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President, Indian Association of Surgical Oncology 1993

Medical Scientist of the Year LIMCA Book 1996

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PORTAL HYPERTENSION EVIDENCE BASED MANAGEMENT

- Portal blood flow 1000 – 1200 ml/mg
- Portal V. supply 72% of O_2 to liver, Arterio / portal venous O_2 – 1 to 1.19 increases during digestion
- Portal pressure – 7mm of Hg
- Collateral Circulation

CIRRHOSIS

- Intra hepatic obstruction –
 - 13% of blood flow to liver while rest 87% to collateral's to systemic

Loss of blood to liver results

- Liver shrinks – impaired capacity to regenerate
- Lack of hepatotrophic factor – Insulin glucagon etc.
- A large porto systemic shunt lead to hepato encephalopathy, septicemia due to intestinal organisms and other circulatory or metabolic effects.

PORTAL HYPERTENSION

Pathology

- Spleen – enlarged thick capsule malphigian bodies are inconspicuous. Sinusoids dilated lined by thickened epithelium, histocytes proliferation erythrophagocytosis, peri arterial hemorrhage siderotic fibrotic nodules.
- - Splenic and portal vein – dilated and tortuous, -endothelial hemorrhage, intra mural thrombosis, intimal plaques with calcification and unsuitable for surgery (Porta-caval).

- In 50% cirrhosis – deeply placed splenic arterial aneurysm seen.
- Portal pressure does not correlate with degree of cirrhosis but - proportionate to nodularity
- The connection between portal and systemic circulation at gastro oesophageal junction is complex.
- Failure of sclerotherapy may be due to failure to thrombose perforating veins.

- Duodenal variceal – filling defect.
- Bile duct collaterals – life threatening
- Colonic rectal Verices becomes prominent after sclerotherapy.

- Portal hypertension – leading to
 - Gastropathy
 - Gastric antral vascular ectasia
 - All these symptoms increases with sclerotherapy, but relived after shunt surgery
 - Congestive jejunopathy and colonopathy

- In Portal hypertension Portal venous pressure is lowered by collaterals – diverting to systemic veins but Portal hypertension is maintained by increase in portal venous flow
- Portal Hypertension
- Collaterals – Congested engorged of both – Portal / Systemic Veins.

- Portal Hypertension does not diminish



- Increase Portal venous flow and maintain portal hypertension



- Increase cardiac output



- Generalised venous dilation



- Normal arterial Blood flow

Splanchnic Vasodilatation is probably the most important factor in maintaining hyper dynamic circulation.

- Endotoxins and cytokines formed in gut are important triggering factors –
- Nitrogen O_2 endothelin-1, prostacyclin – produced by venous endothelium – results in circulatory changes.
- Glycogen. Vasodilatation
- Hepatic venous pressure gradient is difference of wedge hepatic venous pressure & free hepatic venous pressure
 - Normal – 5-6 mm of Hg
 - More than 20 mm in cirrhosis
 - Variceal pressure – 15-20 mm of Hg.

PORTAL HYPERTENSION - SYMPTOMS

- Variceal bleeding
 - 37% - (PAGLIO – et.al. 1994)
 - 61% - (CALESE – et.al. 1990)
- Depends upon – Child's classification

- N. Eng. J.Med,. 319,983- 1998
- Prospective study 34% - A
52% - B
76% - C

SHUNTS

- ECK (1897) First to describe END to SIDE PORTACAVAL SHUNT
- VIDAL – (1903) – PERFORMED P.C. Shunt First
- Whipple (1945) – Introduced porta systemic shunts
- WARREN – described rationals of variceal decompression and maintenance of portal perfusion with Distal spleno renal shunt.

- SHUNTS - Results depends upon its childs
- Classification
- Mortality 15 – 25%
- Survical 50 – 60% - 5 years

- Medical Management + Shunt – Better result by 5 to 10%

- CHILD S C - Poor result – irrespective of procedures.

- CELLO et.al. 1990
- RIKERS et.al. 1994
- SLOSBERG 1997

Treatment Advised

- SHUNTS – CHILDS – A, early B
- SCLEROTHERAPY TIPS CHILDS -C

- Hepato pulmonary syndrome
 - Hepatic - Hypoxemia
 - Intra pulmonary vascular dilatation

It can occur in any stage of classification Childs A-C

- Treatment for Hepatic Pulmonary Syndrome
 - Live Transplantation

- Portal hypertension is the result of increased Intra hepatic resistance and increased splanchnic blood flow
- Each of these depends upon various factors in Liver and splanchnic vessels.

Treatment in Portal Hypertension is the treatment of its complications

- Variceal bleeding is the most important complication which is life threatening, thus given more attention.
- Non operative management of acute variceal bleeding can control bleeding and give time for reassessment for definitive therapy to prevent rebleeding.

- Balloon Tamponade (SANGSTEKEN)
- Sclero therapy – High rate of success
- Pharmacotherapy

- Emergency Surgical treatment only in cases of failure of Non surgical procedures and fit to tolerate the operation.

PROCEDURES

- Potacaval or Mesocaval recommended for moderate to severe bleeding in alcoholic patients
- Distal spleno renal shunt – in non alcoholic moderates bleeding
- Devascularization – limited role in emergency but good for vascular block cases
- Sugiura Procedure appropriate for non alcoholic cases.
- Oesophageal transection for alcoholic cirrhosis
- Liver transplantation end stage – Liver disorders.

- Beta Blockers – modify splanchnic Hemodynamic with variable response
- Endoscopic sclerotherapy – choice –as primary treat- Also needed in repeated bleeding cases with high surgical risk or other complications.

- LEVINE & SIRNEK - 1900
- CELLO et.al. - 1987
- RIKKERS et.al. - 1987
- WARREN et.al. - 1986
- In acutely bleeding patients (Childs – C)
- No difference in evident in operative mortality??
Long term survival or costs in sclerotherapy is important.
- Sclerotherapy has high rebleed rate, but – high mortality in shunt.

SCLEROTHERAPY V/s SPLENORENAL SHUNT

- Warren found superior survival with sclerotherapy (84% V/s. 59%) while Rikkers found no change (All classes of Child)

Inter Position Mesocaval Shunt

- DRAPAMAS (1975) Popularised
 - Encephalopathy - 15%
 - Operative Mortality -A 6%
 - -B 13%
 - -C 58%
 - Emergency Procedures Mortality - 42%
 - MEHIGAN (1980) – Overall Mortality - 18%

Long Term Survival

- **Most Series** – DOWLING THOMASON
STIPA, CAMERON, SMITH, MULCARE,
REZNICK, TERPSTRA, VOGT.
 - 1 YR. - 60% to 84%
 - 5 YR. - 30% to 50%
 - CAUSE OF DEATH IN 60% CASES LIVER
FAILURE

- Intractable Portal Hypertension and Bleeding Oesophageal Varices Refractory To Conventional Treatment Needs – Liver Transplantation

- Distal Spleno Renal Shunt
- Better Result In Non Alcoholic Then Alcoholic

Follow up of Distal splenorenal shunt (Millikan, langer, fischer, reichle, grace, Harley)

- Operative Mortality 7 to 5%
- Rebleeding 2 to 7%
- Encephalopathy 7 to 20%

DSRS V/s SCLEROTHERAPY

- No significant difference in short term survival
- High rebleeding rate in sclera and death
- High death rate in DSRS DUE TO Liver Failure

- DSRS – Utmost Advantage
- Maintains \Hepatic Function and Portal Perfusion

Non Shunt Procedures – Sugiura Procedure

- Result not been duplicated by others
- Low mortality in selective patients (Non Alcoholic – A-B)
- Long Term Survival Better Than Shunt Operations
- In Alcoholic A and B – Results As Good as DSRS
- Good Results in rebleeding after repeated sclerotherapies
- High Mortality in Child C (25-50%) (Alcoholic - More)
- Is Applicable in emergency
- Only – Oesophageal Transaction is helpful in emergency

Liver Transplantation In Chronic Liver Disorders and Variceal Hemorrhage

- THEORETICALLY – 1992
- Is the Ideal treatment as It improves liver resistance and portal hemodynamics

LIMITATIONS

- DONOR
- MORTALITY AND MORBIDITY
- COST OF PROCEDURE
- IMMUNOSUPPRESSIVE DRUGS
- LONG TERM RESULT ...??

Liver Transplant

- Is it established with Clinical datas?
- No
- Indication In variceal bleeding?
- In Delayed Cases – Option ?
- Transplant Patient In waiting – how to manage Emergencies?
- Transplant in existing Shunts?

Portal Hypertension not needed Transplantation

- Extra Hepatic Presinusoidal
- Port Vain Thrombosis
- Cavernomatous Transformation
- Splenic vain Thrombosis
- Intrahepatic – Presinusoidal
- Schistosomiasis
- Sarcoidosis
- Congential Hepatic Fibrosis
- Arsenic Toxicity
- Myeloproliferative – Diseases
- Increased Portal Flow
- Hepatic Artery – Portal V Fistula
- Increased Splenic Flow

V. BLEEDING – SCLEROTHERAPY/ SHUNT / TRANSPLANT

Survival

	1 Yr.	5 Yr.	
Sclerotherapy	50-90%	40-80% (10-20%)	A-B
Porta Caval Shunt	70-90%	40-60% (13-35%)	A-B
Transplant	79-90%	70-80%	C

Transplant Result - Survival Is Very High
In Child's - C

SURVIVAL IN CHILD – A & B

- HANDERSON – 1989, MILLIKAN-1985, 1989, WARREN-1982

	1 Yr.	5 Yr.
Sclerotherapy	95%	80%
Shunts (Selective/Nonsele	70-90%	50-65%
Transplant	85-92%	80-90%

SURVIVAL AFTER ONE YEAR IN MAINTAINED
DEATH IN MAJORITY BEFORE 1 YEAR

QUALITY OF SURVIVAL

	Rebleeding	Encephalopathy
Sclerotherapy	30-60%	15-30%
Shunts	>10%	40%
Transplant	Goodlife 75-85% After 1 year	

CONTRADICTION TO TRANSPLANT

- **ABSOLUTE**

- Extrahepatic Ca
- Extra Biliary Sepsis
- Porta Systemic Thrombosis
- Organ Failures
- Drug Abuse – Alcohol
- Aids
- Non Compliance

CONTRADICTIONS

- **RELATIVE**

- Extremes of age
- Aids without Symptoms
- Multiple Prior Transplant
- Mentally ill or Psychotic with possible non compliance

Prophylactic Therapy for Variceal Bleeding

- DAGRADI – 1972, CONM.-1988, FLEIG-1987, JACKSON-1988, RESNICK-1969, BAKER-1959, CHRISTENSEN-1981
- MORTALITY ON FIRST Variceal Bleeding 50% or More

Risk Factors for First Bleeding

- 2/3 Pts will Never Bleed
- But exposed to harmful complications of undesired treatment
 - Identify patients carrying high risk of bleeding in coming – One to two years

Is Bleeding Related to

- Increase in portal pressure – NO
- Buetzone -1977 – Doffoel-1980, Lebreer-1980, Vinel-1982
- Intra Variceal Pressure – Is linked to Bleeding then portal pressure
- KELEBER-1988, RIGAU-1985

- Variceal Radius + Wall Thickness + Variceal Wall Tension – Is LAPLACES EQUATION demonstrates Increase Risk

Cumulative Evidences From Various Trials Suggest -

- Prophylactic Sclerotherapy or Shunt Procedures – Neither Helpful nor very Harmful. Hence No Role In prophylaxis
- Liver Transplant In Cirrhosis- (Alcoholic) – is the choice of All factors in control
- Drugs Have Greater Role to Play

THANKS