A case of acute budd chiarri syndrome due to heparin induced thrombosis.

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MD

- Dalsukhbhai patel aged 56 yrs moderately built came at 10.30 pm for
- C/o chest discomfort precordial, pain not radiating
- No c/o breathlessness
- No c/o palpitation
- No c/o nausea, vomiting
- No other complains

- Patient is known case of IHD + Dm
- CAG done before 6 months--- 30 %LAD, 40 % RCA
- He was on --- Nitrates
- Aspirin
- Gliclazid + metformin
- Beta blockers
- Statins
- Vitamins
- Was on regular follow up and well preserved with normal lipid profile and DM

- He was Non smoker, Non alcoholic
- Took medicines regularly, also exercised regularly
- No other major complains in past
- No family history of major illness

- Vitals T --N
- P– 104/min
- Bp– 140/90 mm of hg
- RR– 14 / min
- RBS– 164 mg %
- ECG– ST ↓ V1---------V6
- RBS– 144 mg %
- RS, CVS, P/A--- NAD

- Treatment started 10. 40 pm
- Nitroglycerine inj 50mg/ 50 ml ns– 1 ml/hr
- Heparin 1000 units/hr
- Tab Ecosprin 150 mg 1 od
- Tab clopivas 75 mg 1 od
- Tab Betaloc 25 mg od
- Inj Rantac 1 amp I/v tds
- Tab Basiton forte 1 od
- RBS 6 hrly
- Inj actrapid acc to RBS
- O2 inhalation

- Patient seen at 12.30 midnight
- Comfortable, no complains
- P—100/min
- BP– 130/80 mm of hg
- S/E RS, CVS, P/A ---- Normal
- Monitor– ST segment sagging improved
- SpO2 -------- 100 %

NEXT DAY 7 AM

- Pt c/o severe epigastric pain– radiating to back
- perspiration + Nausea, Vomiting 1 time
- RS, CVS--- NAD
- Monitor--- HR---116/min
- RR--- 24/min
- SpO2—100 %
- Bp—100/70 mm of hg
- ECG– sinus tachycardia, no fresh ST—T changes
- RBS– 104 mg %
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7.10 AM

- Inj contramal I/V given
- Inj Pantraprazole I/V given
- NTG stopped

- 7.20 AM No relief
- Pain aggravated
- Inj rantac 1 amp I/v given
- Inj fortwin I/v given
- BP 80/60 mm of hg
- Normal saline 300 ml I/v bolus given.
- Dopamine 2 amp/50 ml ns 4 ml / hr started

7.30 AM

- C/o Abdominal discomfort increased
- Abdominal distension + nt
- P-- 146/min r
- BP– 80/60 mm of hg
- U/O 10 ml --- last hr
- RR--- 28 / min
- SpO2 92% with 10 litres of oxygen

7.40 AM

Urgent usg abdomen---- NAD except gross hepatomegaly.

- X ray Abdomen– propped up– No gas under dome of diaphragm.
- CBC– 10.0 gm %
- TC– 15,100 / cmm
- P 88- l-8, E 4, M 0, B 0
- Urea 40, Creat—1.4, RBS 136 mg %, PT– ct 20,pt38
- Bilirubin 3—2.1—0.9, SGPT 88 ,S alk po4 156, Amylase 158, Lipase 288

8.15 AM

- Reports reveived
- Positive findings-- Abdominal distension
- Hypotension
- Tachypnoea
- Tachycardia
- High PT
- Borderline high amylase, lipase
- Patient not responding to I/V fluids, Vasopressors
- Monitor– Except Tachycardia no fresh cardiac insult

8.20 AM

- Case reviewed
- Called for urgent doppler study of portal circulation
- 8.30 AM Doppler study of portal circulation done
- No flow observed in hepatic veins
- Slow flow in portal veins
- 8.40 AM Diagnosis of acute Budd chiarri syndrome made
- 8.45 AM– ABG done revealed severe metabolic acidosis--------- Patient dialysed.

- Patient dialysed upto 1 noon
- Bit stabilised
- P– 156/ min
- BP 96/70 mm of hg
- SpO2 90 %
- U/O nil
- HIV & HBsAG --negative
- Patient taken up for surgery at 2 noon

- Laprotomy done
- Findings– 3 litres of hemmorrhagic fluid
- Tense engorged hepatomegaly upto Iliac fossa.
- Hepatic vein cannulated and anti thrombin agent aggramed given.
- Abdomen closed
- Post op-- Patient deteriorated
- Severe hypotension
- Hypoxia
- Lactic acidosis

- Patient supported with
- Ventilators
- Vasopressors
- Blood products
- Anti biotics
- Supportive
- Repeat dialysis started

- Ultimately patient goes on deteriorating dies at 2 am in night due to cardiac arrest

DISCUSSION

- Budd –chiari—uncommon condition due to thombotic or non thrombotic occlusion of hepatic venous outflow.
- Obstruction of hepatic veins—congestive hepatomegaly as blood flows in but not out of liver-leads to hepatocellular injury—portal HT and liver failure.
- Causes-Thrombotic diasthesis, myeloproliferative disorders—polycythemia vera, paroxysmal nocturnal hemoglobinuria, pregnancy, tumours, clotting disorders, infections, chronic inflammatory disease.

- Classic triad– abdominal pain, ascitis, hepatomegaly.
- Clinical variants– acute liver failure, subacute liver disease, fulminant liver disease , chronic disease.
- Acute & sub acute form– abdominal pain ascitis, hepatomegaly, jaundice, renal failure.
- Chronic form– progressive ascitis, jaundice absent ,50 % have renal impairment.
- Fulminant form—ascitis, tender hepatomegaly,jaundice and renal failure.

- Physical findings—
- Icterus
- Ascitis
- Hepatomegaly
- Splenomegaly
- Ankle edema
- Stasis ulceration
- Prominence of collatral veins.

- Causes---
- Polycythemia rubra vera
- Paroxysmal nocturnal hemoglobinuria
- Unspecified myeloproliferative disorder
- Anti phospholipid antibody syndrome
- Essential thrombocytosis
- INHERITED THOMBOTIC DIASTHESIS
- Protein c deficiency
- Protein s deficiency
- Anti thrombin 3 deficiency
- Factor 5 leiden deficeincy

- Pregnancy and post partum
- Membranous webs
- Oral contraceptives
- CHRONIC INFECTIONS
- Hydatid cysts
- Aspergillosis
- Amebic abscess
- Syphilis
- Tuberculosis

- Chronic inflammatory disease
- Behcet disease
- Inflammatory bowel disease
- Sarcoidosis
- SLE
- Sjogrens syndrome
- Mixed connective tissue disease.

- Tumors
- Hepatocellular carcinoma
- Renal cell carcinoma
- Leiomyosarcoma
- Wilms tumor
- Right atrial myxoma

- IDIOPATHIC

- Investigations—
- CBC
- URINE, SUGAR
- UREA, CREATININE
- LIVER FUNTION TEST
- AMYLASE, LIPASE.
- ASCITIC FLUID ANALYSIS
- ELECTROLYTES
- ABG
- TESTS OF HYPERCOAGULABLE STATES

- ULTRASOUND
- COLOR DOPPLER -- 85 TO 90 % ACCURATE
- MRI –95 %
- HEPATIC VENOGRAPHY
- LIVER BIOPSY

MANAGEMENT

- Anticoagulation
- Anti thrombolytic therapy
- Angio plasty
- Decompression of hepatic vasculature– trans jugular intrahepatic portosystemic shunt.

- LIVER TRANSPLANT

ABSTRACT

- Heparin induced—thrombosis & thrombocytopenia
- White clot syndrome– rare but recognised complication of heparin therapy.
- Syndrome—Idiosyncratic, immune mediated and not dose dependent.
- Occurs in therapeutic or prophylactic dose in susceptible people.
- Very alarming– many surgeons & physicians are unaware.

- Treatment—
- Immediately stop heparin.
- Start oral anticoagulants.
- Can use lmwh
- If required amputation.
- Thrombolysis & angioplasty if early diagnosed.

THANK YOU.

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