along with Dr Rajneesh Malhotra.

"We had conducted a heart angiography to detect blockages, when we found abnormal narrowing of a blood vessel in the main artery," he said.

Doctors say that while her old age was a risk, she was also a patient of hypertension and chronic obstructive pulmonary disease, making her a very high risk case.

"We first conducted a mitral valve replacement surgery through a small incision and implanted a bioprosthetic valve on January 8. After she stabilised, we implanted a bio-absorbable stent after a week's gap," said Dr. Kumar.

"Keeping in mind her age, where she wouldn't have been able to tolerate long-term strong blood thinners so we chose the bio-prosthetic valve. The decision to use the new generation stents was also to avoid her from using life-long anti-platelet therapy, which is again a kind of blood thinner," he explained.

According to doctors at Max Hospital, Narayan tolerated both the procedures and was fit for discharge later that week.

Role of cardiac MRI in preoperative evaluation of congenitally corrected transposition of great arteries



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Patient came with chief complaints of cyanosis since birth which was progressively increasing.

There was repeated lower respiratory tract infection since birth. Patient came to Max hospital for further evaluation. Cardiac MRI was done in radiology department.

The MRI findings were:

1. Situs solitus with Levocardia.
2. Atrio-ventricular and ventriculoatrial discordance. Morphological left ventricle is lying right to morphological right ventricle, so morphological right atrium is connected to morphological left ventricle and morphological left atrium to right ventricle. Large VSD was seen. (Figure.1 & 2)

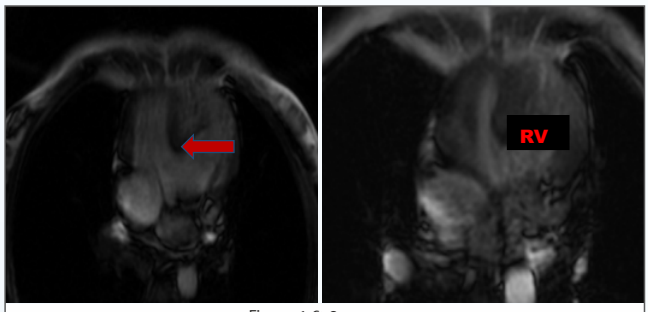


Figure 1 & 2

Pulmonary artery was arising from morphological Left ventricle and lies posterior to Aorta. (Figure.3)

Aorta is left sided with normal branches. Severe pulmonary valvular stenosis with thickened pulmonary valve was seen measuring 11.7mm, supravalvular region 13mm. (Figure.4) Pulmonary branches are confluent showing normal caliber.

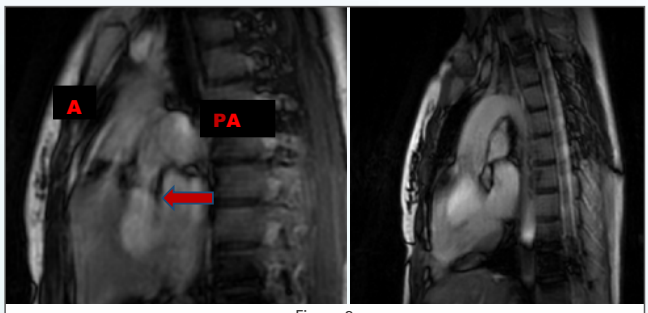


Figure 3

LV and RV function analysis was done which showed normal RV and borderline normal LV function.

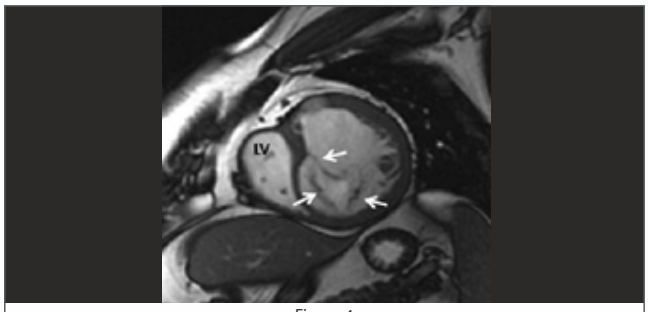


Figure 4

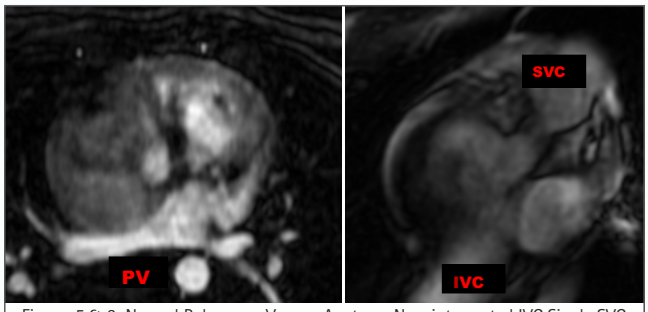
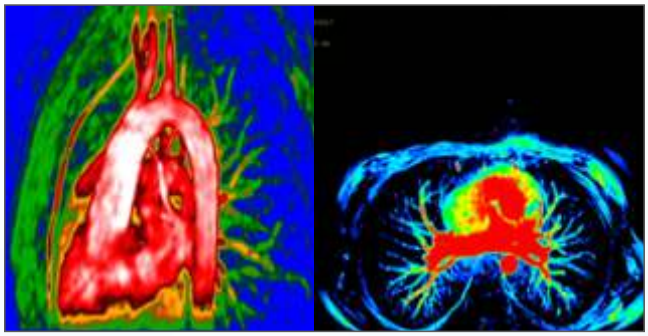


Figure. 5 & 6: Normal Pulmonary Venous Anatomy Non-interrupted IVC Single SVC



MRI DIAGNOSIS

1. Corrected transposition of great vessel. With atrio ventricular and ventricular discordance.
2. Severe pulmonary valvular stenosis with dysplastic thickened pulmonary valve.
3. Pulmonary artery lying posterior and to the right side of aorta.
4. Border line normal left and right ventricular function.

SURGICAL INTERVENTION

Extra Cardiac Fontan was done. The surgery went uneventful and patient was discharged on 15th day of surgery.

DISCUSSION

Congenitally corrected transposition of the great arteries (ccTGA) is a rare defect combining atrioventricular discordance with ventriculoarterial discordance. The atria are connected to the opposite ventricle (left atrium to right ventricle via a tricuspid valve) and the ventricles are connected to the incorrect great artery (right ventricle to aorta). Thus oxygenated blood is circulated systemically by the morphologic right ventricle (RV) and deoxygenated blood returns to the right atrium to be pumped out the left ventricle (LV) to the lungs.
The defect is therefore 'corrected' because of the physiologic flow of blood through the body.

Cardiac MRI is now used in many types of CHD to further define anatomy and to quantify ventricular function and volume. For initial diagnosis, cMRI may be helpful in patients with restricted TTE windows, to define visceroatrial situs, and to delineate complex associated defects. In patients with interruption of the inferior vena cavae, systemic return from the lower body can be difficult to delineate by echocardiography, but is well defined by cMRI. Because echocardiographic evaluation of RV function in ccTGA patients is limited by geometric assumptions, cMRI has become the gold standard for RV function and volume assessment. TV morphology as well as degree of regurgitation can also be determined through cMRI. Prior to performing anatomic surgical repair in a ccTGA patient beyond infancy, cMRI may be useful in evaluation of LV mass, volume, and ejection fraction. Furthermore, if there are concerns about degree of LV dysfunction, perfusion studies with delayed enhancement MRI may be performed to directly investigate scarring of the LV myocardium prior to committing this ventricle to systemic workload. Cardiac MRI may therefore be a useful modality for evaluation of ccTGA patients not only as an adjunct to TTE for initial diagnosis, but also for assessment prior to surgical repair and serial follow-up of the systemic RV.

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Successful management of acute rejection after heart transplant




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BACKGROUND

Survival after heart transplantation has improved considerably over the past 20 years. Improved longevity means prolonged immunosuppression and the concomitant use of drugs to prevent or treat the long-term complications of immunosuppressive agents, such as infection, obesity, hypertension, hyperlipidemia, renal insufficiency, diabetes, osteoporosis, gout and malignancies. Despite prolonged survival, heart transplant recipients continue to take multiple medications. With the large number of heart transplant recipients in the community and the increasing number of immunosuppressive and non-immunosuppressive drugs used by these patients, it is important that the treating cardiologist understand these drugs, their side effects, and the very real potential for drug–drug interactions. These interactions may result in adverse events caused by supratherapeutic and subtherapeutic drug concentrations. The rejection of a transplanted organ is primarily a T-lymphocyte (T-cell)–mediated event, although humoral (B-cell) responses also contribute. The exception is hyperacute rejection, which occurs when preformed antibodies to human leukocyte antigens (HLA) result in an immediate and catastrophic rejection. We here present a case who had heart transplant 3 years ago in Southern part of India and now presented with acute rejection episode.

MATERIAL

A 37 year old female who underwent heart transplantation three years ago for dilated cardiomyopathy. At four in the morning she developed sudden breathlessness and coughing. She visited to nearby hospital in the emergency department where oxygen and diruretics were given. Echocardiography was done over there which revealed drop in EF i.e. 45% which was normal (55%) in routine check up a month ago. Then patient was transferred to our hospital for detection and management of rejection. She was admitted to CTVS ICU where she was closely monitored for ECG and blood pressure. Milrinone infusion was started as an inotorope to support heart. ECHO was done which showed further drop in the ejection fraction to 35% by evening. As she was breathless, tachycardiac, tachypoenic and had crepts all over the chest. Clinically she was going into heart failure. In anticipation of acute rejection she was started on high dose steroid pulse therapy (500mg BDx 3 days).Cyclosporinetrough and peak levels were sent to see adequacy of immunosuppressive dose. Other immunosuppressive drug Cellcept (Mycophenolate sodium) continued in the same (1000mg BD) dose. Fluconazole was also started to avoid opportunistic fungal infection as she was put on high doses of steroid. Her all routine lab investigations were sent along with specific tests including CD4 and CD8. Next day though she clinically looked stable but her EF was

25%. She was taken for Endomyocardial biopsy and coronary angiography to detect rejection and to see if she had developed allograft vasculopathy. Her coronaries were found normal. Pulmonary pressures were with in acceptable limits. Her Cyclosporine trough level was very low so its dose was increased to 37.5 mg from 25 mg BD. All other medications continued as such and strict isolation precautions were taken to avoid catching infection from surrounding. On day two her biopsy showed 2R cellular rejection and no AMR (Antibody Mediated Rejection). She was added tacrolimus 0.5 mg BD and its trough level was checked after 3 days. Meanwhile she was hemodynamically stable. Her ejection fraction started improving. Milrinone was weaned off. She was sent to room and serial ECHO's were continued to see progress. Her TacIrolimus level after 3 days came sub-therapeutic so dose was increased to 1 mg BD. Methylprednisolone slowly weaned from IV to oral preperations. Once Tacrolimus level came in desirable range cyclosporine was discontinued. She was discharged home in a stable condition with a plan to repeat biopsy after 15 days. She came in for follow up endomyocardial biopsy after 18 days and which revealed no rejection. Her ejection fraction improved from previous admission. She has been clinically fine since then. Her tacrolimus trough levels are in therapeutic range now.

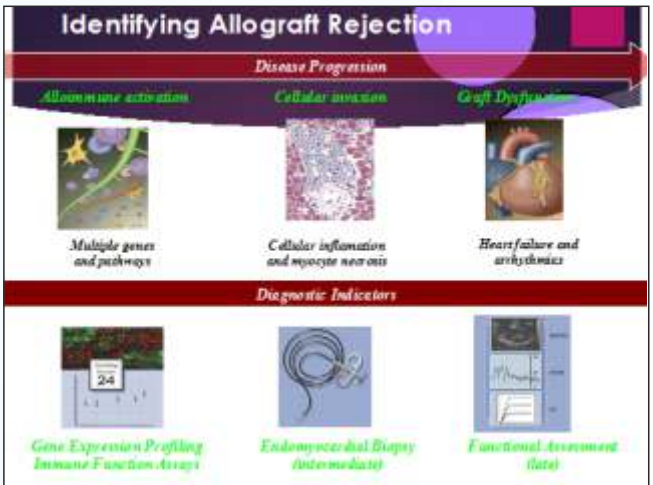


Figure1. Methods of identifying rejection.


Figure 2: Biopsy being taken in Cath lab

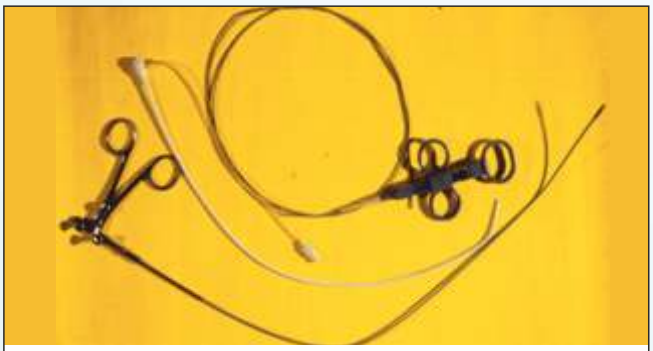


Figure 2: Biopsy being taken in Cath lab

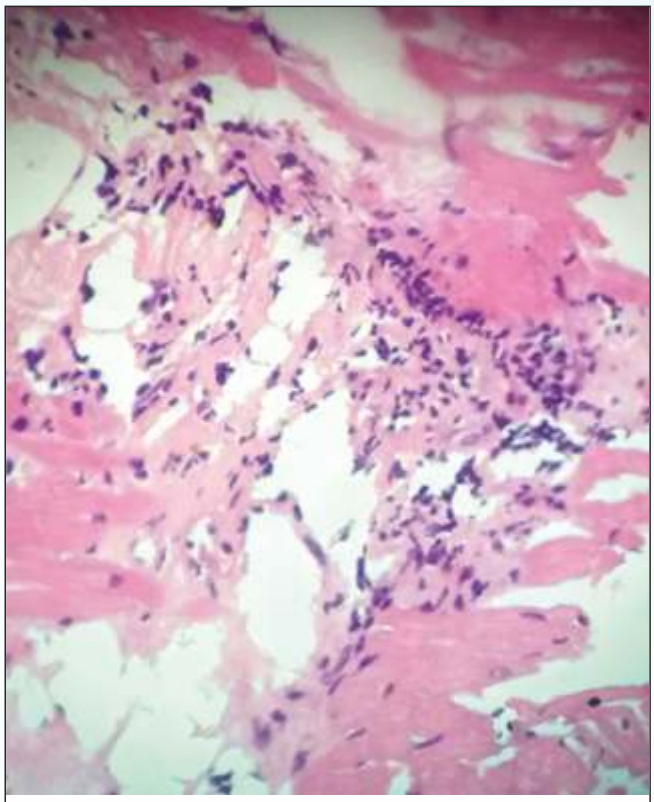


Figure 4: Grade 2R Rejection in the endomyocardial biopsy specimen from right ventricle (at admission)

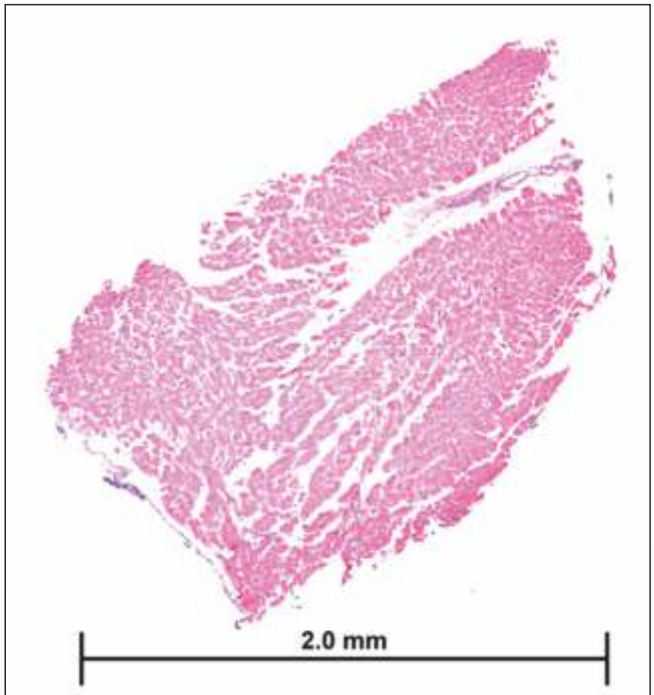


Figure 5: Biopsy after 18 days shows no evidence of rejection (Grade 0)

DISCUSSION

Rejection of the transplanted heart is a major cause of morbidity and mortality in the first year after heart transplantation. Acute cellular rejection may occur at any time after transplantation but is most common in the first 3 to 6 months but this patient showed acute rejection after 3 years of heart transplantation. The diagnosis is usually made by endomyocardial biopsy with a standardised grading scheme ranging from mild to moderate to severe acute rejection. Moderate rejection by endomyocardial biopsy is associated with mononuclear cell infiltrates and myocytolysis. A diagnosis of moderate rejection generally prompts antirejection therapy that varies according to histological severity (grade of rejection) and hemodynamic function. Patients with acute cellular

rejection often notice mild symptoms of fatigue or shortness of breath. More severe rejection may be associated with signs of left heart failure and left ventricular dysfunction. Therapy includes intravenous or oral steroids, depending on severity. Intensification of existing immunosuppressive therapy and/ or switching to another immunosuppressive therapy is usually done. Acute humoral (also called vascular) rejection occurs days to weeks after heart transplantation and is initiated by antibodies rather than T cells. The diagnosis is made by demonstrating immunoglobulin and complement in the vessels of the transplanted heart in an endomyocardial biopsy specimen or by the presence of swollen endothelial cells on hematoxylin and eosin staining (C4d). Our patient was negative for AMR both the times. Chronic rejection occurs months to years after transplantation. In heart transplant recipients, chronic rejection type presentation also there in coronary allograft vasculopathy (CAV) and manifests as diffuse atherosclerosis with myointimal proliferation in the coronary arteries. The diffuse involvement of the coronary arteries results in ischemia and infarction. CAV develops three to five years after heart transplant. That is why coronary angiography was done which revealed normal coronaries in this patient. Rejection (or rescue) therapy refers to immunosuppressive therapy given to reverse an episode of rejection. The intensity and type of rejection therapy depend on the severity and hemodynamic consequences of the rejection. Other noninvasive methods are AlloMap which identifies gene expression, though this method has not yet been standardised.

CONCLUSIONS

Acute rejection after heart transplant can be managed successfully by close monitoring, pulse steroid therapy and intensification of existing or switching to another immunosuppressive agents.

Case Study – Kienböck's Disease

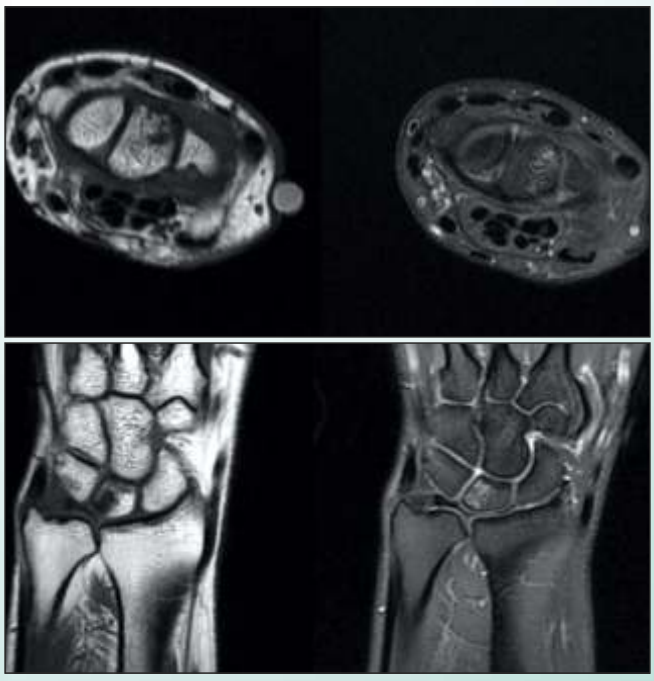
by Dr. Shivani Pujara, Dr. Amit Kumar, *Department of Radiology*

CASE PRESENTATION

A 29 year old lady presented with wrist pain for last 2 months with history of minor trauma. .She was on painkillers but to no relief. X-ray was inconclusive, MRI study was performed.

IMAGING

X-Ray findings were nonspecific. Patchy area of signal alteration hyperintense on PD FS are seen in lunate. However congruity of the bone was maintained. Mild area of signal alteration is seen on TFCC along the ulnar styloid. Minimal joint effusion is seen.



DIAGNOSIS

Kienböck disease is a condition characterised by a vascular necrosis of the lunate bone The Austrian radiologist Robert Kienböck described this entity in 1910. Kienböck disease is often progressive, resulting in joint destruction within 3–5 years if left untreated.

PLAIN FILM

Sclerosis and flattening of the lunate. Fragmentation of the lunate and secondary degenerative disease may develop later.

MRI

It is the most sensitive and specific test and may detect very early disease. Bone oedema (high T2, intermediate T1) may be seen in the acute phase, Sclerosis (low T1 and T2) is usually seen centrally and within the radial aspect of the lunate.

DIFFERENTIALS

- Ulnar impaction syndrome
- Intraosseous ganglia
- Lunate fracture

REFERENCE

- Imaging of Kienböck Disease, Javier Arnaiz, Tatiana Piedra, Luis Cerezal, John Ward, Alex Thompson, Jorge A. Vidal and Ana Canga, July 2014, Volume 203, Number 1
- Kienbock's disease and juvenile idiopathic arthritis, Nicholas M. Desy,* Mitchell Bernstein, Edward J. Harvey, and Hazel Hazel Mcgill J Med. 2011 Jun; 13(2): 8. Published online 2011 Jun.

WELCOME TO THE TEAM

Dr. Neeraj Awasthy

Sr. Consultant & In-charge - Paediatric Cardiology
Max Super Speciality Hospital, Saket

EDUCATION

- M.B.B.S, U.C.M.S, Delhi University, Delhi, Dec'99
- M.D (Paediatrics), Safdarjung Hospital & V.M.M. College, Delhi University, Delhi, April'04
- FNB Fellowship, National Board (Paediatric Cardiology), National Board, India, Escorts Heart Institute & Research Center (EHIRC), Delhi, April'09

EXPERIENCE

- Worked as Research Officer under a WHO Project with AIIMS, Delhi, June 2004 - Sep'04
- Senior Resident, Department of Paediatrics, LNJP Hospital & MAMC Delhi, Sep'04 - Feb'07
- Fellow, Paediatric Cardiology (National Board) at Escorts Heart Institute and Research Centre, Delhi, Feb'07 - Apr'09
- Junior Consultant, Department of Paediatric Cardiology at Fortis Escorts Heart Institute and Research Centre, Delhi, Apr'10 - Jun'12
- Associate Consultant, Department of Paediatrics Cardiology at Fortis Escorts Heart Centre, Jul'12-13
- Consultant, Department of Paediatrics Cardiology at Fortis Escorts Heart Centre, Jun'13

ACCOMPLISHMENTS

- Have around 70 publications in various national & international journals
- Involved with the organisation of the various events promoting care of the children with heart diseases and starting paediatric cardiology programme in various parts of the country

MEMBERSHIP

- Member of MCI, DMC, IAP, PAD, PCSI
- Provisional Membership American Academy of Echocardiography 2013-2014
- Member Cardiology Society of India
- ACLS (American Heart Association) Healthcare provider since 2010 (renewed certification in 2014)
- BLS (American Heart Association) Healthcare provider (renewed certification in 2014)

LOCATION AND DURATION OF OPD

Max Super Speciality Hospital, Saket

- Monday to Friday: 9.00 am - 5.00 pm

Funny Bone

My teenage patient's mother was concerned.

"He must have a temperature," she said, "He hasn't taken our motorcycle out all day."

"Let me ask you," I said, "Do you have a thermometer?"

"No," she said, "A Kawasaki."

A woman gets into a taxi and asks: To maternity hospital, please..

After a while she asks the driver: Do not drive so fast, please, I'm simply working there.

Dentist: Don't worry, it will take me only a minute to pull your tooth out.
Patient: And how much will it cost me?
Dentist: ₹5000
Patient: For a 1 minute job?!
Dentist: If you prefer, I can be pulling it out for one hour...

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*Subject to force majeure and prevailing traffic conditions and within 10 km radius of Max Hospital in Delhi - NCR.