International Journal of Science and Research (IJSR)
ISSN: 2319-7064
ResearchGate Impact Factor (2018): 0.28 | SJIF (2019): 7.583

Successful Endovascular Stenting of the Inferior Venacava (IVC) in a Patient with Budd-Chiari Syndrome (BCS)

Dr. P. Sampath Kumar, Dr. V. S. R Bhupal, DR. K. C. Karthik Naidu, Dr. G. Pranoy

Abstract: ***A 60 year Old man an Agricultural labourer by occupation had Distension of Abdomen, Prominent of veins over abdomen & Swelling of both legs with excoriation for the past 8 years was diagnosed as Budd chiari syndrome with IVC Obstruction. MR venogram showed focal short segmental stenosis causing 80-90 % luminal narrowing of IVC at diaphragmatic hiatus region with mild downstream dilatation of IVC which is 33 mm. IVC angiogram was done which showed obstruction at the junction of IVC and right atrium with thickness of membrane around 5mm. IVC SELF EXPANDING WALL STENT (18 x 60mm) was done and IVC flow was restored. patient symptoms reduced & he is on regular follow up.***

Keywords: PT-INR (Prothrombin-International Normalized ratio), ECG (Electrocardiogram), CBD(common Bile duct), IHBD (intra-Hepatic biliary Dilatation), PCS (pelvicalyceal System), CMD (Cortico Medullary differentiation), IVC (Inferior venacava), SFV (superficial femoral vein), IJV (internal jugular vein), MPA (Multipurpose) LAO (left anterior Oblique), RA (Right Atrium), RV (Right Ventricle), LA (Left Atrium)

1. Introduction

A 60 year Old man an Agricultural labourer by occupation had Distension of Abdomen, Prominent of veins over abdomen & Swelling of both legs with excoriation for the past 8 years was diagnosed as Budd chiari syndrome with IVC Obstruction. MR venogram showed focal short segmental stenosis causing 80-90% luminal narrowing of IVC at diaphragmatic hiatus region with mild downstream dilatation of IVC which is 33 mm. IVC angiogram was done which showed obstruction at the junction of IVC and right atrium with thickness of membrane around 5mm. IVC SELF EXPANDING WALL STENT (18 x 60mm) was done and IVC flow was restored. patient symptoms reduced & he is on regular follow up.

A 60 year Old man Mr. X, An Agricultural labourer presented to the hospital with complaints

1) Distension of Abdomen,
2) Prominent of veins over abdomen,
3) Swelling of both legs with excoriation for the past 8 years.

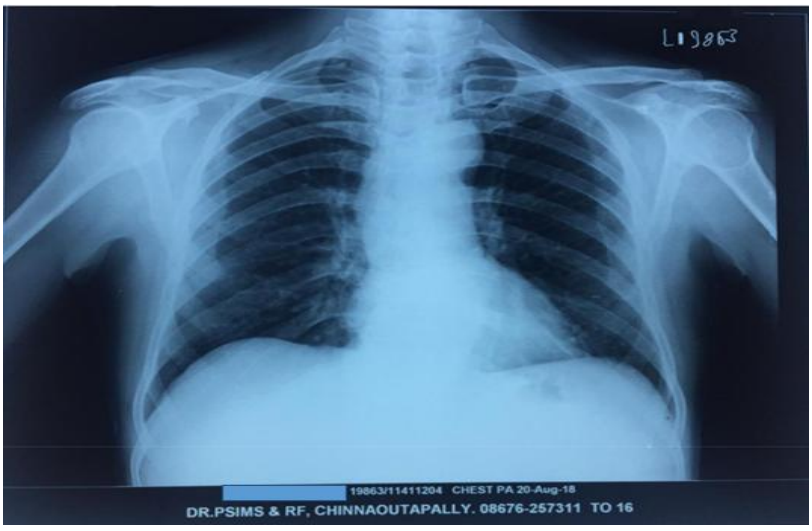
Routine investigations

Hemoglobin	9.6 g%
Total count	7500 cells/cumm
Serum creatinine	1.1 mg/dl
Serum Sodium	146 meq/L
Serum Potassium (K+)	4.4 meq/L
PT – INR	1.73
Viral markers	Non Reactive

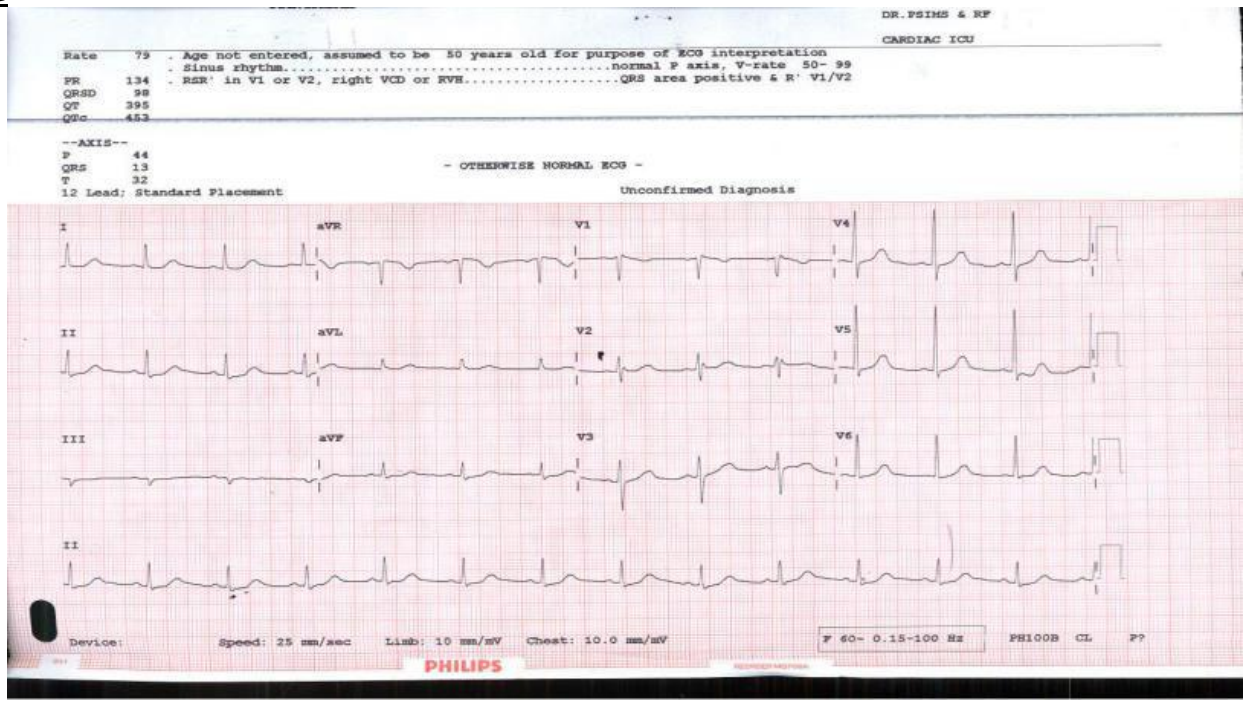
LFTs (Liver function tests)

Serum Total bilirubin = 2.5mg%
Direct Bilirubin = 1.0 mg%
Indirect bilirubin = 1.5 mg%
SGOT = 200 IU/L
SGPT = 256 IU/L
Alkaline phosphatase = 125 IU/L
Serum Total proteins = 4.8 g%
Serum Albumin = 2.8 g%
Serum Globulin = 2.0 g%
A/G ratio = 1.4

Roentogram



ECG

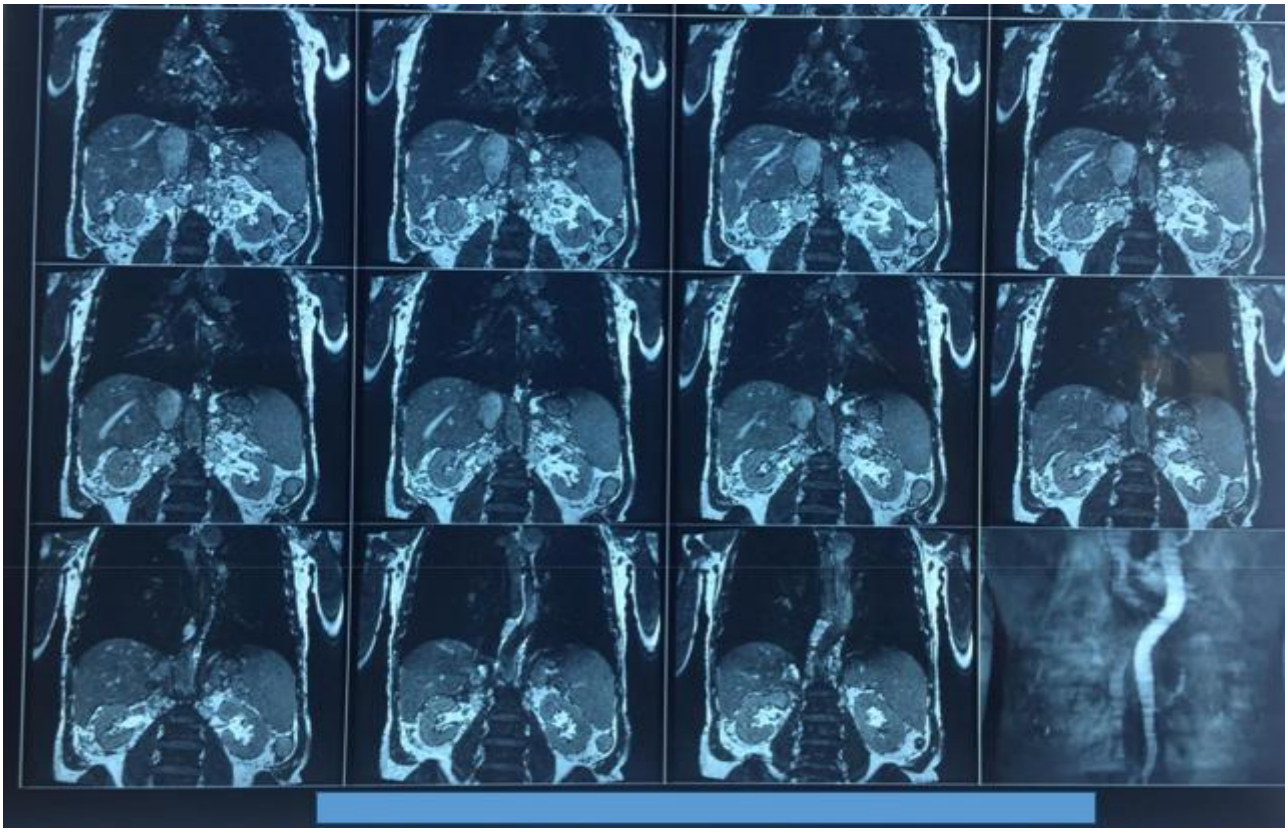


Ultrasound abdomen (21-08-18)

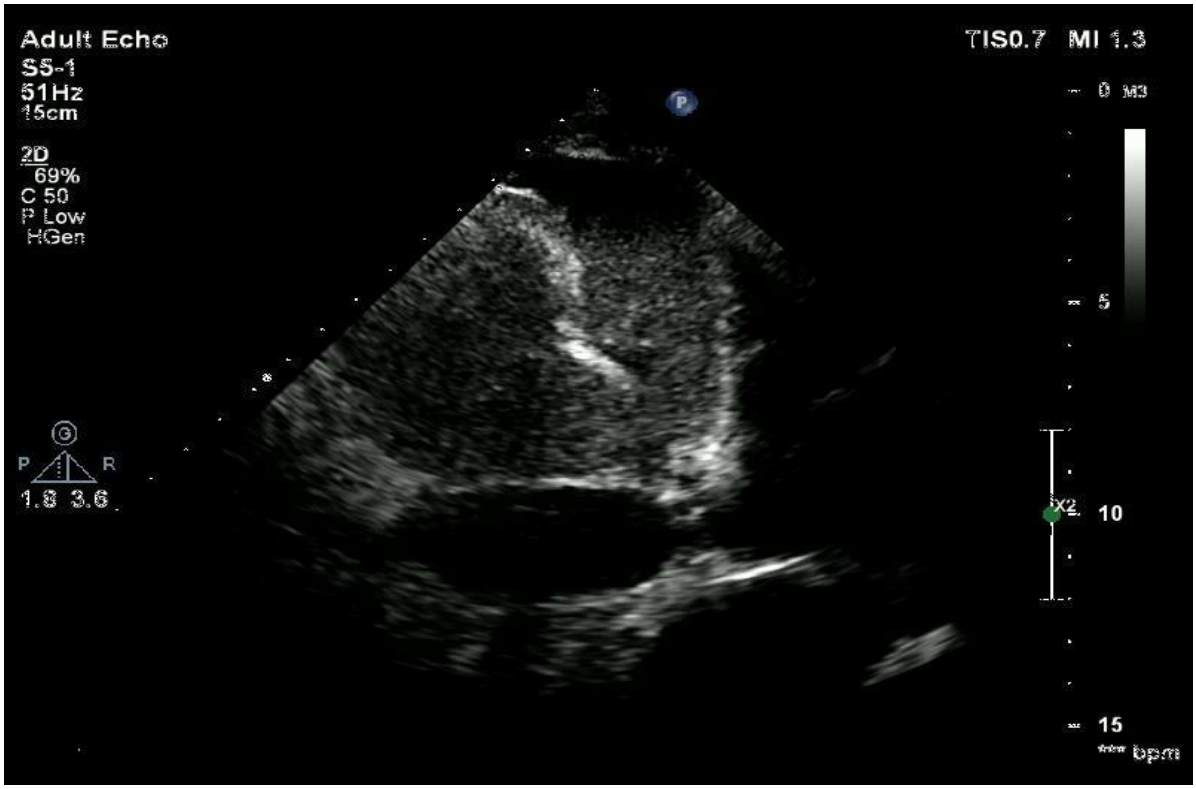
- **Liver**–measures 14.8cm .Normal in Size with **shrunken right lobe** & **hypertrophied Caudate lobe & coarsed Echopattern**
  No IHBD. PV = 13.9mm, CBD – Normal caliber
- **Gallbladder**–Normal
- **Spleen**–Enlarged in size measuring 15.0cm .shape and echo pattern are normal
- **Kidneys**–Normal in size and echopattern. PCS normal .CMD made out and No calculi **Urinary bladder**–Distended. No calculi noted
- **Impression**- Cirrhosis of Liver with Splenomegaly

MRI VENOGRAM OF IVC-

a) Features of cirrhosis of liver with portal hypertension
b) Showed **focal short segmental stenosis** causing 80-90% luminal narrowing of IVC at diaphragmatic hiatus region with mild downstream dilatation of IVC which is 33 mm.
c) Overall features probably represent –Budd chiari syndrome with cirrhosis of liver

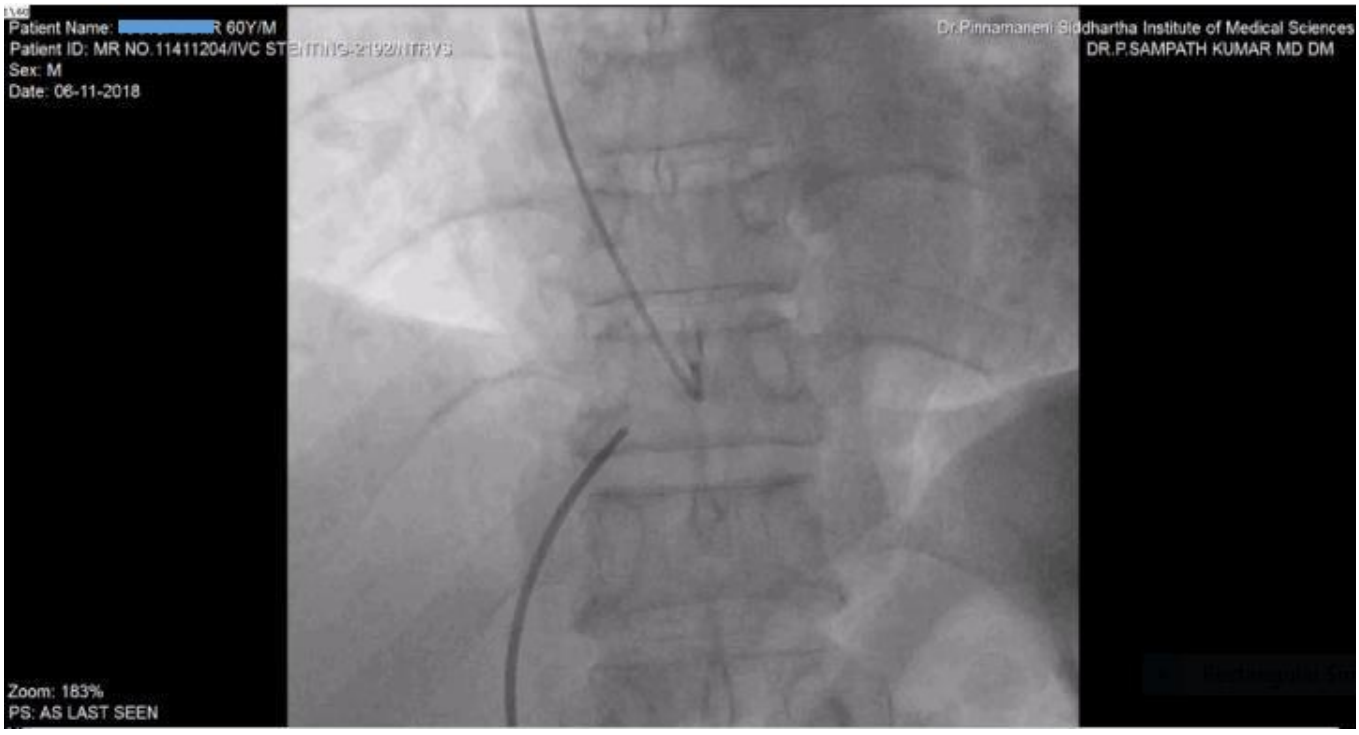


- **2D ECHO**-Dilated IVC& narrowing of IVC before entering into rightatrium.
- Good LV function
- No LV RWMA
- NO MR/AR/PR/TR
- Grade I Diastolic dysfunction

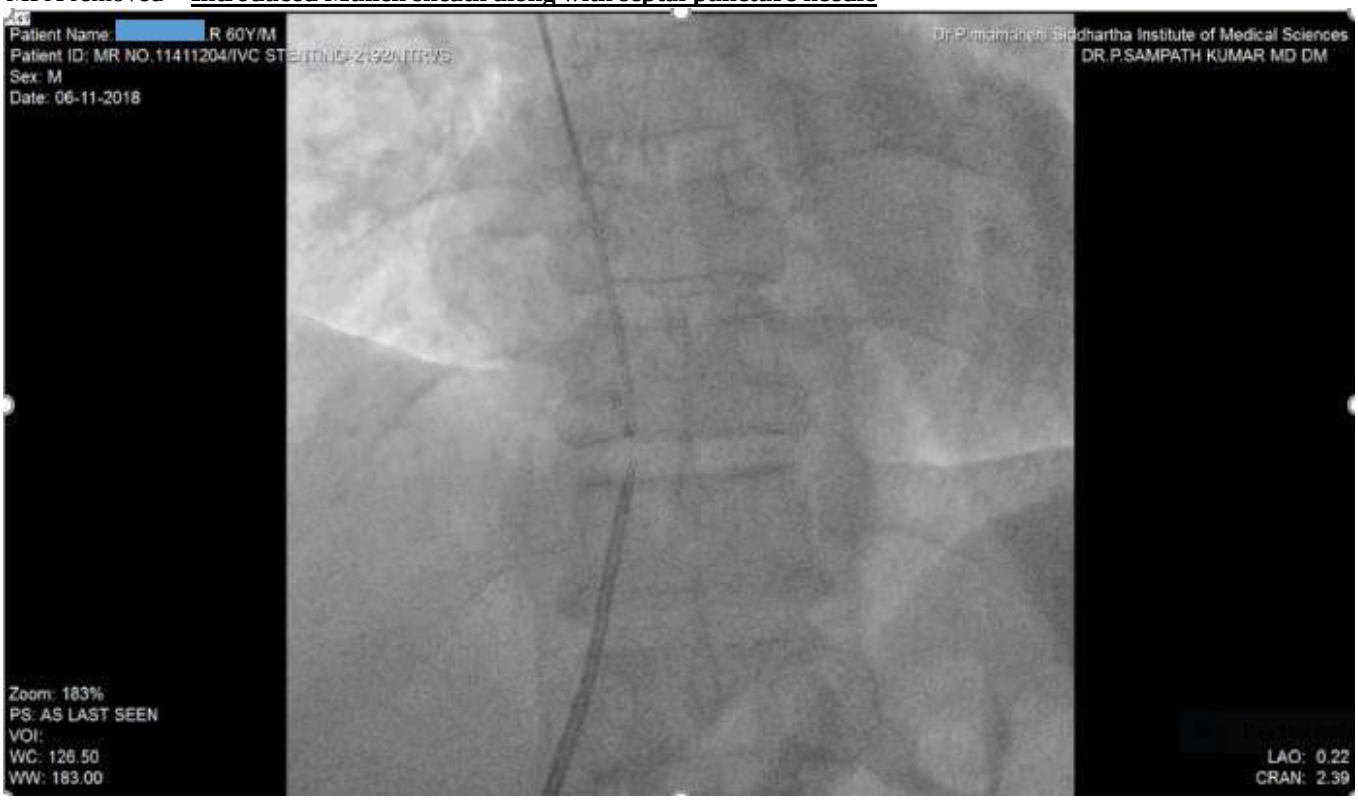


- IVC angiogram was done which showed obstruction at the junction of IVC and right atrium.
- The thickness of the obstruction was assessed by passing catheters from femoral and jugular approach which showed the thickness to be 5 mm.

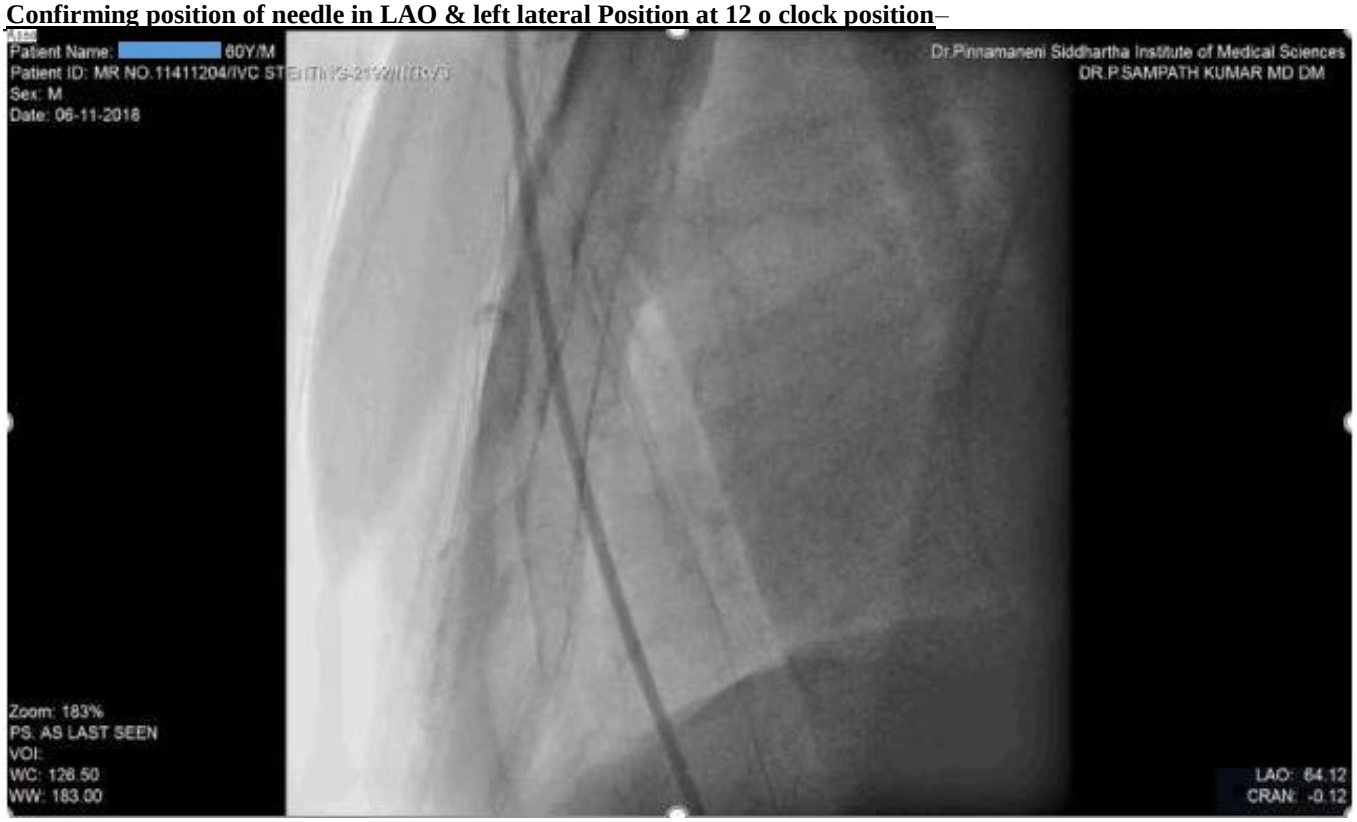
Approach through Right SFV –engaged with MPA1 crossed IVC till the stenotic site **Approach through Right IJV** engaged with JR 3.5 into the RA



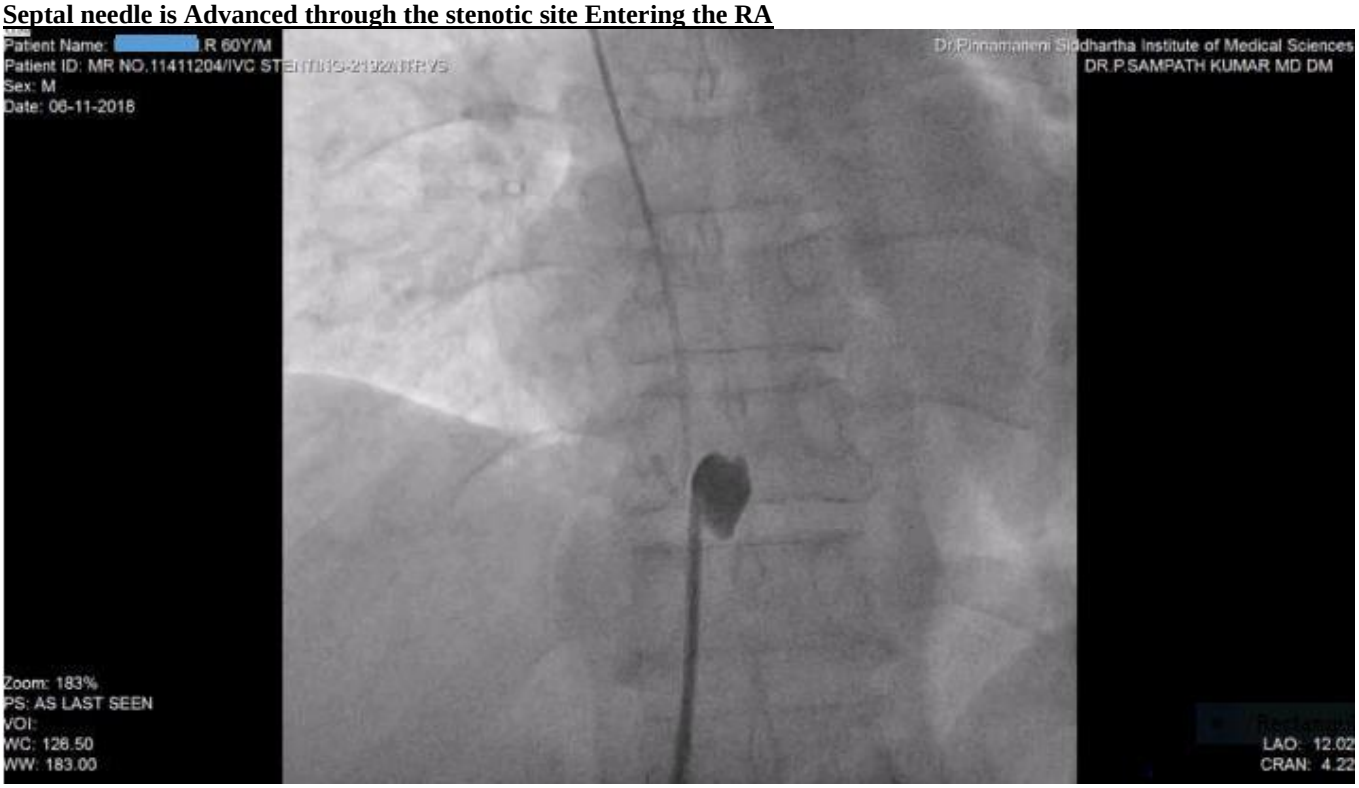
MPA removed – **Introduced Mullen sheath along with septal puncture needle**



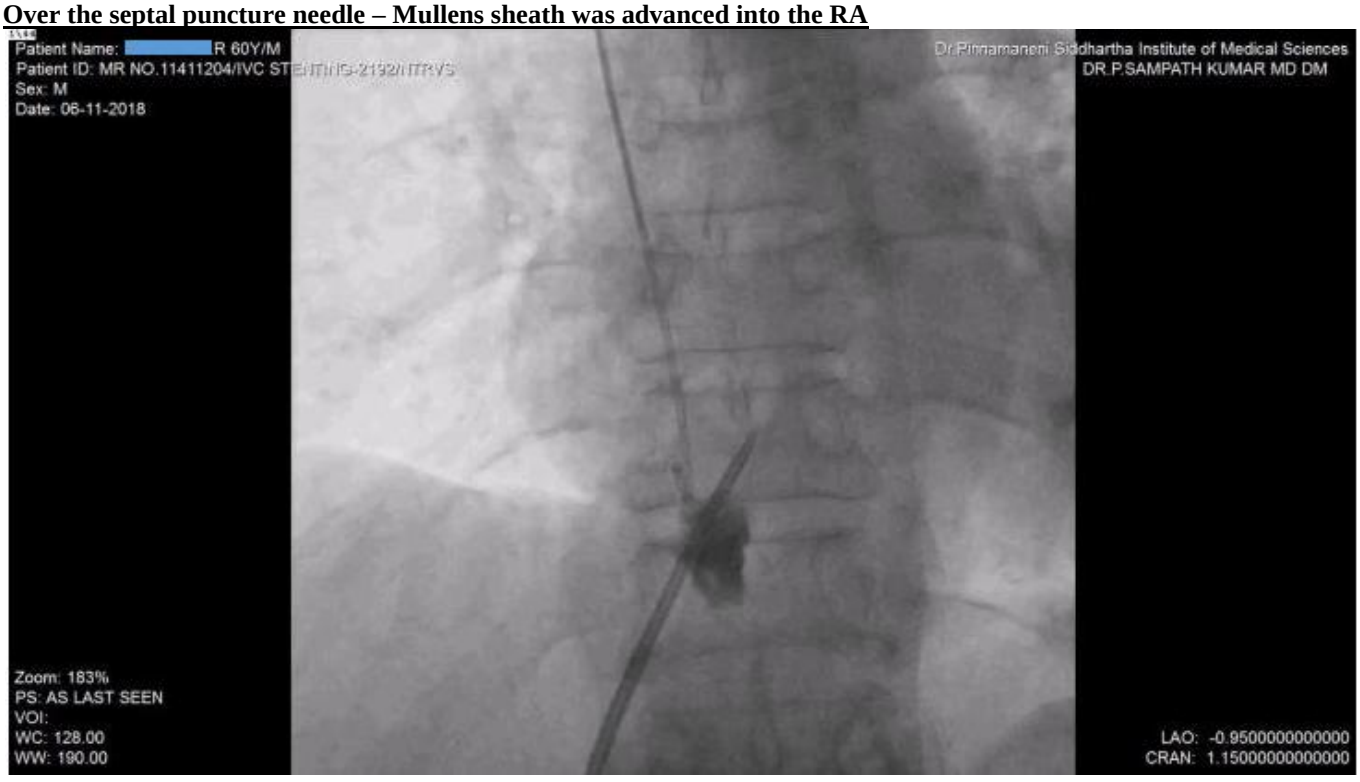
Confirming position of needle in LAO & left lateral Position at 12 o clock position–



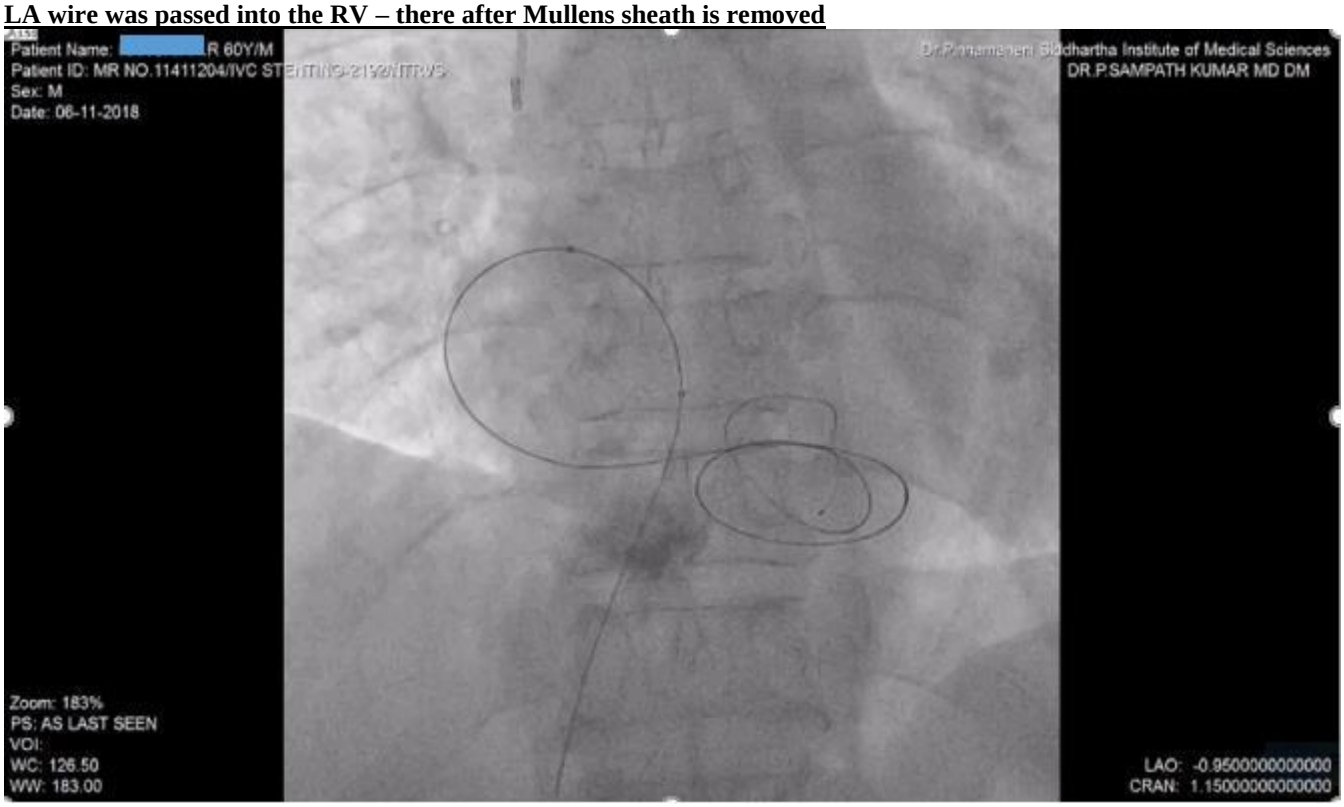
Septal needle is Advanced through the stenotic site Entering the RA



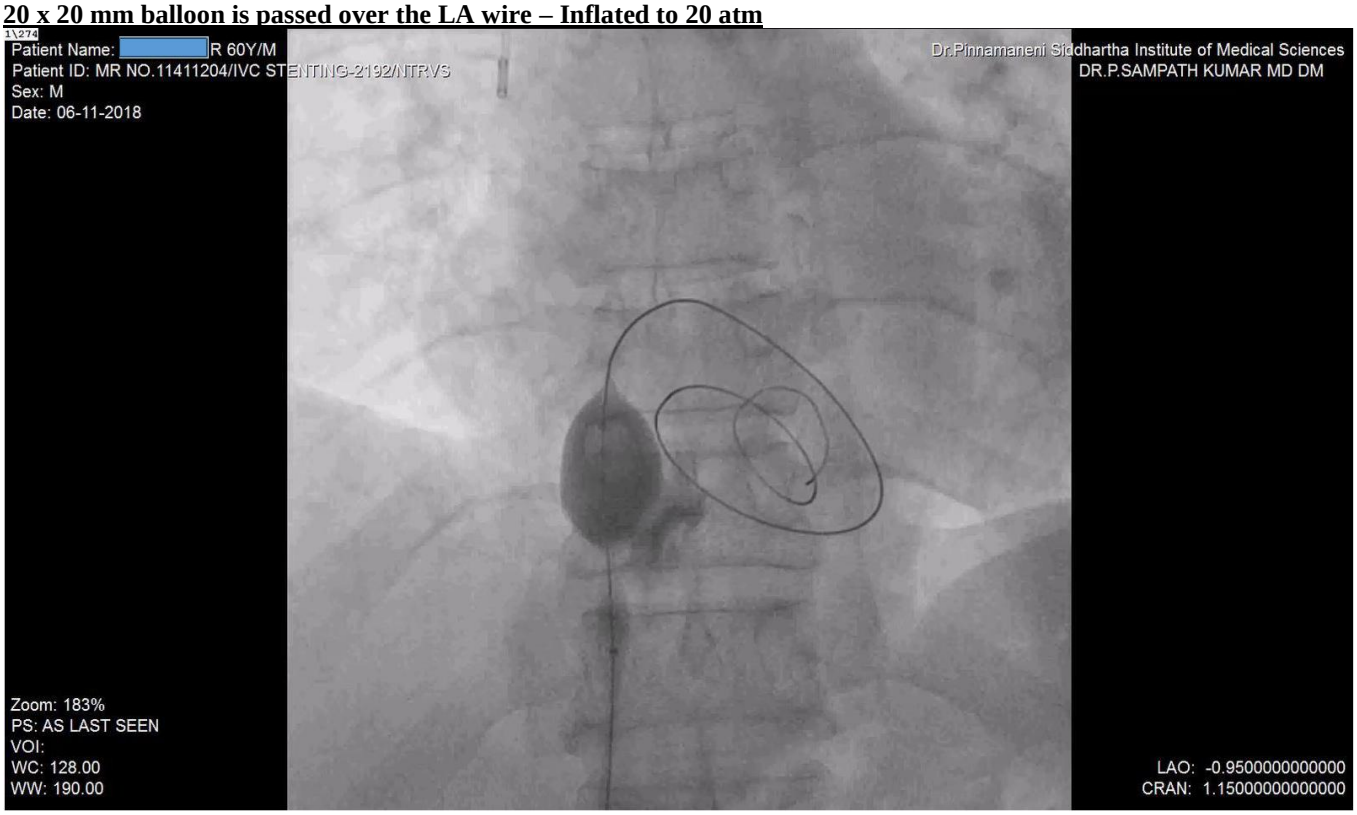
Over the septal puncture needle – Mullens sheath was advanced into the RA



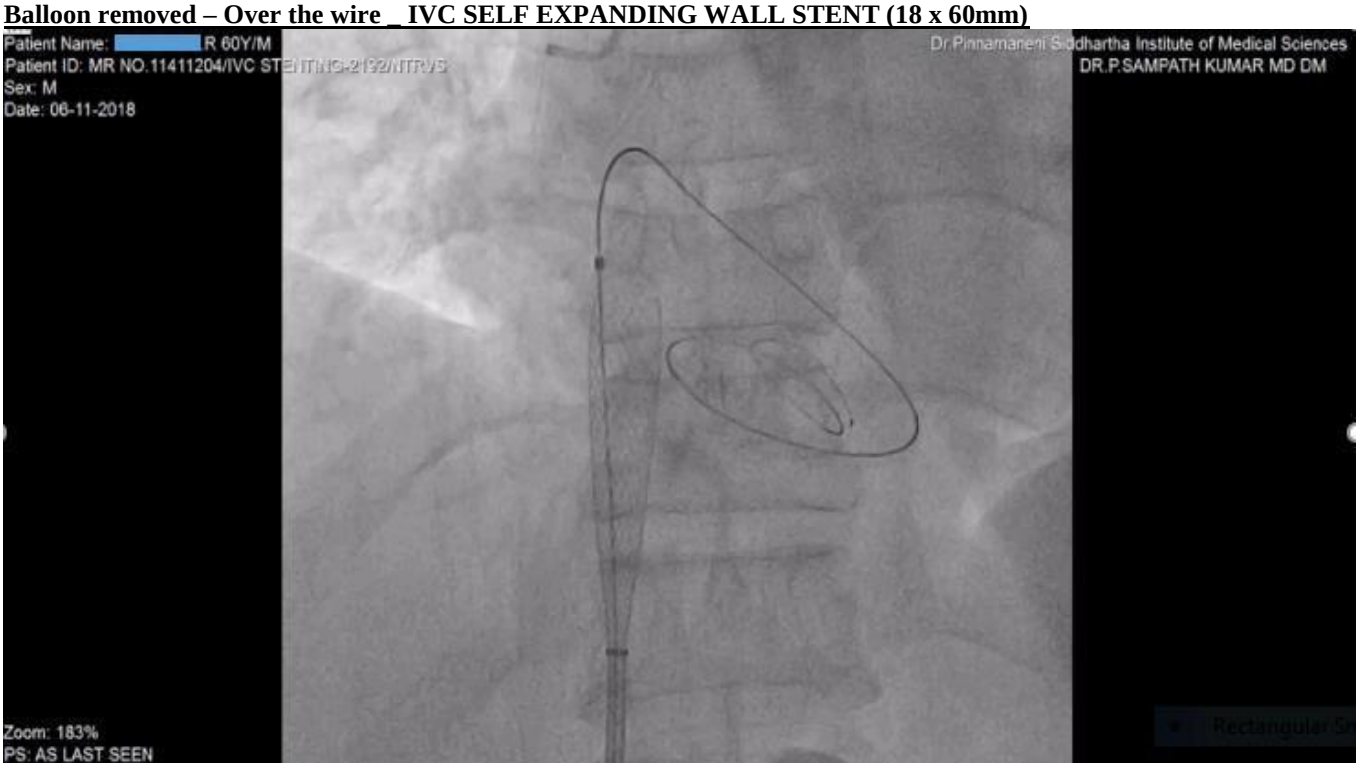
LA wire was passed into the RV – there after Mullens sheath is removed



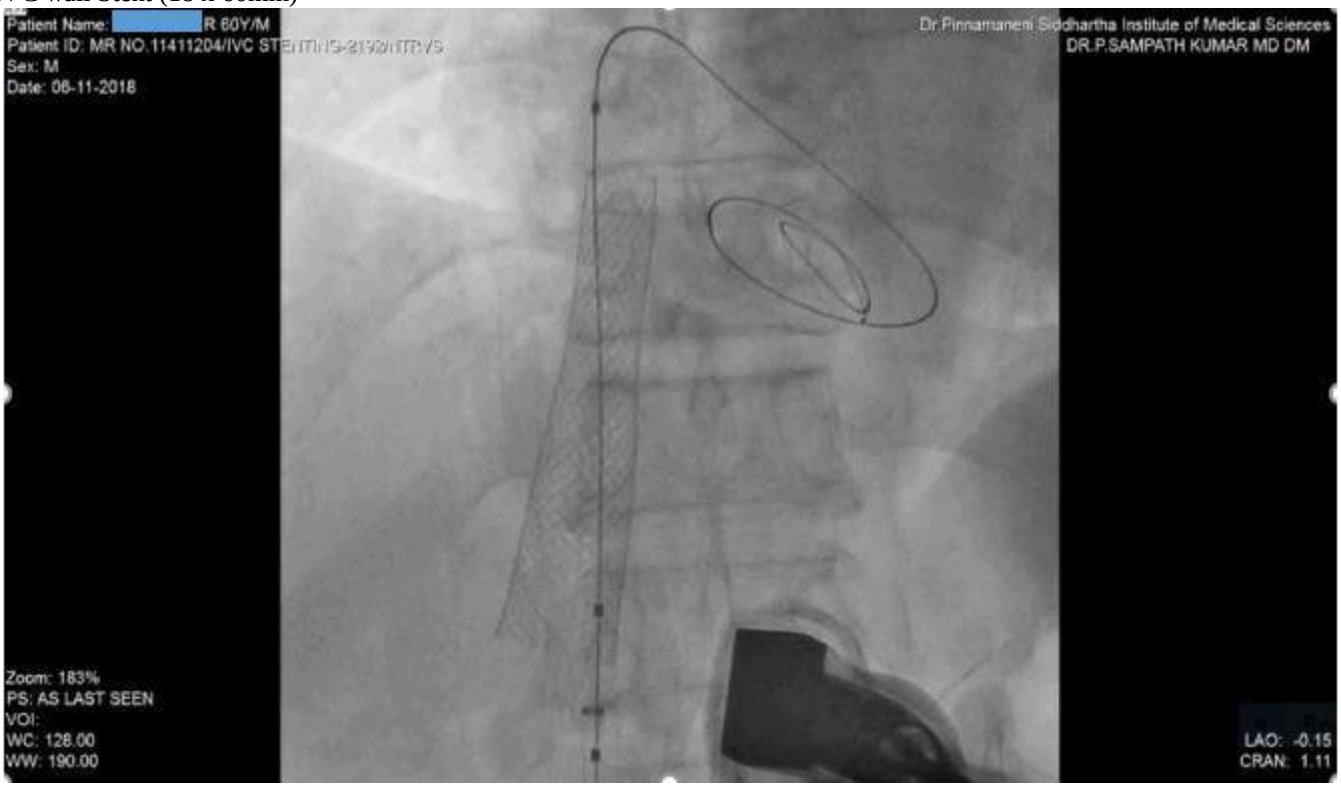
20 x 20 mm balloon is passed over the LA wire – Inflated to 20 atm



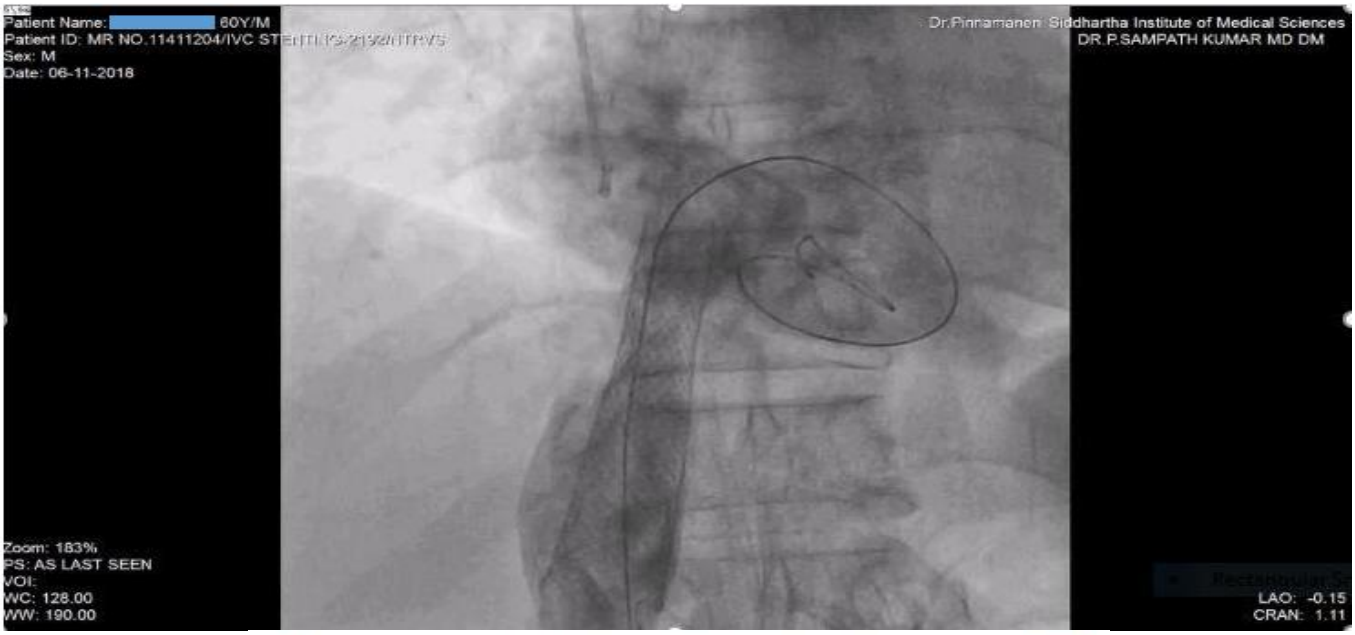
Balloon removed – Over the wire _ IVC SELF EXPANDING WALL STENT (18 x 60mm)



IVC wall Stent (18 x 60mm)



Finally, Contrast was introduced into IVC which showed good flow into the RA



Dr. PINNAMANENI SIDDHARTHA
INSTITUTE OF MEDICAL SCIENCES & RESEARCH FOUNDATION
(Sponsored by Siddhartha Academy of General & Technical Education, Vijayawada)

DEPARTMENT OF CARDIOLOGY

REPORT

DATE: 06-11-2018		MR NO:11411204	CATH NO: 2192
Patient Name	:		Age : 60Y /M
APPROACH	:	Right Femoral Vein & Right jugular	
VESSELTO BE DILATED	:	IVC	
GUIDE WIRE	:	LA WIRE, Mullans sheath	
GUIDING SYSTEM	:	JR 3.5 X 5F,MPA1 X 6F	
STENT	:	18 MM X 60MM X 75CM (WALL STENT)	

IVC-STENT NOTES:

- Approach Right Femoral vein engaged with MPA1 X 6F crossed the IVC till the IVC web.
- Approach through Right IJV engaged with JR 3.5 X 6F into the RA.
- Then simultaneous contrast injected through MPA & JR to know the thickness of the IVC web.
- After removal of MPA through the femoral vein we introduced mullens sheath along with septal puncture needle, then by confirming the position of the needle in LAO & left lateral positions at 12o clock position Septal puncture needle advanced through the IVC web entered into the RA.
- Then over the needle we advanced the mullens sheath into the RA, then the needle was removed and then LA wire was passed into the RV and mullans sheath is removed.
- Now 20mm X 20mm Balloon is passed over the LA wire and Inflated to 20 atm till the waist of the balloon is inflated.
- After removal of balloon, over the wire we introduced the **IVC Self expandable WALL Stent of size 18MM X 60MM** and then the stent is deployed.
- To conform Contrast was introduced through femoral sheath which was showing good flow towards the RA.

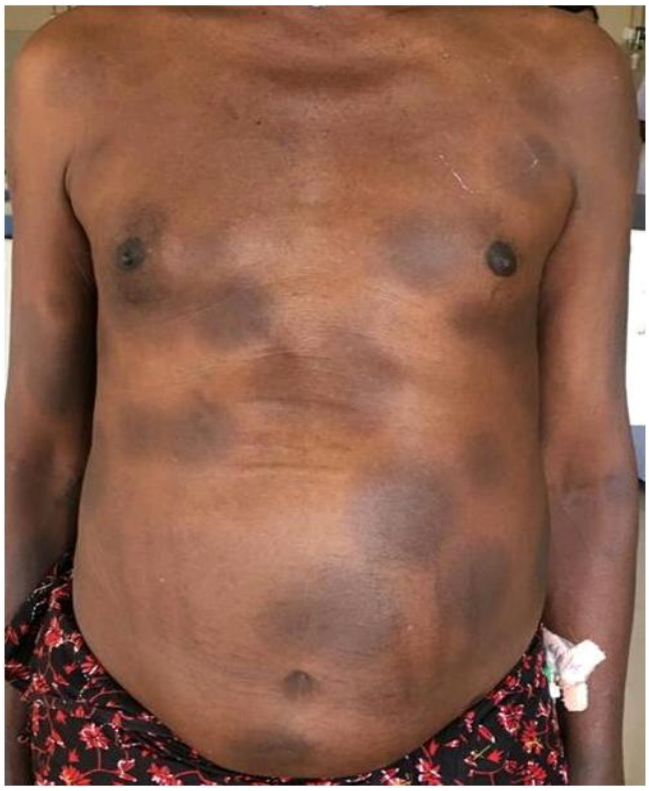
RESULT:

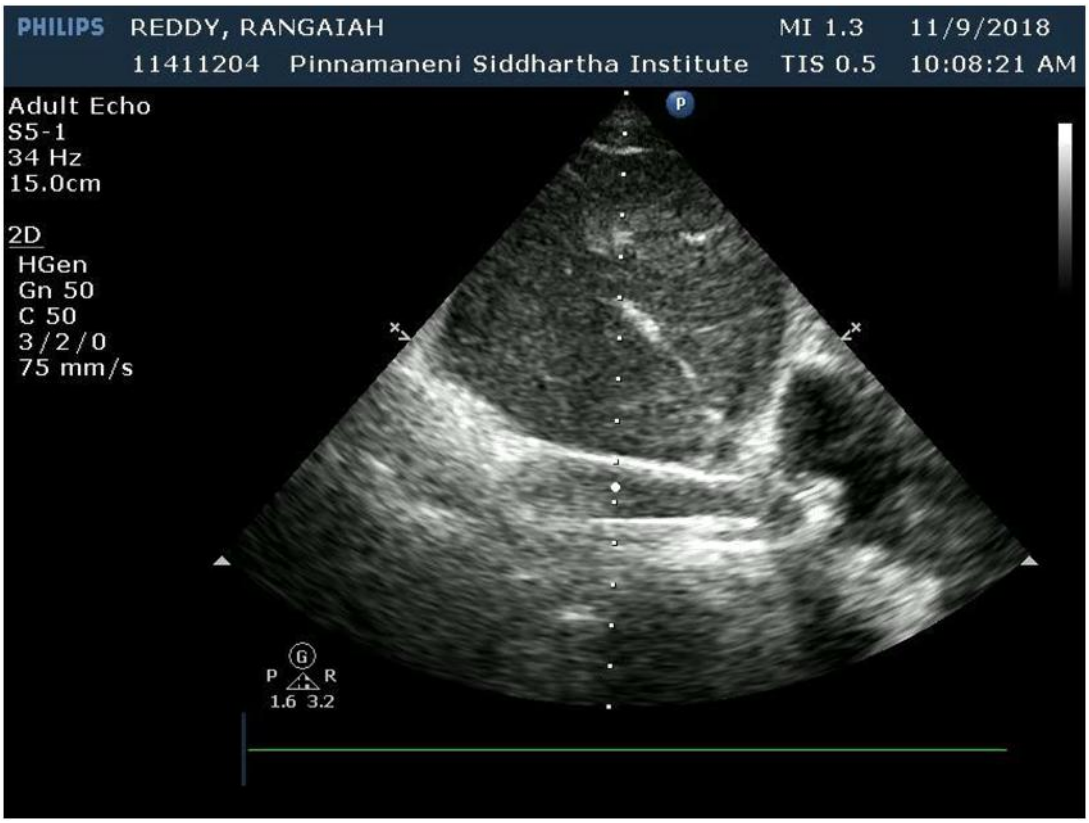
✓ Good position /Good Result

DR.P.SAMPATH KUMAR. MD., DM Professor & HOD
DR.V.S.R.BHUPAL, MD., DM CONSULTANT CARDIOLOGIST
DR.K.C.KARTHIK NAIDU,MD.,DM CONSULTANT CARDIOLOGIST

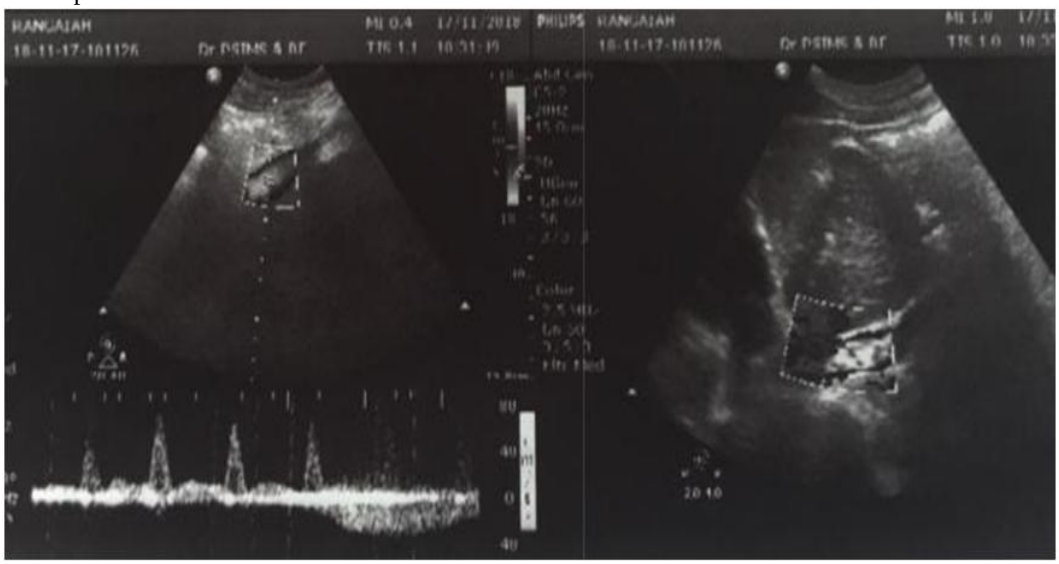
Chinna Avutapalli, Gannavaram Mandal, Krishna Dist., A.P - 521 286
Phone (O) : 91-8676- 257311 to 17, Fax : 91-8676-257222

- Prominent veins over the Abdomen - disappeared with in 1hr of procedure
- The patient began to lose body weight - 1 day after stenting
- Day 2 the patient Ascites and pedal edema slowly disappeared.



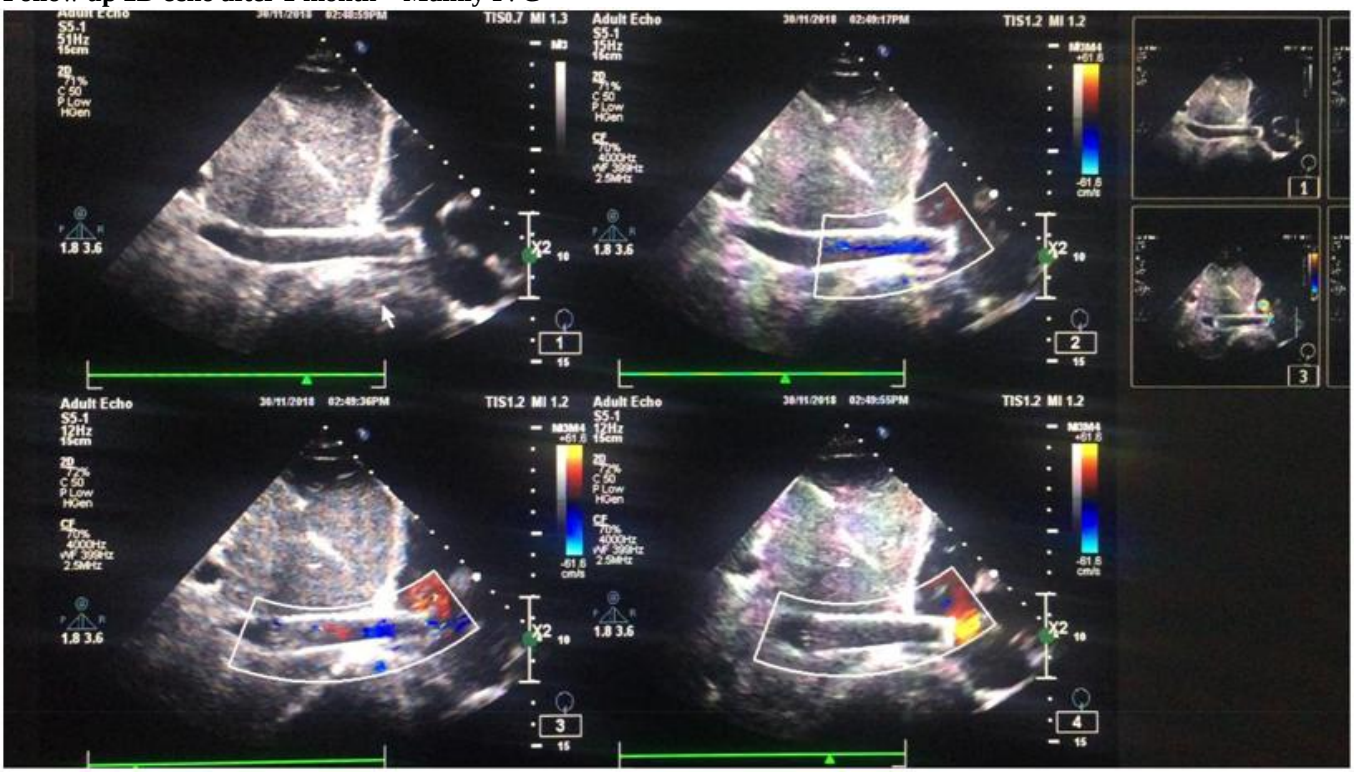


IVC Doppler – Post procedure



1) IVC STENT NOTED INSITU
2) Rest of the IVC shows normal collapse and unidirectional flow
3) No obvious thrombosis noted within visualized part of IVC

Follow up 2D echo after 1 month – Mainly IVC



On treatment &regular follow up

1) Tab. Warf 2mg/3mg alternate days
2) Tab. Atocor CV 75/10 mg po H/S (Atorvastatin 10mg +Clopidogrel 75mg)

Budd-Chiari Syndrome (BCS)

This a rare syndrome occurs in 1/1, 00, 000 in the general population.

Budd Chiari syndrome is a heterogenous group of disorders characterized by Hepatic outflow obstruction at the level of hepatic venules, the large hepatic veins, IVC (or) the Right atrium

Hepatic veno-occlusive disease refers to the obstruction of Hepatic venous outflow at the level of central (or) Sub-lobular hepatic veins (or) both

Hepatic vein (or) IVC lesions include

- Membranous (or)
- Stenotic obstruction (or)
- Thrombosis.

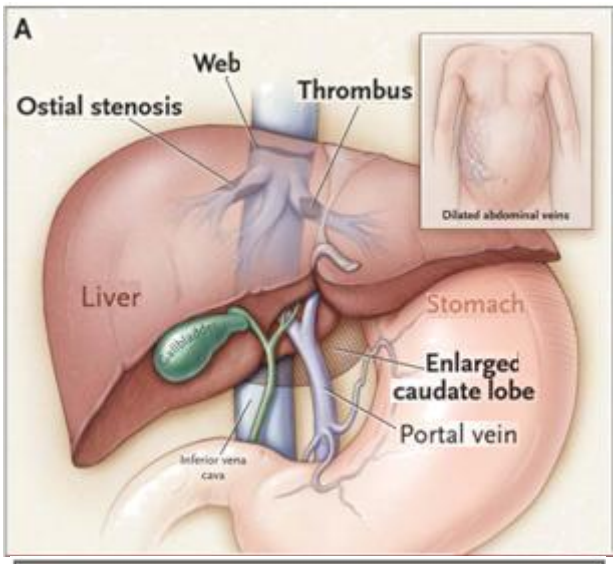


Table 1. Causes of the Budd–Chiari Syndrome.

Common causes
Hypercoagulable states
Inherited
Antithrombin III deficiency
Protein C deficiency
Protein S deficiency
Factor V Leiden mutation
Prothrombin mutation
Acquired
Myeloproliferative disorders
Paroxysmal nocturnal hemoglobinuria
Antiphospholipid syndrome
Cancer
Pregnancy
Use of oral contraceptives
Uncommon causes
Tumoral invasion
Hepatocellular carcinoma
Renal-cell carcinoma
Adrenal carcinoma
Miscellaneous
Aspergillosis
Behçet's syndrome
Inferior vena caval webs
Trauma
Inflammatory bowel disease
Dacarbazine therapy
Idiopathic

- **Thrombosis of the hepatic veins**is the most common cause of BCS in Western countries.
- Whereas **Membranous obstruction of the IVC (or) the Hepatic veins** is more common in Eastern countries.
- Patients with hepatic vein occlusion may present with symptoms and **Signs of portal hypertension** including **Ascites**, **hepatomegaly**, **splenomegaly** and **progressive liver dysfunction**.
- Two underlying causes lead to a manifestation of BCS.
- One is Hepatic vein thrombosis, which is often associated with various coagulation defects and disorders.
- The other is Membranous obstruction of hepatic veins (or) the IVC which occurs more common in Eastern countries and may be congenital in origin.
- As most of the significant symptoms and signs in patients with BCS are Associated with portal hypertension, **treatment is usually intended to relieve portal hypertension**.
- **Portal diversion treatment**, such as **Side-to-Side portocaval**, **Mesocaval and Mesoatrial shunts** have been used, but these surgical methods are also associated with many seriouscomplications.
- Recanalization of occluded major hepatic veins (or) the IVC facilitates hepatic venous outflow drainage
- Thus the hepatic congestion is relieved & portal pressure decreases, which is beneficial for recovery of liver function.
- Endovascular treatment has become a reliable treatment option for patients with symptomatic BCS.
- Restoring hepatic venous outflow is the primary objective when treating BCS.
- **Endovascular treatment is the main therapeutic choice**with advances in interventionaltechnology.
- A trans-jugular intrahepatic portosystemic shunt (TIPS) is currently the most common intervention for patients with BCS in Western countries because obstruction of the hepatic veins is usually extensive.
- Balloon dilatation (or) Stent insertion can be attempted to recanalize the Hepatic venous outflow pathways, when the lumen of a hepatic vein (or) the IVC is partially maintained.
- Hepatic venous pressure can Drop 25-50% after successful endovascular stenting in patients with BCS
- The 5-year survival rate in patients with mild disease and preserved liver function can be as high as 100%.
- Survival rates for patients with progressing BCS are about 85% at 1 year and 77% at 5 years.
- The **common complications of endovascular treatment for BCS** include inadvertent IVC perforation
- The **reported overall incidence of complications is <3%**, and mortality rate is about 1%.
- The **incidences of re-stenosis after balloon expansion and stenting of hepatic veinsare 10-20%** and **10%** respectively.

References

[1] Ludwig J, Hashimoto E, McGill DB, van Heerden JA. Classification of hepatic venous outflow obstruction: ambiguous terminology of the Budd-Chiari syndrome. Mayo Clin Proc1990;65:51-55
[2] Denninger MH, Chait Y, Casadevall N, et al. Cause of portal or hepatic venous thrombosis in adults: the role of multiple concurrent factors. Hepatology 2000;31:587-591
[3] Mitchell MC, Boitnott JK, Kaufman S, Cameron JL, Maddrey WC. Budd-Chiari syndrome: etiology, diagnosis and management. Medicine (Baltimore) 1982;61:199-218

[4] Frank JW, Kamath PS, Stanson AW. Budd-Chiari syndrome: early intervention with angioplasty and thrombolytic therapy.
[5] Yang XL, Cheng TO, Chen CR. Successful treatment by percutaneous balloon angioplasty of Budd-Chiari syndrome caused by membranous obstruction of inferior vena cava: 8-year follow-up study. J Am Coll Cardiol1996;28:1720-1724