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REVIEW ARTICLES **OPEN ACCESS**

ANESTHETIC PLAN FOR REVASCULARIZATION IN KAWASAKI DISEASE WITH SEVERE LEFT VENTRICULAR DYSFUNCTION DEPARTMENT OF CARDIAC ANESTHESIOLOGY

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Kawasaki Disease, Severe Left Ventricular Dysfunction, Arterial Grafting.

ABSTRACT

Introduction: -The purpose of this case presentation is to convey the importance of anesthetic plan in pediatric Kawasaki disease patients who present with severe left ventricular dysfunction. **Case:** A 2 year old male presented with an episode of respiratory failure of unknown etiology. The patient was transferred from an outside facility to our institute for respiratory failure of unknown etiology with cardiomegaly in chest x- ray. **Management and outcome:** Following initial stabilization the patient remained dyspneic. In the emergency department a bedside chest x-ray demonstrated cardiomegaly. A formal cardiac transthoracic echocardiography was obtained which demonstrated findings of coronary vessel aneurysm and hypokinetic myocardium. The patient was transferred to pediatric cardiothoracic surgery after confirming left anterior descending artery(LAD) occlusion with retrograde filling form right coronary artery. His abnormality was ultimately repaired by arterial grafting. **Discussion:** The majority of congenital heart diseases are diagnosed near the time of birth but may present suddenly in otherwise well pediatric patients. Clinical suspicion for heart disease should be in the differential diagnosis of an unresponsive pediatric patient without cardiac murmur and respiratory failure. We review the presentation and clinical findings in patients with Kawasaki disease with severe left ventricular dysfunction.

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INTRODUCTION

Kawasaki disease, initially called mucocutaneous lymph node syndrome (MLNS or MCLS) was reported by Kawasaki in 1967 (1) as a new pediatric disease entity (2). It causes a variety of symptoms and signs, such as high fever, conjunctive injection, a skin rash like measles, red swollen lips, strawberry tongue, cervical lymph node swelling and desquamation of the fingertip skin, which mostly regress spontaneously within 2 weeks (but sometimes symptoms last over a month) unless rupture or obstruction of coronary aneurysms supervene at the acute phase of the illness. The current death rate at the acute phase is approximately 0.05B0.1% following the induction of high-dose intravenous c-globulin therapy (3,4). Laboratory examinations reveal an elevation of C-reactive protein, white blood cell count, erythrocyte sedimentation rate and platelet count and a decrease in hemoglobin level and serum albumin. Recently it was found that the varieties of symptoms described above are related to hypercytokinemia activated in the acute inflammatory phase of Kawasaki disease. Elevations of interleukins 1, 2, 6, 8, 10, interferon and tumour necrosis factor-α are noted in almost all children with this disease (5,6). This syndrome is always preceded by symptoms like upper respiratory infection, but any causative agents such as bacteria, mycoplasma and viruses have not yet been identified. However, recently it has been reported that bacterial superantigens may be related to the etiology of systemic vasculitis of Kawasaki disease (7). Although Kawasaki disease is currently seen worldwide, it is apparent that the disease is more predominant in Orientals than in Caucasians (8), so some genetic predispositions may be related to the etiology of vasculitis, but nothing has been confirmed at the present time. In Japan, more than 150 000 patients have been documented to date and over 8000 new patients were found in a single year (2001).

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The most common age for the onset of the disease is 1 year old. It usually occurs in children less than 4 years old, but sometimes it occurs in adolescents. The gender distribution is 1.5 male dominant. Regarding the treatment for acute illness, aspirin has been used since 1977 with a resultant coronary artery aneurysm or ectasia (at 1 month after the onset) occurring in approximately 20% of patients. Since 1982, high-dose intravenous IgG-globulin administration (200–400 mg/kg for 5days) has been introduced with a resultant occurrence of coronary artery sequel (at 1 month) being reduced to 7% of patients. However there are still many children who develop coronary aneurysms over 7–8mm in diameter (giant aneurysms) that will frequently result in coronary artery stenosis or obstruction in 1 to 20 years. Although the etiology of the disease remains unknown, its serious complications (coronary aneurysm formation and subsequent coronary arterial obstructive lesions) have been proved to cause ischemic heart disease in children. Although the incidence of pediatric ischemic heart disease is fortunately low (2–3% of patients with Kawasaki disease), once the serious coronary arterial lesions develop into pediatric myocardial infarction, the prognosis is more serious than previously imagined.

CASE REPORT

This was a 2.9 year male patient hospitalized for high grade fever, mild dyspnea and later diarrhea . He had same episode 4-5 times in past . He hospitalized almost every time and routine blood investigation was done and everything was within normal limit. After 8-10 day of symptomatic medical management patient relived and discharged, he also had muscle tightness during course of disease and skin exfoliation also seen over palm and sole. During last time of admission symptom was not relieved and severe dyspnea and grunting was present for that patient was referred to otorhinology department for any upper respiratory tract infection where sinuses was examined and chext ray was done.

They found that there was increased mucosal dryness and mucosal thickening which was not cause of dyspnea and chext ray clearly shows cardiomegaly C/T ratio > 0.5. Then patientreferredto our institutefor futher evaluation and management. Here patient admitted in ICU and ECG and TTE was done. TTE shows situs solitus, levocardia , ivc and svc drain to RA and all pulmonary vein drain to LA , AV- VA concordance , PFO present with left to right shunt , IVS intact , dilated LA and LV , no TR and No MR , No PAH , moderate LV dysfunction ,EF – 42% ,FS – 20 %, Dd/Ds – 41/33 mm, hypokinetic LAD and LCx territory , dilated RCA with fusiform aneurysm of RCA ,RCA at (origin -3.3mm , aneurysm -5.8mm, distal RCA – 4.2mm) , dilated LMCA with large aneurysm of LMCA , LMCA at (origin -4mm LMCA aneurysm – 6.7 mm) , left aortic arch , no PDA and co-arctation (suggestive of old kawasaki disease) and ECG shows regular, narrow complex QRS , with T-wave inversion in anterior lead later multislice CT angiography was done which shows LMCA aneurysm (post ostel fusiform aneurysm dilation of 9*9 mm) LAD not opacified (suggestive of complete occlusion)circumflex appear normal , RCA aneurysm (oeteo- proximal fusiform aneurysm dilation ; max size 7*6 mm) . Invasive coronary angiography was done to confirm CT finding and LAD obstruction, which also shows the same result. Coronary revascularization was planned.

PRE OP MANAGEMENT

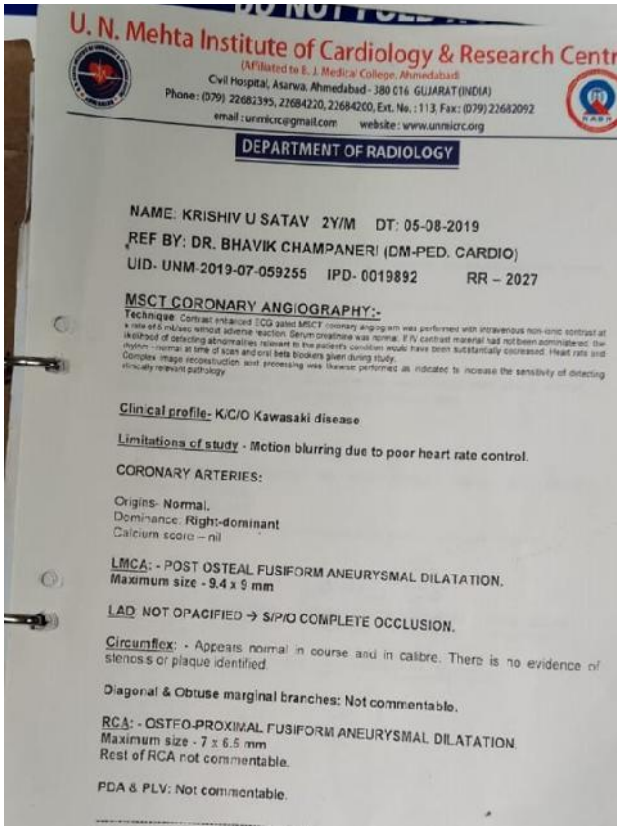
During PAC patient was stable, no sign of any active disease, vital sign within normal limit.If patient had sign of active kawaski disease (polymorphic rashes, change in mucosal membrane and extremities, conjuctival erythema , cervical lymphoadenopathy) then we had to manage symptomatically and with immunoglobulin before taking in OT .

INTAROPRATIVE MANAGEMENT

After shifting to OT, all ASA standard monitor attached, inhalational induction 4 % sevoflurane plus 100% oxygen was performed and 22G I.V peripheral cannula placed in right upper limb and mild sedation of midazolam 0.05mg/kg ,fentanyl5µg/kg and vecoronium 0.1mg/kg loading given . Assisted ventilation with face mask continue till 3 min during that radial line of 22G in right forearm placed and attached to transducer and invasive blood pressuremonitored, then patient intubated with 5.5mm ID endotracheal tube after direct laryngoscopy and tube fixed at 14 cm at right oral commissar, equal air entry confirmed in both lung. It was connected to anaesthesia workstation on pressure controlled ventilation mode (with setting of: - Pinsp 14 cmH2O, respiratory rate 20 breath per minute, PEEP 3cmH2O, I: E ratio 1:2, FiO2 50 %). Then 5 FR CVP line placed in right internal jugularvein. Appropriate inotropic support wasattached and distal port attached to transducer for CVP monitoring. Patient was catherize with foleys catheter of 8 fr for urine outputmonitoring.

ECG, Pulseoximetery, IBP, CVP, Temperature , Capnography, spirometery , gas analyzer , airway pressure, all displayed on monitor. The anaesthetic maintenance was performed with 1.5-2% sevoflurane, fentanyl repeated 10 µg every 30 min and vecuronium in 45 min. Transesophageal echocardiography was done which shows severe left ventricular dysfunction with EF = 10-15 % with global hypokinesia more over to distribution of LAD . Levosimendane started intraoperatively. Painting and draping of recommended part was done in sterile manner and surgery started with additional 20 µg of fentanyl was given before incision and baseline ACT measured. After sternotomy, all cardiac structure and artery was examined there is no evidence of cardiac anomaly only ectasia of RCA and LMCA seen and plan to do LIMA to LAD graft. Patient positioned for LIMA extraction and 100u/kg heparin given, after good extraction patency and flow was confirmed. Aortic and right atrial cannula placed after giving full dose of heparin of 400u/kg; cardiopulmonary bypass was established with moderate hypothermia and alphastat gas handling and 100 ml PCV used in priming along with priming solution; for myocardial protection, cold antegrade blood cardioplegia was given; for renal protection 0.5 g/kg mannitol was given on CPB.

LIMA to LAD was grafted successfully then hot shot given and re-warming started. Under stable condition of sinus rhythm, temperature (36 ^{0}C) hematocrit (34 %), stable hemodynamic and acid base balance, CPB discontinued without any event. Venous and Arterial cannula were removed, the effect of heparin was reversed with protamine with ACT of 128 s. When surgery finished patientshifted to post op PICU, on controlled ventilation with infusion of adrenaline 0.03- 0.1 µg/kg/min .



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DEPARTMENT OF RADIOLOGY

NAME: KRISHIV U SATAV 2Y/M DT: 05-08-2019
REF BY: DR. BHAVIK CHAMPANERI (DM-PED. CARDIO)
UID- UNM-2019-07-059255 IPD- 0019892 RR – 2027

MSCT CORONARY ANGIOGRAPHY:-

Technique: Contrast enhanced ECG gated MSCT coronary angiogram was performed with intravenous non-ionic contrast at a rate of 5 ml/sec without adverse reaction. Serum creatinine was normal. If IV contrast material had not been administered, the likelihood of detecting abnormalities relevant to the patient's condition would have been substantially decreased. Heart rate and rhythm – normal at time of scan and oral beta blockers given during study. Complex image reconstruction and processing was likewise performed as indicated to increase the sensitivity of detecting clinically relevant pathology.

Clinical profile- K/C/O Kawasaki disease

Limitations of study - Motion blurring due to poor heart rate control.

CORONARY ARTERIES:

Origins- Normal.
Dominance: Right:-dominant
Calcium score – nil

LMCA: - POST OSTEAL FUSIFORM ANEURYSMAL DILATATION.
Maximum size - 9.4 x 9 mm

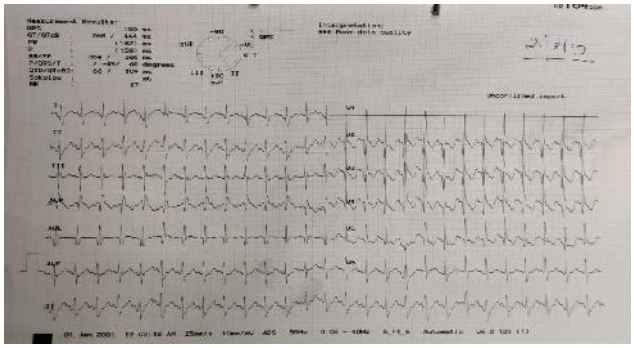
LAD NOT OPACIFIED → S/P/O COMPLETE OCCLUSION.

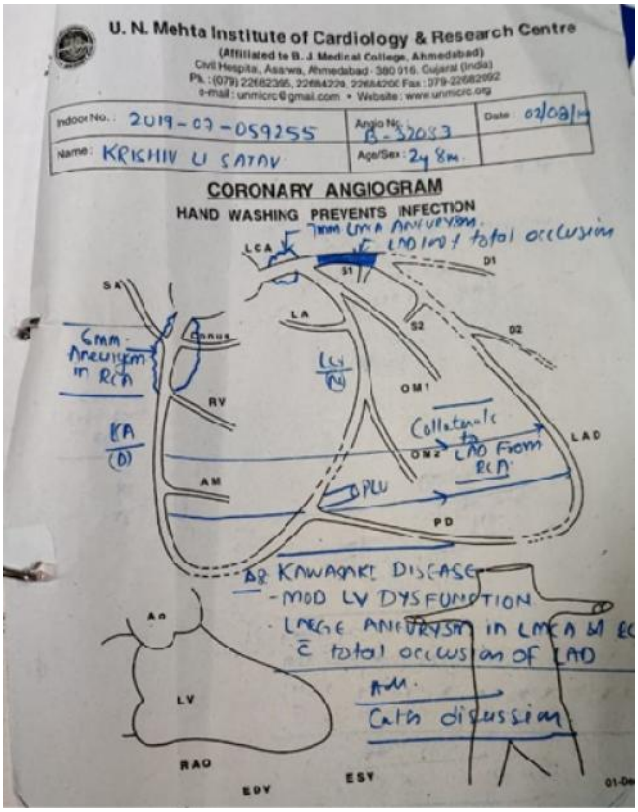
Circumflex: - Appears normal in course and in calibre. There is no evidence of stenosis or plaque identified.

Diagonal & Obtuse marginal branches: Not commentable.

RCA: - OSTEO-PROXIMAL FUSIFORM ANEURYSMAL DILATATION.
Maximum size - 7 x 6.5 mm
Rest of RCA not commentable.

PDA & PLV: Not commentable.





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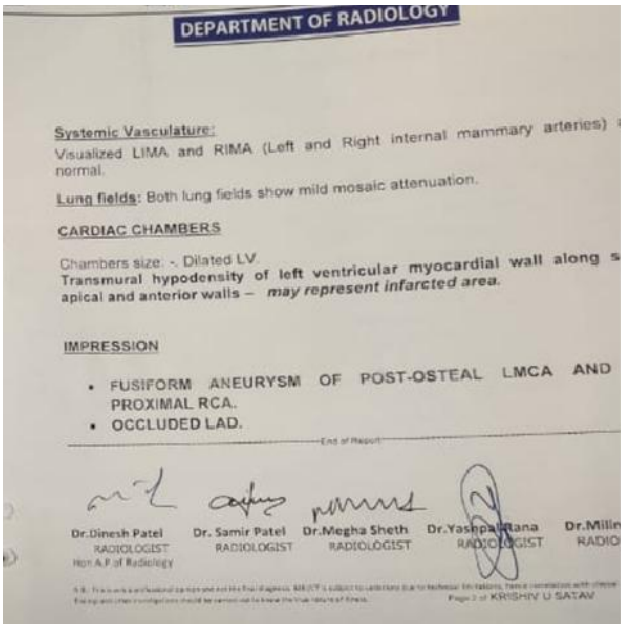
Indoor No.: 2019-07-059255 | Angio No.: B-32053 | Date: 02/08/19
Name: KRISHIV U SATAV | Age/Sex: 2y 8m

CORONARY ANGIOGRAM
HAND WASHING PREVENTS INFECTION



DEPARTMENT OF RADIOLOGY

Systemic Vasculature:
Visualized LIMA and RIMA (Left and Right internal mammary arteries) normal.

Lung fields: Both lung fields show mild mosaic attenuation.

CARDIAC CHAMBERS

Chambers size: -. Dilated LV.
Transmural hypodensity of left ventricular myocardial wall along s apical and anterior walls – *may represent infarcted area.*

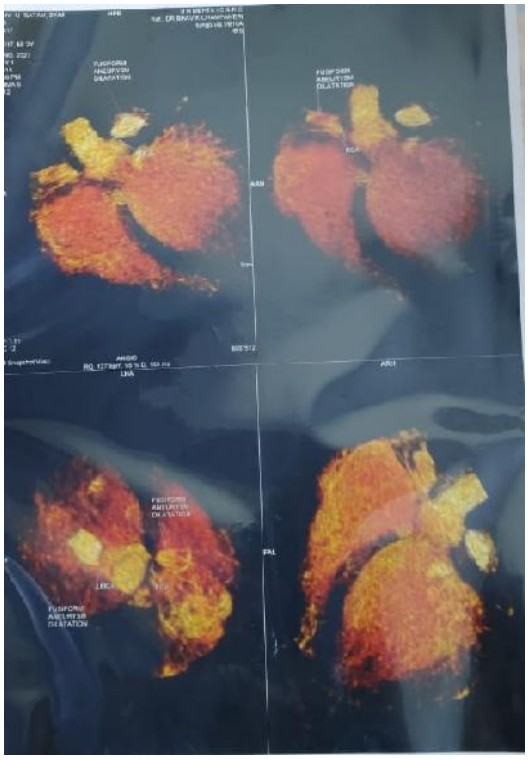
IMPRESSION

- FUSIFORM ANEURYSM OF POST-OSTEAL LMCA AND PROXIMAL RCA.
- OCCLUDED LAD.

End of Report

Dr.Dinesh Patel RADIOLOGIST Hon A.P of Radiology
Dr. Samir Patel RADIOLOGIST
Dr.Megha Sheth RADIOLOGIST
Dr.Yashpal Rana RADIOLOGIST
Dr.Milin RADIO

KRISHIV U SATAV



POST OPERATIVE MANAGEMENT

In picu vital was stable on adrenaline, levosimendane, noradrenaline infusion. Dexmeditomidine was started at variable dose of 0.5 µg/kg/min. Patient was ventilated for one day. Fluid given according to body surface area. TTE done on 2nd day which shows improvement in heart contractility.Inotropic support was tapered off according to mean blood pressure and hemodynamic stability. Unfractioned heparinwas given at 10 IU/kg/hr with subsequent adjustment according to APTT.When hemodynamic and arterial blood gas was within normal limit and follows extubation criteria. Then, we slowly wean off ventilator according our institutional protocol and extubated successfully on 2nd day.

Site(s) view / vol/ rate : Left & Right Coronary Angio

PTCA/LAB : Guiding Catheter : Balloon : Guidewire : Stent(s) : Time : Guidewire : dia & length Pressure Pressure

VALVULOPLASTY : Balloon ____ Size ____ Length Inflation - mi

Report :

Catheter Course:

Right Femoral Artery: SF sheath -> DTA -> Arch -> Aortic root

Procedure: RCA hooked with 4F JR catheter, left system can not be hooked, non-sele

shoot taken with 4F LIMA kept in left coronary cusp

LMCA : Large aneurysm of size 7 x mm

LAD : Proximal total occlusion with retrograde filling from RCA

LCX : Normal

RCA : Proximal large aneurysm of 6 mm

LV Angio : Not done

Renal : Not done

Final Diagnosis:

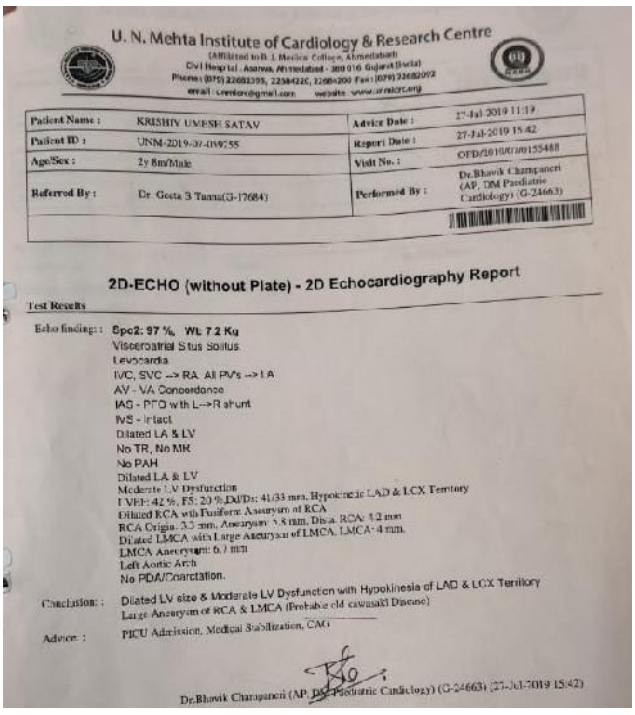
CAD : KAWASAKI Disease

Moderate LV Dysfunction

Large aneurysm of RCA & LMCA with LAD total occlusion

Recommendation:

Cath discussion



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Patient Name :	KRISHIV UMESH SATAV	Advice Date :	27-Jul-2019 11:19
Patient ID :	UNM-2019-07-019255	Report Date :	27-Jul-2019 15:42
Age/Sex :	2y 8m/Male	Visit No. :	OPD/2019/07/019255488
Referred By :	Dr. Geeta B Tanna(G-17684)	Performed By :	Dr.Bhavik Champaneri (AP, DM Paediatric Cardiology) (G-24663)

2D-ECHO (without Plate) - 2D Echocardiography Report

Test Results

Echo findings : Spo2: 97 %, Wt: 7.2 Kg
Visceroatrial Situs Solitus
Levocardia
IVC, SVC --> RA. All PV's --> LA
AV - VA Concordance
IAS - PFO with L-->R shunt
IVS - Intact
Dilated LA & LV
No TR, No MR
No PAH
Dilated LA & LV
Moderate LV Dysfunction
LVEF: 42 %, FS: 20 % Dd/Ds: 41/33 mm, Hypokinetic LAD & LCX Territory
Dilated RCA with Fusiform Aneurysm of RCA
RCA Origin: 3.3 mm, Aneurysm: 5.8 mm, Dista. RCA: 3.2 mm
Dilated LMCA with Large Aneurysm of LMCA, LMCA: 4 mm,
LMCA Aneurysm: 6.7 mm
Left Aortic Arch
No PDA/Coarctation.

Conclusion : Dilated LV size & Moderate LV Dysfunction with Hypokinesia of LAD & LCX Territory
Large Aneurysm of RCA & LMCA (Probable old kawasaki Disease)

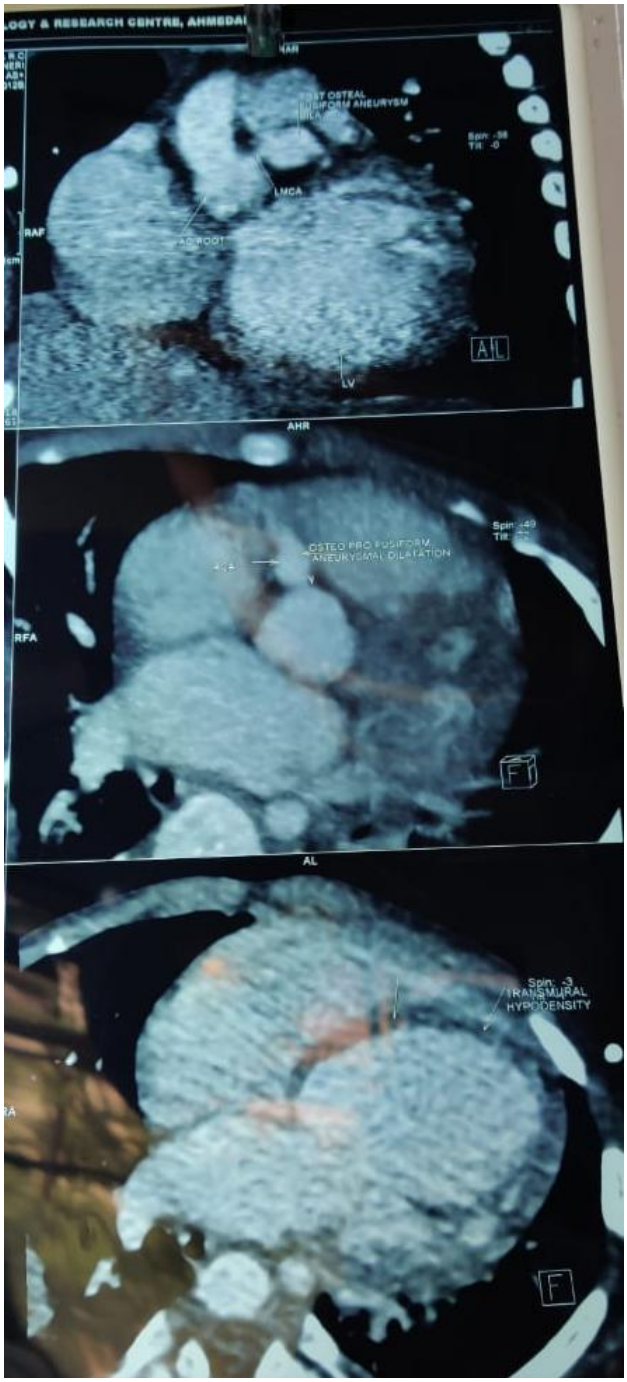
Advice : PICU Admission, Medical Stabilization, CAG

Dr.Bhavik Champaneri (AP, DM Paediatric Cardiology) (G-24663) (27-Jul-2019 15:42)

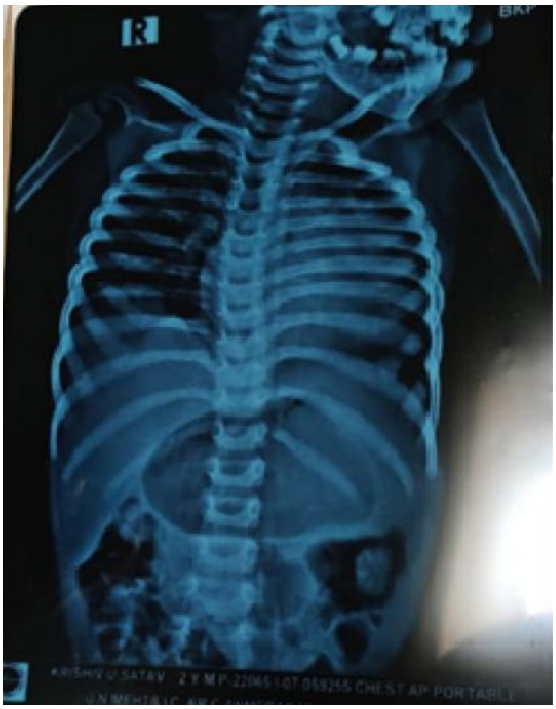
Patient was shifted to pediatric ward after 4 days then under good general condition discharged 3day after with indication of aspirin (3-5mg/kg) orally once a day.

DISSUSION

Coronary revascularization is indicated when myocardial ischemia due to significant coronary stenosis is present and causing chest pain or there is clinical evidence of ischemia on examination (9). In this patient angiogram detected aneurysm of RCA and left circumflex artery and LAD complete occulusion . and retrograde filling by collaterals via RCA .



Its etiologic pathogen andassociated immunological reactions have yet to beclarified. Immediately after commencement of the clinical symptoms fever and typical lesions of either the mucous membrane or the skin, dilatation of the coronary arteries can occur in approximately 10±20% of patients. In half of these patients, the aneurysms are regressing within 1 or 2 years, and no abnormalities are found by angiography in the coronary- arterial system. In the remaining, the aneurysms can persist with obviously irregular lumens of the coronary arteries. In 3% of the patients initially having aneurysms, coronary-arterial obstruction progresses. The time-span between the onset of the disease and development of the coronary-arterial stenosis leading to CABG varies from several months to 20 years. The indications for CABG are to be determined not only by ®ndings derived from angiography, but also on the basis of other clinical factors, such as severity of myocardial ischemia, history of myocardial infarction and ventricular performance, as have been discussed before (10-13).



Successful CABG using SVG for this disease was first reported by Kitamura et al. in 1976 (14), and they also reported the efficacy of the use of ITA as a bypass graft in pediatric patients in 1985 (15). In our case, improvements in functional status in the intermediate term after such surgical procedures are undoubtedly encouraging. It should be noted, however, that the patency rate of SVG in the longer terms was less than ideal, particularly in small children. Presumably, degeneration of SVG progresses more frequently and more rapidly in small children. The growth in the patient's body size, furthermore, can be another factor of occlusion of the bypass graft. The arterial graft can grow (15), while SVG may not.

CONCLUSION

We report a case of kawasaki disease with aneurismal coronary artery. Patient is symptomatic due to low ejection fractionand recurrent pneumonia . CABG was indicated due to obstructed LAD and LIMA grafed to LAD which significantlyimproves flow across LAD and also improve ejection fraction in post op recovery area. It is one of several possible procedures to avoid the risk of future cardiac events in adulthood. Recommendation of such an intervention is a difficult decision with multiple considerations and individualization.

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