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Pulse

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- Conclusion.
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Case reports and case studies of practical interest to clinicians are also invited for publication.

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BARC HOSPITAL NEWSLETTER

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While at school at vidya bhavan, I still remember my biology classes on human body. The importance of healthy liver and kidneys to the well functioning human body was a salient feature of those classes. It was later in the medical school that the importance of these organs was understood.

Both the organs have large endothelial surface area for interaction with circulating drugs and toxins. They have a very high blood flow and may get equally affected in response to an ischemic insult. Furthermore, both liver and kidneys depend on high energy phosphates, for their high metabolic needs, making them susceptible to toxins that disrupt energy metabolism. Hence, drugs affecting the mitochondrial energy generating reactions, may cause combined renal and hepatic damage. This is especially true for centrilobular region of the liver and the medullary thick ascending loop of Henle in the kidney. Another mode of combined hepatic and renal injury due to drugs, is a generalised hypersensitivity reaction. This is observed after repeated exposures to the same drug.

Both organs are involved in excreting the toxic products of metabolism and exogenous drugs. Liver and kidneys also collaborate in regulation of extra cellular volume. Superimposition of hepatic failure on renal failure or vice versa, deprives the human body of two major detoxification mechanisms. Combined hepatic and renal failure affects the haemostasis and host defence mechanism, leading to risk of major haemorrhage / sepsis. Also in combined hepatic and renal failure, the systemic diseases can manifest as multiorgan failure, infections, toxic insult to liver and kidneys, hepatorenal syndromes and acute tubular necrosis.

Keeping the importance of these organs in view, in this issue of 'Pulse', we have tried to cover mainly certain aspects of pathology related to the liver. In the forthcoming issue, we plan to deal with certain aspects of renal pathology.

(Dr. Amrita Misri)

"The liver, that great maroon snail: no wave of emotion sweeps it. Neither music nor mathematics gives it pause in its appointed tasks."

-Dr. Richard Selzer
Surgeon, Author, New York.

Viral Hepatitis A to Z: An Overview

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Introduction

Human liver is a key organ, which plays a central role in the synthesis, breakdown and storage of multiple substances, and has an incredible, although limited, capacity to regenerate itself. Jaundice is a hallmark of liver disease, and has been first described by Hippocrates in 400 B.C. Hepatitis is a general term describing inflammatory (infectious or non-infectious) process causing liver disease. Differential diagnosis of acute hepatitis is very broad. Infectious causes include viruses (Hepatitis viruses A-E, Epstein-Barr Virus, Cytomegalovirus, Herpes Simplex Virus, Mumps, Rubella, Rubeola, Varicella-Zoster Virus; Yellow Fever Virus; Coxsackie B Virus, Adenovirus, Human Immunodeficiency Virus), bacteria (Leptospira, Brucella, Bartonella, other spirochetes), fungi (histoplasma, candida), and rickettsial organisms. Non-infectious causes include drug induced, autoimmune or ischemic hepatitis, Wilson's disease, Budd-Chiari syndrome, and in pregnant patients acute fatty liver of pregnancy and HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets).

The term viral hepatitis generally refers to infections caused by hepatotropic, in other words "liver loving" viruses. Five main pathogenic entities have been identified so far: hepatitis A, B, C, D, and E. The role of several recently identified viruses: hepatitis G virus (or GB virus C), Transfusion Transmitted Virus (TTV) and SEN virus, is not clear at this time.

Viral hepatitis constitutes a significant healthcare burden in many countries in the world. It has been estimated that approximately 1.4 million cases of viral Hepatitis A occur worldwide annually, 2 billion people have been infected with hepatitis B with approximately 350 million developing chronic infection, and about 300 million people have been infected with Hepatitis C with about 80% developing chronic infection.

It is virtually impossible to distinguish on clinical grounds alone the etiologic agent of viral hepatitis therefore serologic diagnosis is an essential part of the diagnostic workup. Clinical disease can range from asymptomatic infection, marked only by elevated liver function tests, to symptomatic illness with weakness, fatigue, low grade fever, nausea, right upper quadrant abdominal pain, and fulminant hepatitis with signs of liver failure such as confusion, stupor, coma, ascites and peripheral edema. Signs include hepatic tenderness, hepatomegaly, splenomegaly, jaundice, lymphadenopathy, skin rash and bradycardia. Extrahepatic manifestations, secondary to immune complex mediated tissue damage, may manifest as serum sickness - like reaction, glomerulonephritis, polyarteritis nodosa, essential mixed cryoglobulinemia and porphyria cutanea tarda.

There are four classical stages of acute viral hepatitis: the incubation period, the preicteric phase, the icteric phase and the convalescent phase. Chronic hepatitis denotes a prolonged course of the disease lasting more than 6 months.

It is characterized by persistent inflammation leading eventually to liver fibrosis, cirrhosis, and in some cases liver cancer. Liver destruction is not caused directly by viral replication but rather through virus specific CD8+ cytotoxic T lymphocytes and CD4+ T helper cells, which are part of the adaptive immune response to viral infections. The early innate immune mechanisms such as apoptosis, interferon alpha and beta and natural killer cells are neither cytopathic to the liver nor very effective in viral clearance.

We present here a short overview of hepatotropic viruses, potential treatment, prevention, and prophylaxis. It is by no means a comprehensive review and the reader is referred to other excellent sources for a more in depth study.

Hepatitis A virus was identified in 1973. It is a RNA virus belonging to the Picornaviridae family. There are 6 different genotypes with genotypes 1 through 3 responsible for human disease, and genotype 1 being most prevalent worldwide. Hepatitis A is transmitted mainly by fecal oral route and blood borne transmission is very rare. No transmission through saliva has been documented. Poor personal hygiene habits, lack of hand-washing and poor sanitation contribute to local epidemics. It is easily spread through close contacts. Uninfected person acquires the infection by ingesting food or beverage contaminated with the stool of an infected person (who can shed the virus from 2 weeks before to one week after the onset of clinical symptoms). Incubation period is approximately 28 days (range 15-50 days). Clinical symptoms and signs include fatigue, malaise, nausea, vomiting, abdominal pain, dark urine, light colored stools, and jaundice. Clinical disease is usually self limited, and persists for approximately three weeks; treatment is supportive with rest and hydration. Fulminant illness can develop especially in those with chronic liver disease. Mortality is approximately 0.5% for people less

than 50 years old, and it increases to about 2% for those over fifty. Prevention is paramount. There are two very effective vaccines available (Havrix and Vaqta) for those one year and older. Even one dose of the vaccine administered prior to exposure is 94-100% effective in preventing clinical hepatitis A. Protective antibody levels last for about 20 years. Post-exposure prophylaxis for those not previously vaccinated is available. Individuals older than 12 months should receive hepatitis A vaccine or hepatitis A immunoglobulin immediately after exposure, vaccine being preferred in those younger than 40. Children less than 12 months old, immunocompromised patients, those with chronic liver disease or contraindications to the vaccine should receive hepatitis A immunoglobulin, which provides protective antibodies lasting 3-6 months.

Hepatitis B virus is a DNA virus belonging to Hepadnaviridae family. It was identified in 1973 preceded by the landmark discovery of Australia antigen by Blumberg in 1967. It is highly infectious, about 50-100 times more than HIV, and it can survive outside of body for at least seven days. It is mostly transmitted through blood, or blood containing fluids. It has been also found in semen, vaginal secretions, and saliva. There are reports of transmission through pre-mastication. Percutaneous transmission rate is very high and approaches 30% compared to 3% for hepatitis C and 0.3% for HIV. Maternal fetal transmission does occur. Incubation period ranges between 6 weeks to 6 months. Fulminant disease develops in about 1% of infected individuals, especially in those concomitantly infected with hepatitis D virus. Clinical symptoms are similar to other forms of viral hepatitis and serologic diagnosis is necessary. This virus causes both acute and chronic hepatitis. Infants and children are at significantly higher risk of progression to chronic disease when compared to adults. About 90% infants, 30% children less

than five years of age and 10% adults will progress to chronic liver disease. This is marked by chronic and progressive necroinflammatory state of the liver, which leads to cirrhosis and hepatocellular carcinoma in 15-25% of individuals who do not clear the infection. As with hepatitis A, the treatment for acute infection is supportive only. There are several drugs available for treatment of chronic hepatitis B infection. This may involve pegylated interferon or antiviral drugs such as tenofovir, entecavir, lamivudine, adefovir, telbivudine, as single agents or in combination. Currently first line agents are tenofovir or entecavir based on the most recently published guidelines. Interferon is also an option for patients who do not have cirrhosis. As with hepatitis A, prevention is extremely important especially in infants and children at high risk for acquiring hepatitis B as well as all healthcare workers. There are two vaccines available: Recombivax HB and Engerix B. Vaccines are quite effective with more than 90% of those immunized developing protective antibodies which last for at least 15-20 years. It is worth mentioning that combined Hepatitis A and B vaccine (Twinrix) is available for use in adults. Hepatitis B vaccine and Hepatitis B Immunoglobulin are recommended for post exposure prophylaxis.

Hepatitis D virus was identified in 1977. It is a defective RNA virus, which belongs to a family Deltaviridae. It does not have the replicative machinery to multiply on its own, however when coinfection or superinfection with hepatitis B occurs it may lead to fulminant liver failure and progress to chronic liver disease and cirrhosis more rapidly. There are 3 different genotypes. Genotype 1 is found mainly in Europe and United States, genotype 2 in East Asia, and genotype 3 in South America (which can cause severe liver disease known as Amazon Black Fever). Treatment is supportive for acute infection. Liver transplantation can be considered for fulminant

acute hepatitis D. Interferon alpha has been shown to have some success in treatment of chronic hepatitis D.

Hepatitis C virus was identified in 1989. It is a RNA virus that belongs to the family Flaviviridae. The average incubation period is 50 days (range 14-120 days). Transmission occurs through blood or contaminated bodily fluids. Those at highest risk are: recipients of blood products particularly before 1990 (prior to testing of blood products for hepatitis C), intravenous drug or intranasal cocaine users, individuals with multiple sexual partners or extensive body piercing and tattooing, men who have sex with men (MSM), dialysis patients, and health care workers. Most people are asymptomatic during the acute phase of infection and recover, however about 80% develop chronic infection which leads to liver cirrhosis and hepatocellular carcinoma. Serologic diagnosis involves screening by Hepatitis C Antibody (ELISA) and confirmation of positive tests by Recombinant ImmunoBlot Assay (RIBA) and/or HCV RNA viral load by PCR. Hepatitis C virus has 6 major genotypes. It is important to know the genotype to determine the duration of treatment and predict response to therapy. Genotypes 1 and 4 respond poorly to treatment with an overall success rate of 50% and require a prolonged course of treatment (48 weeks). Treatment duration for genotypes 2 and 3 is 24 weeks with a response rate about 80%. Treatment involves weekly subcutaneous injections of pegylated interferon and ribavirin and must be individualized according to viral genotype. Major side effects of treatment include depression, hypo- or hyperthyroidism, flu-like symptoms and pancytopenia and they are common enough to require close patient follow up and monitoring. Liver biopsy is recommended prior to treatment especially for genotypes 1 and 4 to determine the extent of liver fibrosis and the disease activity. Therapy is contraindicated for those with active alcohol use, poorly controlled

depression, solid organ transplant, autoimmune hepatitis, untreated thyroid disease, pregnancy, severe concurrent medical disease, age less than 2 years, and hypersensitivity to drugs used for treatment. Chronic hepatitis C is the leading indication for liver transplant in the United States. Currently there is no vaccination available for hepatitis C. There is no effective pre- or post-exposure prophylaxis. New treatment modalities are in the development phases, awaiting approval. Most likely they will not replace the current therapy but serve as additional agents to the existing combination therapy, hopefully improving therapeutic success but at the same time increasing the side effects of an already difficult treatment.

Hepatitis E virus was identified in 1983 during the waterborne epidemic of viral hepatitis in Delhi, India. It is a RNA virus endemic to Southeast and Central Asia. It belongs to family Hepeviridae. There are 4 different genotypes. Genotypes 1 and 2 mainly affect humans and are responsible for most local outbreaks and genotypes 3 and 4 cause infections in animals and sporadic cases in humans. Average incubation period is about 40 days (range 20-60 days). Hepatitis E like hepatitis A spreads by the fecal oral route. Poor personal hygiene and poor sanitation facilitate the spread of the virus. Local epidemics have been linked mainly to contaminated water sources with highest rates in Southeast Asia, Middle East, North Africa and Mexico. Sporadic cases are usually of animal origin and have been linked to eating shellfish, contaminated animal meat or direct contact with an infected animal.

Acute disease caused by Hepatitis E is clinically indistinguishable from other viral causes of acute hepatitis. Chronic disease is very rare and has been reported mainly in immune-compromised patients. Fulminant disease is rare but has mortality rate up to 4%. Pregnant females especially in the third trimester are at highest risk

of developing fulminant hepatitis. Mortality rate in this group approaches 25%. A large retrospective study conducted in a single center in India showed however that mortality rates in pregnancy were about the same in cases of acute liver failure irrespective of etiology. Vertical transmission from mother to fetus does occur and may result in fetal demise. Hepatitis E, like hepatitis A, can cause superinfection in patients with chronic liver disease, and can lead to acute liver decompensation and increased mortality.

There is no specific treatment for hepatitis E. A case report has been published recently describing successful treatment of chronic hepatitis E with ribavirin. Further studies are needed to establish the role of ribavirin in the treatment of not only chronic hepatitis but also acute hepatitis E in high risk patients. Hepatitis E vaccine has recently been developed in China and has been shown to be effective in preventing clinical disease.

Non- A to E hepatitis viruses (non-ABCDE) is a general term describing several viral entities which were discovered in the search for the cause of post-transfusion hepatitis.

Hepatitis G/GBV-C virus belongs to family Flaviviridae and it was discovered in 1995/1996 from plasma of a surgeon with acute hepatitis. Initially it was identified as two distinct viral entities however further analysis confirmed it is the same virus. There are 3 different genotypes isolated (GBV-A, GBV-B, GBV-C). Their exact role in the pathogenesis of liver disease is unknown. It has a high prevalence among general population, but is most commonly present in people infected with HIV, Hep C and those with history of multiple transfusions, organ transplant recipients and hemodialysis patients. There are multiple reports that GBV-C co-infection with HIV can reduce progression of HIV disease.

The identity of a putative agent named Hepatitis F Virus was not confirmed by further research.

Transfusion Transmissible Virus (TTV), identified in 1997 and SEN Virus, discovered in 1999 are DNA viruses belonging to the Circovirus

family. Prevalence of these viruses among healthy individuals is high in some countries, especially Japan and at this point neither of them is believed to cause liver disease or exacerbate existing chronic disease in case of a superinfection.

Serologic Diagnosis of Viral Hepatitis during acute, remote/immune and chronic phases

Hepatitis	Acute	Remote/Immune	Chronic
A	Hep A IgM antibody	Hep A IgG antibody	None
B	Hep B surface antigen Hep B e antigen Hep B core IgM antibody Hep B DNA viral load (PCR)	Hep B surface antibody Hep B core IgG antibody	Hep B surface antigen * Hep B e antigen Hep B core total antibody Hep B DNA viral load (PCR)
C	** Hep C antibody Hep C RNA viral load (PCR) Hep C genotype RIBA	Hep C antibody	Hep C antibody Hep C RNA viral load PCR
D	Hep D virus IgM Hep B surface antigen Hep D viral RNA PCR		Hep D virus total antibody Hep D viral RNA PCR Hep B surface antigen
E	Hep E IgM antibody Hep E viral RNA PCR (blood or stool)	Hep E IgG antibody	None
G/GBV-C	Hep G viral RNA PCR	Hep G IgG antibody	None

* - May or may not be present.

** - May not be present in early acute disease.

Mode of transmission

Fecal Oral	Bloodborne/Percutaneous	Sexual
Hepatitis A and E	Hepatitis B, C, D, G, TTV, SEN-V	Hepatitis A, B, C, D, G

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Approach to Hepatitis B

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There are 500 million chronic carriers of Hepatitis B virus (HBV) in the world and HBV is the second most important carcinogen after tobacco, causing 60%–80% of all primary liver cancers. HBV belongs to the Hepadna group of viruses and is a DNA virus which is 100 times more contagious than HIV and is more difficult to eradicate than HIV. India is in the intermediate zone of prevalence for HBV, ranging from about 2 to 5%.

Hepatitis B virus is transmitted parenterally, sexually, vertically from mother to child mainly at the time of delivery or in India more commonly horizontally -typically from one family member to another through saliva or another route. The chances of a household contact showing evidence of HBsAg infection is higher among siblings than spouses, with the risk going up to 50%. Tooth brushes, razor blades, handling of sharp things, kissing and biting and mouthing of toys by children, have been associated with an increased risk of transmission.

Presence of HBsAg for more than 6 months suggests chronicity.

In a patient presenting with acute onset of jaundice, the presence of HBsAg could mean any of the following:

1. the patient is suffering from acute hepatitis B (anti HBc IgM should be positive)
2. the patient could be a chronic carrier of hepatitis B with re-activation of the virus (here too anti HBc IgM may be positive)
3. Does not differentiate acute from chronic hepatitis – patient could be a chronic

carrier of Hepatitis B with another superadded viral hepatitis like E or A or delta virus.

In a patient presenting with acute viral hepatitis, the more severe the attack of jaundice or the more fulminant the infection, the higher is the immunity and lower is the chance of chronicity. Those with a lower immunity are more likely to have anicteric attacks and become chronic. In fact, most patients with chronic Hepatitis B are picked up incidentally and often there is no high risk history available.

The common modes of incidental detection are:

- Blood donation screening
- Pre-operative screening
- Pregnancy screening
- Pre-employment screening
- Going to the Gulf for a job, now other countries also ask for HBsAg
- Family member testing positive.

In a patient with chronic hepatitis B

10-20% will develop cirrhosis

25% of these will develop decompensated liver disease

6-15% of those with chronic disease will develop hepatocellular carcinoma.

Serology testing in Hepatitis B

HBsAg > 6 months – chronic carrier/chronic hepatitis B

Anti HBc IgM positive - acute hepatitis B, also in re-activation of a chronic hepatitis B

Table 1: Hepatitis B phases of infection

	Immune Tolerant	Immune active	Inactive	Re-activation e negative
HBeAg	+	+	-	- Anti HBe +
HBV DNA (IU/ml)	High > 20,000	High > 20,000	Low < 2000	High > 2000
ALT	Normal	High	Normal	Normal or High
Inflammation & fibrosis	Absent	Present	Absent/ regressed	Present

Anti HBc IgG alone – past infection with Hepatitis B

HBsAg & anti HBc IgG - chronic hepatitis B.

HBeAg (needs to be done only in the setting of chronic hepatitis B) - replicative phase where the HBV DNA (viral load) is high and the patient is infective.

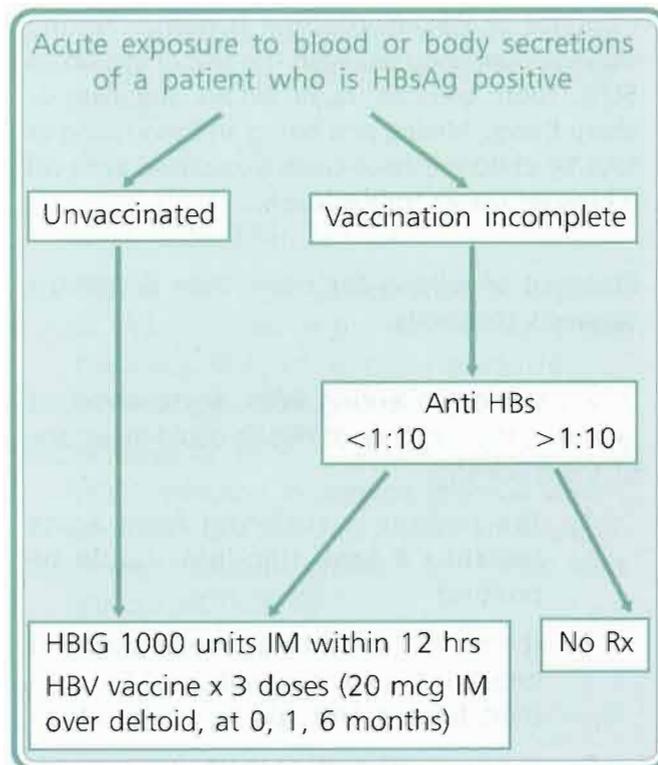
Anti HBe - non-replicative phase, however if HBV DNA > 2000 IU/ml, it implies that the patient is a pre-core mutant.

Anti HBs – protective antibody seen in patients with recovery from previous infection or after receiving the vaccination for Hepatitis B (vaccine contains HBsAg particles).

The following groups need to be screened for hepatitis B:

- Patients with abnormal ALT (SGOT)
- Household members of known HBV carriers
- High-risk sexual behaviors
- Injection drug users
- Immunocompromised patients
- Dialysis patients

- Organ/tissue transplant recipients
- Occupational exposure
- Inmates in remand homes
- Pregnant women
- Individuals infected with HCV or HIV



Incidentally tested HBsAg positive

1. Confirm whether chronic carrier (Continues to be positive for HBsAg + ve after 6 months)
2. Viral Replication status – HBeAg, HBV DNA viral load
3. Severity of liver inflammation and function
4. Hepatic imaging – USG abdomen for presence of cirrhosis and portal hypertension
5. Screening of family members.

Need for anti-viral treatment

High viral load and ALT, predict progression of liver disease to cirrhosis and the development of a hepatocellular carcinoma.

Updated AASLD (American Association for Study of Liver Disease) guidelines recommend lowering normal ALT cutoff to 30 IU/mL for men and 19 IU/mL for women.

Objectives of Therapy

Biochemical Response

- Improvement in AST/ALT and Liver Synthetic function (albumin, prothrombin time).

Histological response

- Decrease hepatic inflammation
- Decrease rate of progression to fibrosis
- Decrease incidence of long-term sequelae (cirrhosis, end-stage liver disease, hepatocellular carcinoma).

Virological response

- Reduction in HBV DNA.

Serological response

- HBeAg loss and HBeAg seroconversion to anti HBe in those who are HBeAg positive
- HBsAg loss and seroconversion to anti HBs.

Treatment

Treatment is recommended in the immune active and the re-activation phases of the liver disease and not in the immune tolerant or inactive phase of the disease (Table 1).

In general, treatment is recommended when ALT (SGOT) is more than twice ULN (upper limit of normal) and HBV replication HBV DNA > 2000 IU/ml in HBeAg -ve and > 20,000 IU/ml in HBeAg +ve.

If ALT is between 1-2 times the upper limit of normal, a liver biopsy may be done especially if the patient is more than 40 years of age and patient is treated if at least moderate necro-inflammation and significant fibrosis are seen and observed in those whose ALT is \leq 1 times ULN).

Any decompensated cirrhotic patient with ascites, encephalopathy, variceal bleeding or any other complication of cirrhosis should be treated.

In those patients who are being observed, ALT is repeated every 3-6 months and HBeAg every 6 months in those who are positive. HBV DNA is also quantified every 6 months, and if the patient is HBV DNA negative with normal ALT for more than a year (Inactive carrier), the HBV DNA may be done yearly.

Treatment in HBeAg positive patients

Duration of treatment with pegylated interferon is 1 year and conventional interferon is 16 weeks and with oral anti-virals for at least 6 months after seroconversion to anti HBe positive state has occurred.

Treatment in HBeAg negative patients

Duration of treatment with pegylated interferon and conventional interferon is one year and with oral anti-virals is at least for 1 year beyond HBVDNA becoming negative.

Drugs

Entecavir or Tenofovir or pegylated / conventional interferon are the preferred first line drugs.

Lamivudine, Adefovir or Telbivudine are not preferred as single agents because of higher risk of resistance.

Conventional or pegylated interferon is not preferred in those with cirrhosis.

Patients who develop end-stage decompensated liver disease due to hepatitis B should be considered for liver transplantation. With potent antiviral drugs, the chances of recurrence of Hepatitis B after transplantation have reduced considerably.

Recommendations for Hepatocellular carcinoma (HCC) screening in HBsAg positive individuals by 6 monthly ultrasound abdomen and AFP in the following

- HBV carriers at high risk for HCC - men > 40 and women > 50 yrs
- Presence of cirrhosis
- Persons with a family history of HCC
- Persistent or intermittent ALT elevation
- High HBV DNA level >2,000 IU/mL.

Counseling of HBV-Infected Patients

- Have sexual contacts and household contacts vaccinated
- Use barrier protection if partner not vaccinated or nature immune
- Not to share toothbrushes or razors
- Cover open cuts and scratches
- Clean blood spills with detergent or bleach
- Not donate blood, organs or sperms
- Can participate in all activities including contact sports
- Children should not be excluded from participation in school activities and should not be isolated from other children
- Can share food, utensils
- Limit use of alcohol
- Vaccination against hepatitis A (if anti HAV IgG negative).

- Breast-feeding of infants of chronic HBV carriers - no additional risk for the transmission of the hepatitis B with appropriate immunoprophylaxis, (Hepatitis B immune globulin 100 units IM given within 12 hours of birth and hepatitis B vaccine).

Prevention

- Hepatitis B recombinant vaccine has been recently incorporated in the national immunisation programme.
- High risk adults should be vaccinated
- Doctors and paramedical staff should follow "universal precautions" in dealing with blood and body fluids of all patients assuming they are positive
- Standard methods of waste disposal should be followed.

Key messages

- HBV infection is an epidemiologic and a clinical challenge and is preventable
- Screening at-risk individuals identifies those with HBV infection
- ALT and HBV DNA predict development of cirrhosis/hepatocellular carcinoma
- Candidates for anti-HBV treatment include patients with active liver disease (high ALT) and high levels of HBV DNA.

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Universal Precautions for Health Care Personnel

	WASH Before and after patient contact.
	GLOVES Before touching Blood and body substances.
	GOWN When soiling is likely to occur.
	MASK AND GOGGLES When it is likely that mucous membranes or eyes will be splashed with blood or body fluids.
	SHARPS Place used needles in sharp container. DONOT recap.
	WASTES Use appropriate colour coded bags for waste disposal.
	LINEN If linen is heavily soiled with body substances, double wrap it before placing it into laundry bag.

by: Dr. Amrita Misri

Anaesthesia for Liver Disease

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Introduction

Liver is the largest internal organ which plays a critical role in homeostasis of many physiologic systems including drug and nutrient metabolism, synthesis of plasma proteins, detoxification and elimination of many endogenous and exogenous substances.

Acute and chronic liver disease impairs response to anaesthesia and surgery, in proportion to the degree of hepatic dysfunction. Also, certain anesthetic agents can induce alteration in postoperative hepatic function.

Drug metabolism and liver disease

Majority of drugs including anaesthetic drugs are metabolised in liver. Since the liver has a large functional reserve, these functions are affected only in end stage liver disease.

- Absorption - Since portal blood flow is decreased in cirrhosis, there is a decrease in the first pass metabolism of drugs.
- Biotransformation -
 - *Phase I* - In early alcoholic liver disease, Cytochrome P450 is induced resulting in rapid drug metabolism. In late stages, activity of enzymes decreases resulting in delayed drug metabolism.
 - *Phase II* - Conjugation with sulphates, glucuronic acid e.g. propofol, lorazepam, oxazepam. This is preserved till advanced disease, but as liver blood flow decreases, drug metabolism also decreases.

- Protein binding - Hypoalbuminemia results in increased levels of free drug resulting in profound drug effect.
- Volume of distribution - Ascites, retention of water and electrolytes cause increase in volume of distribution of drugs.
- Excretion - Obstructive jaundice results in decreased biliary excretion of drug and its metabolites
- Pharmacodynamics - Altered blood brain barrier in hepatic encephalopathy increases the sensitivity of the brain to CNS depressants.

Liver Function Tests

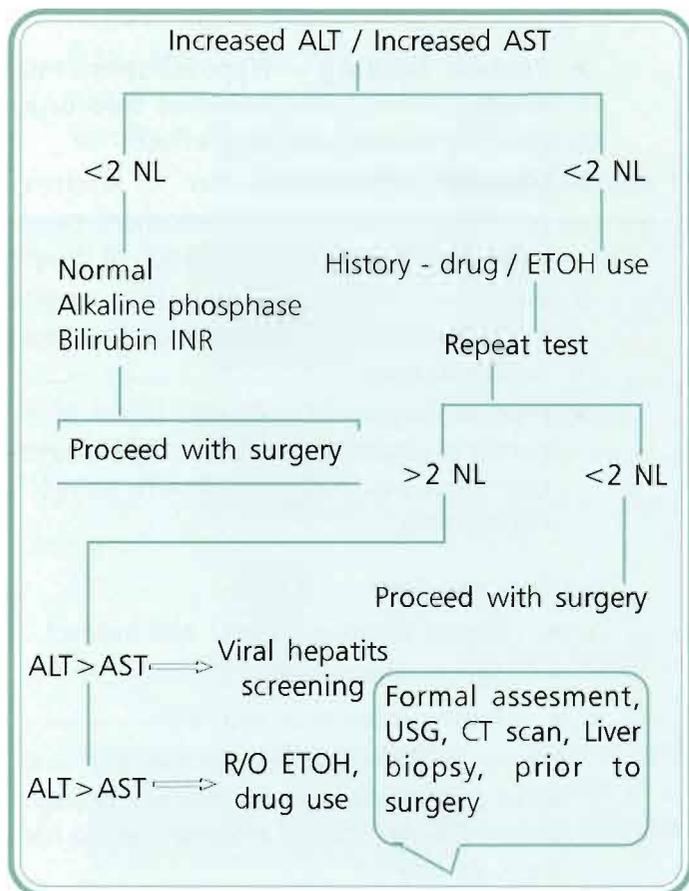
- Serum Bilirubin - Direct and Indirect
- Serum Albumin
- Prothrombin time with INR

(These indicate synthetic functions of liver. INR is a more sensitive indicator as it changes within 24 hrs than S.Albumin whose half life is 21 days).

- Liver transaminase levels - Alanine aminotransferase (ALT), aspartate transaminase (AST), sensitive to mild liver damage.
- Serum Alkaline phosphatase (ALP) - Increased with biliary obstruction.
- Gamma - glutamyl - transferase (GGT) - more sensitive to hepatobiliary disease, alcoholic liver disease.
- Immunological tests - antinuclear antibodies in chronic active hepatitis, anti smooth muscle antibodies in primary biliary cirrhosis, alpha - fetoproteins marker of hepatoma.
- Imaging studies - Ultrasonography, CT, ERCP, MRCP.

Assessment of Risk factors for postoperative hepatobiliary complications

1} Asymptomatic preoperative liver test abnormalities)



2} Acute hepatic disease

- Hyperacute hepatic failure - within 7 days
- Acute hepatic failure - 7 - 28 days
- Subacute hepatic failure - 28 days - 6 mths.

Causes

- Viral:- Hepatitis A - G, CMV, Epstein barr virus, Herpes simplex
- Drugs - Paracetamol toxicity, idiosyncratic reaction, Halothane
- Toxins - Carbon tetrachloride, Amanita phylloids mushrooms
- Others - HELLP Syndrome, Reye's Syndrome, Wilson's disease.

Management

- ✓ Surgery should be postponed for atleast 30 days after LFT returns to normal
- ✓ Close watch must be kept for deteriorating INR, encephalopathy, hypoglycaemia, acidosis, hypovolemia, hypotension.
- ✓ N acetyl cysteine infusion (in case of paracetamol overdose)
- ✓ Orthotopic liver transplant is the definitive treatment.

3} Steatosis and steatohepatitis

Obesity, diabetes, excessive alcohol intake cause fatty liver. There is a higher risk of postoperative liver failure if histopathology reveals >30 % fatty infiltration.

4} Chronic hepatitis

Any hepatitis lasting >6 months is chronic hepatitis.

Causes

- Chronic hepatitis B develops in 3 % of infected.
- Chronic hepatitis C develops in 75% of infected.
- Others - Alcohol induced, autoimmune, metabolic, drugs (INH, methyl dopa) induced hepatitis.

5} Cirrhosis

Presence of hepatic fibrosis with regeneration nodules is cirrhosis.

Causes

Acquired - Alcohol induced, viral hepatitis, drug induced, secondary biliary cirrhosis, veno - occlusive disease.

Inherited - Primary biliary cirrhosis, haemochromatosis, Wilson's disease, galactosemia, sickle cell disease.

Elective surgery is contraindicated with CTP class C. Mortality increases if sepsis, renal failure, bleeding, encephalopathy or ascites is present.

Surgical Risk Assessment with Modified Child Turcotte Pugh (CTP) score

Parameter	1	2	3
Albumin(gm/dl)	>3.5	2.8-3.5	<2.8
Prothrombin time Seconds prolonged INR	<4 <1.7	4-6 1.7-2.3	>6 >2.3
Bilirubin (mg/dl)	<2	2-3	>3
Ascites	None	Moderate	marked
Encephalopathy	None	Gr I-II	Gr III-IV

Class A: 5-6 points, Class B:7-9 points, Class C:10-15 points.

Preoperative assessment:*Preoperative laboratory investigations*

- ✓ Complete blood count - Haemoglobin – low - bleeding, malnutrition, chronic liver disease WBC count - suggestive of infection Platelet count - Low - Minimum 75,000 acceptable, below that platelet transfusion required.
- ✓ Blood glucose level - Hepatic storage of glycogen, glucose metabolism impaired.
- ✓ Renal function tests and Serum electrolytes - Urea is falsely low, affected with hepatorenal syndrome.
- ✓ Coagulation profile - Prothrombin time (PT) is good marker, International. normalised ratio(INR), accepted upto 1.2
- ✓ Arterial blood gas on room air - hypoxia ie. below 60 if hepatopulmonary syndrome, portopulmonary hypertension, infection.
- ✓ Liver function Tests.

Pre - Operative Preparation

- Anti - aspiration prophylaxis - Proton pump inhibitors and H2 - antagonists to decrease gastric acid secretion and prokinetics to increase gastric emptying are used.
- Premedication - Benzodiazepines in titrated low dose under observation to relieve anxiety. But this is avoided in encephalopathy and acute liver failure.
- Morning dose of diuretics (eg. spironolactone), B blocker (eg.

propranolol), lactulose, vit K, antibiotics if any must be continued.

- Written informed consent must be taken. In case of end stage liver disease, CTP class B and C; a high risk consent must be taken. Potential risks such as massive transfusion of blood and blood products, respiratory failure with postoperative mechanical ventilation, renal failure and dialysis, postoperative management in ICU etc should be discussed preoperatively.
- Patient must be kept 'nil by mouth' for 6 hrs for solid food.
- Adequate cross matched blood and blood products should be kept ready.

Goals of Anaesthesia

- To avoid hepatotoxic drugs
- To maintain hepatic perfusion and oxygen delivery
- To prevent and treat complications,

Monitoring*Noninvasive monitoring*

ECG, Pulsoximetry, NIBP, capnography, temperature.

Invasive monitoring

CVP, Arterial line (for BP & ABG sampling), nasogastric tube, urinary catheter for hourly urine output.

Anaesthesia drugs and liver disease

	Safe	To be used with caution (reduced dose)	Contraindicated
Premedication	Lorazepam	Midaz, diazepam	
Induction	Propofol, Thiopentone		
Maintenance	Isoflurane, desflurane, sevoflurane, nitrous oxide	Enflurane	Halothane(??)
Muscle relaxants	Atracurium, Cis-atracurium	Vecuronium, Succinylcholine	
Opioids	Remifentanil	Fentanyl, alfentanyl, morphine, Pethidine	
Analgesics	Paracetamol	NSAIDS, local anaesthetics	

Regional Anaesthesia

Indicated if coagulopathy is not present.

Since local anaesthetics are metabolised by liver, their dosage should be reduced.

Perioperative complications in liver disease

- **Bleeding** - Due to deficiency of clotting factors, thrombocytopenia, or thrombosthenia.
- **Encephalopathy** - Surgery, sedatives, high protein diet, GI bleed, infection, trauma, hypokalemia, constipation etc. can cause derangement of amino acid metabolism and increase in serum ammonia levels thereby precipitating hepatic encephalopathy.

Grades of hepatic encephalopathy:

- 0 - Alert and oriented
- 1 - Drowsy and oriented
- 2 - Drowsy and disoriented
- 3 - Rousable stupor, restlessness
- 4 - Coma, unresponsive to deep pain

- **Hypoglycemia**
- **Ascites** - fibrotic changes in the liver leads to portal HTN, salt and water retention and low albumin level resulting in ascitis.
- **Infection** - Respiratory, Urinary or spontaneous bacterial peritonitis.
- **Renal failure**
 - **Prerenal** - Due to decreased circulating blood volume or increased renovascular resistance.
 - **Renal** - Due to Acute tubular necrosis or Hepatorenal syndrome.
- **Cardiovascular** - Hyperdynamic circulation, Cardiomyopathy (due to cirrhosis. Alcohol too can decrease cardiac output), Pulmonary hypertension (due to portopulmonary hypertension) may occur.
- **Pulmonary (Hepatopulmonary Syndrome)** - Intrapulmonary shunts and ventilation - perfusion mismatch along with pleural effusion, diaphragmatic splinting from ascites decreases PaO₂ which is resistant to treatment with high oxygen concentration.

➤ Post operative jaundice / Liver dysfunction

Although postoperative liver dysfunction is common, significant liver dysfunction is rare. Hepatitis due to volatile agents is very rare and is a diagnosis of exclusion.

Causes of post operative jaundice

- *Prehepatic cause* -
 - Blood transfusion
 - Hematoma resorption
 - Hemolytic anemia(sickle cell anemia, G6PD, prosthetic valve)
- *Hepatic cause* -
 - Exacerbation of pre existing liver disease
 - Hepatic ischemia- due to hypotension, hypovolemia, hypoxia, cardiac failure
 - Septicemia
 - Drug induced(antibiotic)
 - Viral hepatitis
- *Cholestasis* -
 - *Intrahepatic* - benign, infection, drug - induced
 - *Extrahepatic* - pancreatitis, gall stones, bile duct injury.
 - Halothane Hepatitis - Initial exposure to halothane is associated with transient rise

in liver enzymes and low morbidity. After repeated exposure, halothane oxidative metabolite binds to liver cytochrome to form a hapten which then induces a hypersensitivity reaction and very rarely fulminant hepatic failure.

Conclusion

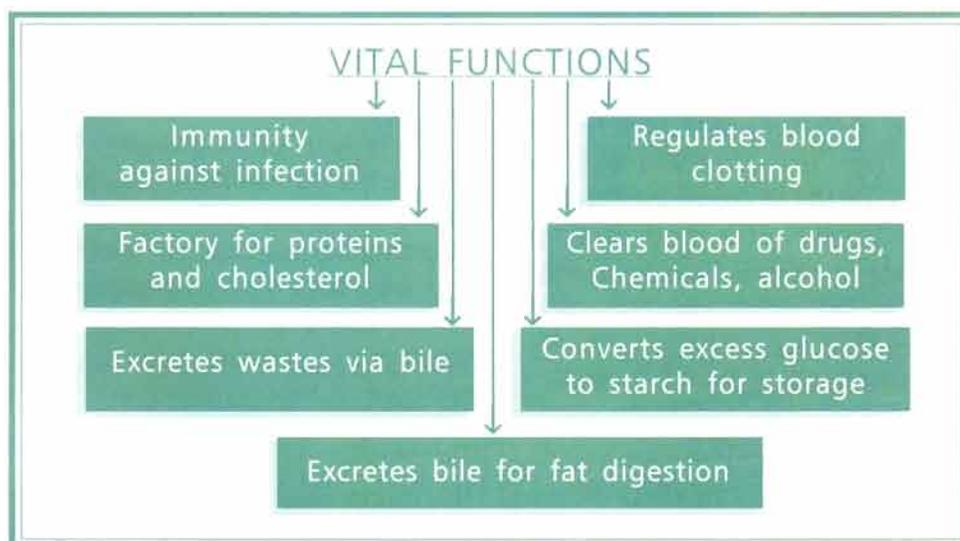
Significant liver disease poses a risk for both surgery and anaesthesia. Hence proper preoperative assessment of liver function and optimisation is important. There is a significant mortality after surgery in patients with liver disease which is directly related to type of surgery and severity of liver disease. Main causes of death are haemorrhage, sepsis, renal failure.

Liver transplantation is now a well established treatment for endstage liver disease with a good 1 year survival of 80%. A team effort involving surgeons, hepatologists, intensivists & anaesthetists is important to ensure a favourable outcome.

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WHAT DOES LIVER DO?



Hepatitis B Virus Infection in Children - Prevention

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Introduction

Hepatitis B virus (HBV) infection is a global public health problem. It is estimated that there are more than 350 million HBV carriers in the world, of whom roughly one million die annually from HBV-related liver disease. It is of particular concern in the pediatric setting, because newborns acquiring HBV infection by perinatal transmission have a greater than 95 percent chance of becoming chronic carriers and up to 25% of chronic carriers die of chronic liver disease as adults.

Epidemiology

The prevalence of HBV carriers varies from <2 percent in low prevalence areas (United States and Canada, Western Europe, Australia and New Zealand), 2 to 7 percent in intermediate prevalence areas (India, Mediterranean countries, Japan, Central Asia, Middle East, and Latin and South America), to >8 percent in high prevalence areas (southeast Asia, China, sub-Saharan Africa).

Modes of Transmission

The predominant mode of transmission of HBV varies in different geographical areas. Perinatal infection is the predominant mode of transmission in high prevalence areas. In comparison, horizontal transmission, particularly in early childhood, accounts for most cases of chronic HBV infection in intermediate prevalence areas, while unprotected sexual intercourse and intravenous drug use in adults are the major routes of spread in low prevalence areas.

Vertical transmission of HBV

Vertical transmission of HBV can occur if the mother has an acute HBV infection near the time

of delivery or if the mother is a chronic HBsAg carrier. Transmission is especially frequent if the maternal serum also is positive for HBeAg. It is 85 to 90 percent in infants born to HBeAg positive mothers and 32 percent in infants born to HBeAg negative mothers. HBV infection is generally asymptomatic, and almost all infected infants become chronic HBsAg carriers. Vertical transmission of hepatitis B also depends on viral load of mother, it occurs in 9 to 39 percent of infants of highly viremic mothers (>8 log copies/mL) despite postnatal vaccination.

Breastfeeding

Breastfeeding does not appear to increase the risk of transmission. With appropriate immunoprophylaxis, breastfeeding of infants of HBV carriers poses no additional risk for the transmission of HBV. Infants, who received HBIG and the first dose of vaccine at birth may be breastfed, as long as they complete the course of vaccination.

Horizontal transmission

Children may acquire HBV infection through horizontal transmission via minor breaks in the skin or mucous membranes or close bodily contacts with other children. HBV can survive outside the human body for a prolonged period; as a result, transmission via contaminated household articles such as toothbrushes, razors, and even toys may be possible. Although HBV DNA has been detected in various bodily secretions of hepatitis B carriers, there is no firm evidence of HBV transmission via body fluids.

Transfusion

The incidence of transfusion - related hepatitis B decreased significantly after the introduction of

hepatitis B surface antigen (HBsAg) and anti-HBc (hepatitis B core antibody) are used for donor screening. To further reduce the risk of transfusion associated HBV infection, nucleic acid testing (NAT) is being considered in screening blood donors for HBV.

Sexual transmission

Sexual transmission remains the major mode of spread of HBV in developed countries. It is estimated that heterosexual transmission accounts for approximately 39 percent of new HBV infection among adults. Sexual transmission of hepatitis B can be prevented by vaccination of spouses.

Percutaneous inoculation

Percutaneous transmission usually happens among intravenous drug users who share syringes and needles. Household contacts can also transmit hepatitis B through the sharing of razors or toothbrushes. Certain practices like acupuncture, tattooing, and body piercing have also been associated with transmission of hepatitis B. Public health education and the use of disposable needles or equipment are important in preventing this mode of transmission.

Nosocomial infection

HBV is the most commonly transmitted blood-borne virus in the healthcare setting. Transmission generally occurs from patient to patient or from patient to health care personnel via contaminated instruments or accidental needle sticks. Healthcare workers, particularly surgeons, pathologists, and physicians working in hemodialysis and oncology units, have the highest risks of HBV infection. Nosocomial transmission can be prevented by screening of blood and blood products, use of disposable needles and equipment, proper sterilization of surgical instruments, enforcement of infection control measures and vaccination of healthcare workers.

Prevention

Infection with HBV is one of the most important causes of chronic hepatitis, cirrhosis of liver and hepatocellular carcinoma. These outcomes can be prevented by early childhood immunization. It is for this reason that the World Health Organization has recommended universal Hepatitis B vaccination.

Vaccine

The currently available vaccine containing the surface antigen of Hepatitis B is produced by recombinant technology in yeast and adjuvanted with aluminum salts and preserved with thiomersol. Hep B vaccine is available as single and multidose vials and should be stored at 2 to 8° C. The vaccine should not be frozen; frozen vaccine should be discarded. The dose in children and adolescents (aged less than 18 years) is 0.5 ml/10 µg and in those 18 years and older is 1 ml/20 µg. It should be injected intramuscularly in the deltoid/anterolateral thigh. Gluteal injections should be avoided due to low immunogenicity. The vaccine is extremely safe and well tolerated. The classical schedule is 0, 1 and 6 months. The vaccine is highly immunogenic and seroconversion rates are greater than 90% after a three dose schedule. Seroconversion rates are lower in the elderly, the immunocompromised and those with chronic renal failure. Four doses at 0, 1, 2 and 12 months of double dose may be given in these patients. Routine testing for anti HBsAg levels after completion of the immunization schedule is recommended in children born to HBsAg positive mothers, health care workers and those with co morbidities. Antibody titers greater than 10 mIU/ml signify a response and are considered protective. Non responders should be tested for Hepatitis B carrier status. If found to be negative the same three dose schedule should be repeated. 50% of non responders may respond to the second series; the rest are permanently susceptible. Routine boosters are not needed in healthy children and adults.

Studies have shown that individuals who had responded to the vaccination series and had levels of 10 mIU/ml after vaccination are protected against hepatitis B disease for life even if the levels drop to below protective levels or are undetectable later. This is due to immune memory. In the immunocompromised and those with co morbidities such as chronic renal disease, levels should be checked periodically and booster vaccination given whenever levels drop to below protective levels.

Recommendations for Use

Hep B vaccine may be given in any of the following schedules:

- a. Birth, 1 and 6 months
- b. Birth, 6 and 14 weeks
- c. 6, 10 and 14 weeks

Catch up vaccination with Hep B vaccine as a 0, 1, 6 schedule should be offered to all children/adolescents who have not been previously vaccinated with Hep B vaccine. This is to address problems related to horizontal mode of transmission of the virus. Pre vaccination screening with anti HBsAg antibody is not cost effective and is not recommended. Catch up vaccination is particularly important for contacts of HBsAg positive patient. Pre vaccination screening for HBsAg should be done in these contacts. All available brands of Hepatitis B vaccine are equally safe and effective and any may be used. Interchange of brands is permitted but not routinely recommended

Hepatitis B Immunoglobulin (HBIG)

HBIG provides passive immunity and is indicated along with Hep B vaccine in management of perinatal/occupational/sexual exposures to Hepatitis B in susceptible individuals. The dose of

HBIG in adults is 0.06 ml/kg and in neonates/infants 0.5 ml. HBIG should be stored at 2 to 8° C and should not be frozen. HBIG provides temporary protection lasting 3-6 months. HBIG should never be given intravenously. HBIG is also used alone following exposure to Hepatitis B in patients who are non responders to Hepatitis B vaccination (genetic reasons/immunocompromised status). In this situation two doses of HBIG 1 month apart are indicated.

Infant Born to Hepatitis B Positive Mother

Pregnant women should be counseled and encouraged to opt for HBsAg screening.

- (A) If the mother is known to be HBsAg negative, Hep B Vaccine can be given along with DTP at 6, 10 and 14 weeks/6 months as there is no special requirement to start vaccination at birth itself. The 6 – 10 – 14 weeks schedule may be easier to implement in the context of the national immunization program as higher vaccination coverage maybe achieved with earlier administration of vaccines.
- (B) If the mother's HBsAg status is not known, it is important that Hep B vaccination should begin within a few hours of birth so that perinatal transmission can be prevented. Any one of the following schedules may be used for this purpose; birth, 6 and 14 weeks or birth, 6 weeks and 6 months
- (C) If the mother is HBsAg positive (and especially HBeAg positive), the baby should be given Hepatitis B Immune Globulin (HBIG) along with Hep B vaccine within 12 hours of birth, using two separate syringes and separate sites for injection. The dose of HBIG is 0.5 ml IM. HBIG may be given up to 7 days of birth but the efficacy of HBIG after 48 hours is not known. Two more doses of

Hep B vaccine at 1 month and 6 months are needed. The closely spaced schedule should not be used. If HBIG is not available (or is unaffordable), Hep B vaccine may be given at 0, 1 and 2 months with an additional dose between 9 - 12 months. The efficacy of prophylaxis with both HBIG and Hep B vaccine is 85 - 95% and that with Hep B vaccine alone (1st dose at birth) is 70 - 75%. All infants born to HBsAg positive mothers should be tested for HBsAg and anti HBsAg antibodies at the age of 9 - 15 months to identify carriers/non responders.

Hepatitis B vaccine and premature infants

Preterm infants with birth weights >2 kg produce an immune response to the HBV comparable to that in term infants. Hence HBV immunization is to be administered to premature infants according to the HBsAg status of the mother and the birth weight of the infant as follows

- If the mother is HBsAg-negative and the birth weight is >2 kg, the infant should be immunized as if he or she were full - term
- If the mother is HBsAg-negative, the birth weight is <2 kg, and the infant is medically stable, the infant should be immunized at 30 days of chronologic age or at hospital discharge if discharge occurs before 30 days of age.
- If the mother is HBsAg-positive, the vaccine series should be initiated and HBIG administered as soon after delivery as possible, regardless of birth weight. Infants who weigh <2 kg at birth will require a fourth dose of vaccine a birth dose is not counted towards the three dose series.
- If the mother's HBsAg status is unknown and the birth weight of the infant is <2 kg, the infant should receive monovalent HepB vaccine and HBIG within 12 hours of birth if maternal status cannot be determined by that time or is positive. Once the maternal HBsAg status is determined, subsequent doses of HepB vaccine should be administered accordingly.

Immunoprophylaxis summary for hepatitis B

	<i>Pre exposure prophylaxis</i>	
0-18 years		Hepatitis B vaccine series
	<i>Post exposure prophylaxis</i>	
Perinatal exposure		HBIG and vaccine series
	<i>Mucosal/percutaneous</i>	
Known titer 10 mIU/mL		No intervention
Immunized, low/no titer		Hepatitis B vaccine single dose
Unvaccinated		HBIG and vaccine series

Liver Diseases in Pregnancy

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Jaundice was prevalent in India even in the Rig Vedic period, since about 1500 BC (Wilson, 1850). Jaundice in pregnancy is an important medical disorder in the developing countries. It is responsible for about 10% of maternal deaths in India. Jaundice is the yellow discoloration of the skin, sclera and mucous membrane due to increased level of bilirubin in the serum (more than 3 mg/dl). Internal tissues and body fluids are coloured yellow, except brain, as bilirubin does not cross the blood-brain barrier.

Physiological Changes in the Liver in Pregnancy

Understanding physiologic changes in normal pregnancy is vital if pathologic conditions are to be correctly defined. The liver has several important functions such as protein synthesis, metabolism, excretion, inactivation of number of substances. There is no change in blood flow in liver. Protein synthesis increases in pregnancy, with rise in coagulation factors VII, VIII, X and fibrinogen. Fibrinogen usually gets doubled by end of pregnancy. Normal pregnancy may show palmer erythema and vascular spiders. In normal pregnant women compared with control, a study of biochemical test showed little difference. Biochemical test shows increased S. Alkaline phosphatase in the last trimester. The rise is due to leakage of placental alkaline phosphatase into maternal circulation and increased maternal bone turnover. Alkaline phosphatase levels may remain elevated for upto 6 weeks post partum.

S. bilirubin is slightly lower related to hemodilution. This may also account for reduced albumin, urea, uric acid concentration.

Jaundice may be peculiar to pregnancy such as – acute fatty liver, cholestatic jaundice or jaundice complicating toxemias.

Jaundice may be an intercurrent such as – viral hepatitis or gall stones.

Early Pregnancy

- Hyperemesis gravidarum

Late Pregnancy

- Intrahepatic cholestasis of pregnancy
- Fatty liver of pregnancy
- Pre-eclampsia or eclampsia
- HELLP syndrome

40% of sufferers from acute fatty liver of pregnancy show e/o eclampsia.

Classification of jaundice in pregnancy

Jaundice peculiar to pregnancy

- Hyperemesis gravidarum.
- Intrahepatic cholestasis.
- Acute fatty liver of pregnancy.
- Pre - eclampsia.
- HELLP Syndrome.

Intercurrent jaundice in pregnancy

- Viral hepatitis.
- Drug induced hepatitis.
- Cholelithiasis.
- Hemolysis.

Hyperemesis Gravidarum

In early pregnancy, when severe, shows raised bilirubin and transaminase levels as high as 200 IU/L. Liver biopsy is normal or shows fatty changes. Changes are probably related to malnutrition which returns to normal within a few days of delivery.

Liver histology shows large and pleomorphic mitochondria with paracrystalline inclusions.

Intrahepatic Cholestasis of Pregnancy (ICP)

It is probably caused by the increased levels of estrogen found during pregnancy. ICP occurs usually in the 3rd trimester of pregnancy, but can begin earlier. There is a rise in the bile acid due to incomplete clearance by the liver giving rise to intense pruritis. Symptom is pruritus. Urine is dark, stools are pale and weight loss may be great. Investigations confirm elevated serum levels of bile acids, hyperbilirubinaemia and bilirubinuria which return to normal after 7 to 14 days of delivery.

Management

Primarily symptomatic for pruritus. The prognosis is good. It is a self-limiting disease and needs no special treatment.

- Nutritional support,
- Vit K supplements,
- Cholestyramine may be indicated. Termination of pregnancy may be considered, if the fetus is mature.
- Ursodeoxycholic Acid (UDCA) is a hydrophilic bile acid that improves cholestasis by stimulating biliary excretion of toxic bile acids.
- All women with ICP should be closely monitored during 3rd trimester, especially in twin pregnancy, if it is before 32wks, or previous stillbirth.
- Fetal surveillance should include biweekly NST.
- Pregnancy should be terminated at or before 38 wks.

Acute Fatty Liver of Pregnancy (AFLP)

Acute fatty liver of pregnancy is rare. Incidence is 1 in 10,000 – 15,000 pregnancies. Seen typically in obese primis in last trimester. Its cause is not known. Maternal mortality is commonly in nulliparous and in those who have a male fetus or in twins. Symptoms include nausea, vomiting, abdominal pain, anorexia, jaundice. 50% patients will have signs of pre eclampsia.

In some cases the etiology of AFLP involves abnormalities in mitochondrial fatty acid

oxidation. Beta-oxidation of fatty acids in hepatic mitochondria is a complex process requiring several essential enzymes: mitochondrial trifunctional protein and its subunit, long chain 3 - hydroxyl - CoA dehydrogenase (LCHAD), are the two enzymes of this metabolic process, whose autosomally inherited genetic mutations are most closely associated with AFLP, especially the G1548C mutation of LCHAD. Maternal heterozygosity for LCHAD deficiency reduces the maternal capacity to oxidize long-chain fatty acids in both liver and placenta and this, together with the metabolic stress of pregnancy and fetal homozygosity for LCHAD deficiency, causes accumulation in the maternal circulation of potentially hepatotoxic LCHAD metabolites.

Management

Spontaneous resolution follows delivery but caesarean section is contraindicated due to coagulation failure. Observation, if mother's status deteriorated the pregnancy should be delivered. Coagulopathy, renal failure, hypoglycemia and infection are treated.

The prognosis is relatively favorable if intensive care is adequate. Intra - abdominal hemorrhage may necessitate laparotomy for clot evacuation. Hepatic transplantation is sometimes necessary.

Pre Eclampsia

Icterus is extremely rare and is reported only in severe cases. It is doubtful whether the jaundice in these cases was due to preeclampsia per se or due to acute fatty liver of pregnancy.

HELLP Syndrome (Hemolysis, elevated liver enzymes & low platelets)

HELLP syndrome is associated with pre-eclampsia and affects 0.1%-0.6% of all pregnancy. It develops ante partum in 70% and postpartum in 30%. Patient presents with right upper quadrant pain, malaise, nausea, vomiting. Syndrome usually resolves after delivery. Rarely it may worsen progressively after delivery, leading to severe thrombocytopenia, septicemia, DIC, multisystem organ failure including liver failure. Vascular endothelial damage leads to platelet deposition, thrombocytopenia and fibrin deposition in

sinusoids. The resultant ischemia accounts for the focal and diffuse hepatocellular necrosis and hemorrhages. Elevated liver enzymes are due to periportal hepatic necrosis. Jaundice is infrequent and often terminal.

Management of HELLP syndrome is primarily supportive. Patient should be monitored intensively until delivered safely. The delivery should be accomplished by the safest route. Perinatal mortality is 10-60%. Maternal mortality is 1.5-5%. Subcapsular hemorrhage is treated conservatively. Hepatic artery embolization with gel foam can be used to control hemorrhage. Surgery or even transplant may be required.

Viral Hepatitis

Viral Hepatitis is the common cause of jaundice in pregnancy. Viral hepatitis is caused mostly by 5 hepatotropic viruses – A, B, C, D, E and G. clinical significance of Hepatitis G is not clear. Cytomegalovirus, Epstein – Barr virus and Herpes Simplex virus are rare causes of hepatitis.

About 2/3rd of patients remain asymptomatic (asymptomatic hepatitis) or may have only viral prodrome without jaundice (anicteric hepatitis). Prodromal symptoms of acute viral hepatitis precede jaundice by 1 or 2 weeks and include fever, anorexia, malaise, nausea, vomiting, fatigue, arthralgia, myalgia and headache. Cholestasis symptoms such as pruritus and clay stools are seen in Hepatitis E and A. Liver becomes enlarged and tender. During recovery jaundice starts receding. The duration of post icteric phase ranges from 2 to 12 weeks. Increase in serum levels of ALT and AST is due to liver cell necrosis.

Treatment is mainly supportive: High caloric diet, IV hydration. Drugs metabolized by liver should be avoided, Ursodeoxycholic Acid or Cholestyramine may be used if there is marked pruritus.

In conclusion, the commonest cause of icterus in pregnancy is viral hepatitis. The acute infections caused by HAV and HEV are prevented by improved sanitation, supply of protected water

or by using boiled water for consumption. Immunization is possible in high risk patients in HAV and perhaps shortly in HEV also. Phase III trials with new HEV vaccine are being conducted in Nepal where HEV hepatitis is endemic (7) (Stevenson, 2000). In cases of HBV and HCV, infections may be prevented by proper screening of blood donors, use of sterile needles and syringes, and modification of sexual behavior. Each pregnant woman should be routinely screened for HBV and if positive, the infant should be immunized soon after birth.

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Gastric Carcinoid – a case report

Dr. Jayesh Kalbhande

Medical Officer

Department of Surgery

A 44 yr male patient presented with symptoms of epigastric fullness, after eating food, associated with discomfort. In view of symptoms of acid peptic disease, an endoscopy was carried out. Endoscopy showed multiple small subcentimeter size polyps in stomach. He also had Diabetes and hypothyroidism; both these conditions were controlled with medications.

His OGD scopy showed multiple subcentimeter-size polyps, located more in body and fundus, whereas antrum and pylorus did not show any polyps. Multiple biopsies from the polyp were taken. The histopathology report of the biopsy showed that polyps were carcinoid tumor.

To further confirm the diagnosis of carcinoid tumor, his serum chromogranin A level was measured which was elevated, 106.3 (Normal 2 – 18) and 24 hours urinary 5 HIAA (Hydroxy Indol acetic acid) levels was measured which was also elevated, 8.8 mg (Normal 2.0 to 6.0 mg). Both these reports confirmed the diagnosis of carcinoid tumor.

To further investigate the nature of polyp, his serum gastrin level was measured, which was elevated, 413pg/ml (Normal 77 to 181 pg/ml). Thus he had multiple carcinoid tumor associated with hypergastrinaemia. Type I or Type II gastric carcinoid are associated with hypergastrinaemia.

Type I gastric carcinoids are associated with atrophic gastritis hence gastric pH is high and Type II gastric carcinoids are associated with gastrinoma, hence gastric pH is low. To differentiate between Type I and Type II gastric carcinoid, his gastric PH was measured by pH metry, which showed hypochlorhydria state. His CT scan of abdomen and pelvis was carried out to look for presence of a pancreatic mass

(Gastrinoma) or presence of metastatic disease as seen in malignant transformation of gastric carcinoid. The CT abdomen and pelvis showed normal finding.

His MRI brain was done to rule out pituitary tumor because Type II carcinoid is associated with multiple endocrine neoplasia (MEN I), with associated pituitary lesion. His MRI brain pituitary fossa was normal and no pituitary gland tumor was found. His serum cortisol levels were checked and found normal, which again indicated normal function of pituitary gland thus ruling out any pituitary gland functional abnormality.

Type I gastric carcinoid as seen in this patient is associated with hypochlorhydria secondary to atrophic gastritis. To confirm the presence of atrophic gastritis, anti parietal cell and anti intrinsic factor antibody levels were carried out. Anti parietal cell antibody level, was strongly positive i.e. 1:320 Normal being 1:20 and anti intrinsic factor antibody was Normal 1.6 mcg% (refer the range 6 – 20 mcg%). This confirmed that the patient had auto immune disorder causing atrophic gastritis.

Thus the diagnosis of Multiple Gastric carcinoid of Type I variety secondary to Atrophic gastritis. He was explained the benign nature of this disease. He was given a choice between

- 1) Wait and watch - endoscopic surveillance only.
- 2) Endoscopic removal of polyps.
- 3) Antrectomy.

He chose endoscopic removal of polyp as the treatment choice. Most of his polyps were removed endoscopically. As the polyps were multiple, their complete excision was not possible. He is right now under endoscopic surveillance for recurrence of polyps.

Discussion

Gastric carcinoid tumors arise from enterochromaffin (ECL) cells. Gastric carcinoids are rare lesions, accounting for less than 2% of all carcinoids tumors and less than 1% of all stomach neoplasms. The male: female ratio for gastric carcinoid is about 1:2, with 64% of carcinoids found in women, whereas males are almost twice as likely to develop non-carcinoid gastric-cancer.

These tumors are classified into three distinct types.

Type 1 Gastric carcinoid: They account for (70%-85%) of total Gastric carcinoids and are associated with chronic atrophic gastritis type A, characterized by decreased acidity, resultant hypergastrinemia causing ECL cell hyperplasia. The lesions are located in the gastric fundus and body, are multiple, small, limited to the mucosa or submucosa, without angioinvasion, well-differentiated and tend to display benign behavior. They are more frequent in females.

Type 2 Gastric carcinoid: They account for 5%-10% of total Gastric carcinoids, are associated with Gastrinoma (Zollinger Ellison Syndrome). They are most often seen in Multiple Endocrine Neoplasia-1. They have small duodenal or pancreatic gastrinomas causing hypergastrinemia which leads to ECL cell proliferation. Type 2 gastric carcinoids are usually multiple and small. They may show malignant change; up to 35% of cases are metastatic at presentation. Unlike GC-1, GC-2 is equally frequent in male and female patients.

Type 1 and type 2 gastric carcinoids are both associated with hypergastrinemia. In Type 1 gastric carcinoid, hypergastrinaemia is secondary to hypo/achlorhydria caused by the destruction of gastric parietal cells. Hypochlorhydria stimulates formation of more gastrin from gastrin secreting cells which are located in antrum of stomach. Type 2 gastric carcinoids are caused by gastrinoma tumor. Gastrinoma produce large amount of gastrin which cause

hyperchlorhydria. Therefore pH of gastric juice is useful to discriminate the presence of atrophic gastritis from Gastrinoma (ZES) / (MEN1). Atrophic gastritis is characterized by increased gastric juice pH and presence of anti - parietal cells and/or anti intrinsic factor antibodies. Gastrinoma ZES/MEN1 is associated with low gastric juice pH and basal acid output ≥ 15 mEq/h.

Type 3 Gastric carcinoid: They account for 15%-25% of gastric carcinoids. They are not associated with hypergastrinaemia. They are single tumors and are usually malignant and are associated with aggressive course. They may be present with lymph node and distant metastases in more than 50% of cases. Lesions are typically solitary, larger than 1-2 cm, ulcerated and deeply invasive. They are usually located in the gastric fundus and body, but may also occur in the antrum.

Management

Type 1 Gastric carcinoid

1) Observation

European NeuroEndocrine Tumor Society (ENETS) Consensus Guidelines have suggested that annual surveillance is appropriate when dealing with patients with Type 1 GC of less than 10 mm in size. They are mostly benign. The average life span of Type 1 gastric carcinoid matches with that of normal general population. They usually do not show malignant transformation. However, tumor more than 1 cm in size, may rarely show malignant change. Hence they require regular surveillance.

2) Endoscopic mucosal resection

In case of tumors more than 10 mm in size and not involving the muscularis propria at EUS examination and if they are less than 6 in number, endoscopic resection is an optimal approach.

3) Antrectomy

In case of tumor size more than 10 mm and more than 6 in number, Antrectomy is an optimal choice of treatment.

Type 1 Gastric carcinoid is associated with atrophic gastritis which causes hypochlorhydria.

Hypochlorhydria stimulates the 'G' cells to produce more of gastrin. The Gastrin producing 'G' cells are located predominantly in antrum which is a distal part of stomach. Gastrin has physiological action of producing more acid output, which may then correct the hypochlorhydria. However the acid producing parietal cells are destroyed by the parietal cell antibody. Therefore Hyper-gastrinaemia thus produced, never corrects the hypochlorhydria. At the same time hypergastrinaemia stimulates proliferation of enterochromaffin cells, thus producing gastric carcinoid. Removal of antrum would remove most of the gastrin producing 'G' cells and thus will cure hypergastrinaemia and enterochromaffin cell proliferation will be stopped, thus preventing the formation of new carcinoid tumors.

In the presence of deep gastric parietal wall invasion and positive margins following endoscopic mucosal resection, surgical resection of the tumor should be carried out. These tumors often being multiple and recurrent, antral resection, aimed at avoiding chronic ECL cell stimulation by ongoing hypergastrinemia, is recommended, which is effective in 80% of Type 1 tumors.

Type II Gastric carcinoid

The management of Type 2 GC has to be approached in the context of the MEN-1 syndrome that is present in these patients. As for Type 1 GC, endoscopic treatment is an option for benign tumors. Gastric surgery should be performed only in highly selected patients, particularly if the histological examination shows the features of poorly differentiated endocrine tumors.

Type III Gastric carcinoid

Management of Type 3 GC is fairly clear and comparable to that used for gastric

adenocarcinomas, which includes partial or total gastrectomy with extended lymph node resection.

Prognosis

Gastric carcinoids have overall survival rate of 78%. Type I gastric carcinoid tumors have a life expectancy comparable to that of the general population. Type 2 Gastric carcinoid overall survival is closely related to the course of the associated gastrinoma, with a 5-year survival of 62%-75%. Type 3 GCs have the worst prognosis and are typically associated with an overall 5-year survival of < 50%.

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Adenocarcinoma of the jejunum: a case report

Dr Susan Cherian, Medical Officer, Dr Shivali Mahagaonkar, Resident Medical Officer,
Dr R.K.Kulkarni, Head Pathology Unit
Dr Jayesh Kalbhande, Medical Officer, Surgical Unit

Abstract: Adenocarcinoma of the small intestine is found 40-60 times less than its counterpart in the colon. The most common site of adenocarcinoma in the small intestine, is the duodenum. Adenocarcinoma of the jejunum is a very rare entity. The symptomatology is generally vague. Survival of patients is poor due to detection at an advanced stage of tumor. Here we present a rare case of adenocarcinoma of the jejunum.

Introduction

Malignant disease of the small intestine is rare. The frequency is 0.7-1.6 per 100000 population. Although the small intestine represents 75% of the length and 90% of the surface area of the alimentary tract, small bowel malignant tumours account for only 2% of all gastrointestinal neoplasm.^{1,2} In cases of jejunal carcinoma, as the site of the lesion is not accessible to routine endoscopy, the malignancy is detected late and in most of the cases is locally advanced.

Case report:

Our patient was a 79 yrs old lady who presented with on and off epigastric dull aching pain. She had an occasional non-projectile non-bilious vomiting. There was on and off constipation since 6 months but no history of obstipation and abdominal distention. She had 10 kg weight loss during this period. There was no history of malaena or jaundice.

Her OGD scopy showed erythematous gastritis and hence she was treated with proton pump inhibitor. Her haemogram was normal with hemoglobin of 11 gm%. Stool occult blood was negative which meant that there was less possibility of bleeding lesion in the gastrointestinal tract. However, she was still symptomatic, therefore to rule out any terminal

ileal and colonic pathology, ileocolonoscopy was performed, in which entire colon and terminal ileum was visualized and was found to be normal. Random colonoscopic biopsies on histopathology showed normal colonic mucosa. The CT scan of abdomen and pelvis with contrast was also performed, which showed normal findings. In spite of performing all the above tests and treatment with proton pump inhibitor, the patient still remained symptomatic, so a small bowel enema was performed. Small bowel enema revealed a concentric growth with shouldering effect on either side in the mid jejunal coils with marked proximal dilation.

Diagnostic laparoscopy with Exploratory Laparotomy was planned. Intraoperatively, there was no free fluid in the abdomen. 3cm long jejunal stricture was present in the second loop of jejunum with a mesenteric node in the vicinity. Around 8cm of the bowel on each side of the stricture was resected along with the mesentery and lymph nodes. Jejunal anastomoses was done. The resected specimen was sent for histopathology.

On gross examination, a jejunal segment of length 15cms and greatest diameter of 3.5cm with attached mesentery was received. There was an area of stenosis visible externally, On cutting open, an area of stricture measuring 1.0x1.5cms was seen. The mucosa over the stricture was ulcerated, greyish-white, infiltrative and the thickening extended deeply into the entire thickness of the wall. Consistency was firm. The area was 9cm from proximal end of resection and 11cm from the distal end (Fig. 1). Rest of the mucosa was normal, did not show any other polyp/stricture. Six lymph nodes were dissected from the mesenteric fat at the area of stricture.



Fig. 1: Gross photograph showing a stricturous grayish-white growth in jejunum

Histopathology of the sections taken from the area of stricture, showed moderately differentiated adenocarcinoma (grade II) (Fig. 2).

The mucosa was ulcerated, tumor cells were seen infiltrating through the muscularis propria into the subserosa—pathologic staging pT3 (Fig. 3).

The mucosa adjacent to the tumour show features of high grade dysplasia. Lymphatic emboli were seen. Vascular emboli were not seen. The proximal, distal and peritoneal surgical margins were uninvolved by tumour. No intratumoral/peritumoral lymphatic response was seen. Sections from the adjacent jejunal mucosa away from the neoplastic growth did not show

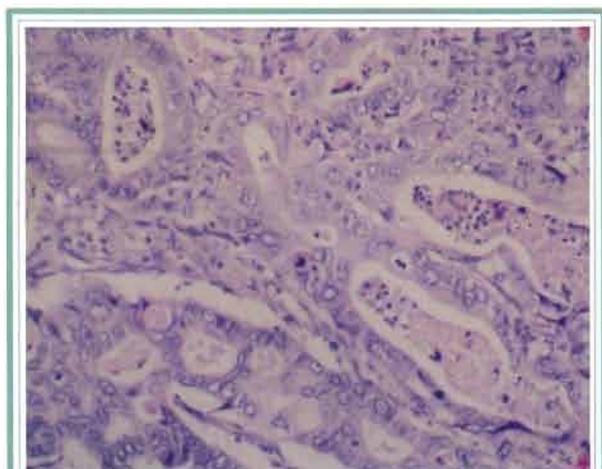


Fig 2: Moderately differentiated adenocarcinoma

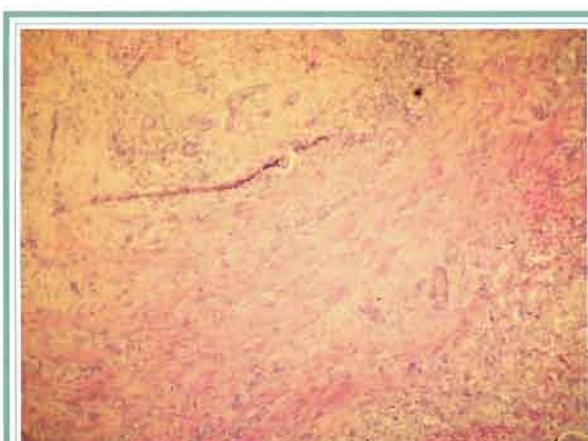


Fig. 3: Adenocarcinoma of the jejunum infiltrating beyond the muscularis propria

any features suggestive of celiac disease like subtotal villous atrophy/blunting associated with crypt hyperplasia and an increase in chronic inflammatory cell infiltrate in the lamina propria. Features of Crohn's disease were also not seen in the adjacent bowel mucosa and wall. Adenomatous polyps were not present in the resected segment of the intestine. Two of the six lymph nodes showed metastasis (Fig. 4).

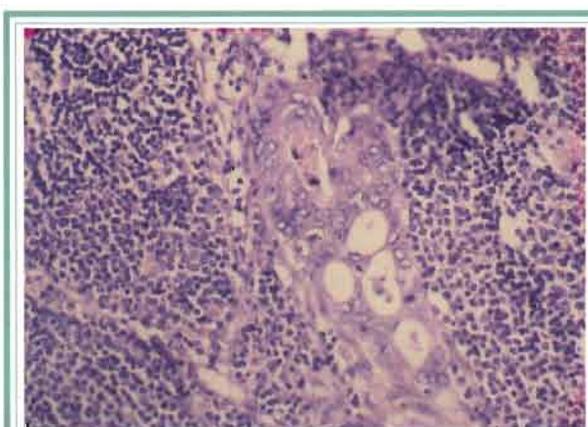


Fig 4: Adenocarcinoma of jejunum metastatic to the lymph node.

Post-operative recovery was good. The patient was not given chemotherapy / radiotherapy in view of her age and was to be kept under observation with regular follow - up. CEA level was less than 1.0ng/ml (normal range 0.0-8.0ng/

ml) post-operatively. She died 7 months later following recurrence.

Discussion

Neoplasms of the small intestine are uncommon accounting for approximately 2% of all gastrointestinal tumours. The common histologic types of malignant tumours, of the small intestine, are as follows: Adenocarcinoma 45%, carcinoid 29%, lymphoma 16% and sarcoma 10%.³ Carcinomas, which account for somewhat less than a third of the total, generally grow in a "napkin-ring" stenosing fashion but, due to the fluid nature of the intestinal contents, tend to present late in their course.⁴⁻⁵ In a study by Dabaja et al. the common presenting symptoms of small bowel adenocarcinoma were abdominal pain (66%), obstruction (40%) and bleeding (24%). Three fourths of the cases, were stage III (any T –N1-M0) or stage IV (any T-any N-M1) at presentation.⁶ The rarity of these tumours combined with non-specific signs and symptoms can delay prompt diagnosis and treatment. Furthermore, their inaccessibility by endoscopic instruments and the limitations of radiographic techniques hinder their early detection.

Small intestinal adenocarcinomas are rare tumours. Several conditions are also known to be associated with a higher incidence of primary carcinoma in the small bowel. Annual incidence of small intestinal adenocarcinomas is 82 times more common in patients with celiac disease than in the normal population, making it as common a tumour as colon cancer in these patients.⁷ Known predisposing factors for the development of small intestinal adenocarcinomas include: Crohn's disease, adenomatous polyps and Peutz-Jegher's syndrome.² Lioe et al. noted a preexisting villous adenoma adjacent to six (24%) adenocarcinomas, five jejunal and one ileal.² Bridge et al. noted a similar but less common association (12%).⁸ Perzin et al. reported that 23 of 51 cases of small intestinal adenocarcinomas had evidence of adenoma and carcinoma in the same lesion.⁹ Survival is dependent on the presence or absence of nodal

involvement at presentation. Five year survival with node negative disease is 68%, while the overall figure varies from 15-20%.¹⁰

Cancers of ileum and jejunum tend to have a distinct course from cancers of duodenum and ampulla and hence are considered separately. In our case as in most others the cancer was picked up only after it had spread locally. Because of the rarity of malignancy in the jejunum, most cases with a stricturous growth at this site have been mistaken for tuberculous in etiology, hence enteroscopy and biopsy play an important role in the diagnosis of high small bowel strictures.¹¹ Local spread through the wall and to the lymph nodes is an important prognostic factor of the tumor, size of the tumor not being important. In most of the studies carried out, the patients have had poor survival rates.¹² Hence early recognition of these tumors requires a high index of suspicion, which is rare given the rarity of the tumor.

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Suggestions for Hospital Visitors

- Visitors should make plans to visit the patient during posted visiting hours.
- The number of visitors in patient's room at a given time should be limited.
- Children below 12 years of age should not visit the patient.
- Visitors who have experienced coryza, cough, fever, sore throat, vomiting or any infectious conditions e.g. chickenpox, mumps, etc. should not visit the hospital.
- Flowers, fruits, snacks etc. should not be brought in by visitors for the patients.
- Visitors should maintain 'no smoking' policy.
- Visitors should maintain a quiet environment and avoid unnecessary noise.

- Dr. Amrita Misri

Publications / Presentations / Achievements

Departments:

ENT

Poster Presentation

Title: Hearing Aids Statistical Analysis and Satisfaction Survey in Patients with Presbycusis

Authors: Dr. Nalini Bhat, Dr. Pushkar Kasat, Smt. Harshada Tawade.

Presented by: Dr. Nalini Bhat.

Venue: Annual Conference of Indian Society of Otology, at Coimbatore.

Date: 5th-7th February 2010.

Dr. Nalini Bhat was awarded the first prize for poster presentation.

Fellowship

Dr. Nalini Bhat was selected by the Executive Committee of Indian Society of Otology for 'Cause Clinic Indian Society of Otology foreign travel fellowship' for 15 days from 22nd June to 6th July, 2009 in Cause Ear Clinic, Columbiens, France.

Dr. Nalini Bhat Head ENT Unit participated in 'Standard Chartered Mumbai Marathon 2011' on 16th January, 2011. She completed the distance of 21.1Km in a duration of 2hours 33minutes and 2 seconds.



Medical

Paper Presentation

Title: Case of Paracetamol Poisoning

Authors: Dr. Rohan Jadhav, Dr. P.N. Jangale, Dr. A.R. Kulkarni

Presented by: Dr. Rohan Jadhav

Venue: 8th DAE Medimeet, Kaiga

Date: 10th December, 2010.

Obstetrics and Gynaecology

Paper Presentations

Title: Labour Induction in previous one LSCS with unfavourable cervix.

Authors: Dr. Shilpa Singh, Dr. Rashmi Singh, Dr. Santoshi Prabhu, Dr. D.P. Joshi, Dr. N. Mishra, Dr. Amrita Misri.

Presented by: Dr. Shilpa Singh

Venue: 39th Annual Conference of Mumbai Obstetrics & Gynaecological Society at ITC grand Centre.

Date: 5th December, 2010.

This paper was awarded 'Dr. N.A.Purandare prize' for Operative Obstetrics Category.

Title: Rare and Interesting Cases

Authors: Dr. Amrita Misri

Presented by: Dr. Amrita Misri

Venue: 8th DAE Medimeet, Kaiga

Date: 10th December, 2010.

Title: Diagnostic Accuracy of Hysteroscopy in Evaluation of Postmenopausal bleeding.

Authors: Dr. Nigamanand Mishra, Dr. Amrita Misri, Dr. D.P. Joshi, Dr. Santoshi Prabhu, Dr. B.R. Rao Bahadur.

Presented by: Dr. Nigamanand Mishra

Venue: 8th DAE Medimeet, Kaiga

Date: 10th December, 2010

Title: Effect of peritoneal closure on future adhesion formation.

Authors: Dr. Santoshi Prabhu, Dr. D.P. Joshi, Dr. N. Mishra, Dr. Amrita Misri.

Presented by: Dr. Santoshi Prabhu

Venue: 54th All India Congress of Obstetrics & Gynaecology, Hyderabad International Convention Centre, Hyderabad, India.

Date: 7th January, 2011.

Fellowship

Dr. Santoshi Prabhu, Medical Officer was awarded fellowship by the Indian College of Obstetricians and Gynecologists (FICOG) at 54th All India Congress of Obstetrics & Gynaecology, Hyderabad International Convention Centre, Hyderabad, India on 8th January, 2011. Prof. Lord Naren Patel was the guest of honor for the convocation.



Dr. P.C. Mahapatra, President of Federation of Obstetrics & Gynaecological Society of India offering fellowship to Dr. Santoshi Prabhu

Ophthalmology

Publication

Topic: Bilateral Multiple Primary Iris Cyst- A Case Report.

Authors: Dr. Rahul Baile, Dr. Snehal Nadkarni, Dr. V. Karira, Dr. Meghana Sahasrabudhe, Dr. Juilee Kelkar.

Journal: *Saudi Journal Of Ophthalmology*, Year 2011, Volume 25, Pages 85-91.

Paper Presentations

Title: Comparative Study of Post-Operative Astigmatism Using Clear Corneal Superior versus Clear Corneal Temporal Incision by Single Surgeon

Authors: Dr. Rahul Baile, Dr. Snehal Nadkarni, Dr. V. Karira, Dr. Meghana Sahasrabudhe, Dr. Juilee Kelkar

Presented by: Dr. Juilee Kelkar

Venue: *VIII International academy for advances in ophthalmology EYE ADVANCE 2010 and XVIII Annual conference of Bombay Ophthalmology Association FOCUS2010* Mumbai, India

Date: August 2010.

This paper secured the Best Paper Award.

Title: Manual small Incision cataract surgery - Blumenthal and Viscoexpression

Authors: Dr. Rahul Baile

Presented by: Dr. Rahul Baile

Venue: 30th Annual conference of Maharashtra Ophthalmic Society Sangli, Maharashtra.

Date: October 2010

Poster Presentations

Title: Bilateral Optic Disc drusen, a case report, Poster

Authors: Dr. Rahul Baile, Dr. Snehal Nadkarni, Dr. V. Karira, Dr. Meghana Sahasrabudhe, Dr. Juilee Kelkar

Presented by: Dr. Rahul B. Baile

Venue: *VIII International academy for advances in ophthalmology EYE ADVANCE 2010 and XVIII Annual conference of Bombay Ophthalmology Association FOCUS2010* Mumbai, India

Date: August 2010

This poster secured The Best Poster Award.

Title: Comparison of Post Operative Pseudophakic Refraction on 1st and 2nd Post Operative Follow-ups Authors: Jeewan Prakash Srivastava, Dr. Snehal Nadkarni, Dr. V. Karira, Dr. Rahul Baile

Presented by: Jeewan Prakash Srivastava

Venue: EIVOC 2010 Chennai, India

Date: August 12th -14th 2010.

Title: Superficial keratectomy, PTK and MMC as combined treatment for Salzmann's nodular degeneration.

Authors: Dr. Rahul B. Baile

Presented by: Dr. Rahul B. Baile

Venue: World Ophthalmology Congress Berlin, Germany

Date: June 2010

Paediatrics

Publication

Title: Case Report: Morning Glory Syndrome associated with vascular anomaly-hypoplasia of the left common carotid artery.

Authors: Dr. Umesh Kalane, Dr. Sangeeta Sawant, Dr. Alpa Amin, Dr. Shilpa Sule, Dr. Tanuja Karande and Dr. Santosh Kumar.

Journal: *Journal of Pediatric Neurology* 2010; Vol.8 (4): 417-420.

Pathology

Publication

Title: Bull's Multirule Algorithm, an excellent means of internal quality control for hematology analyzers.

Authors: Shagufta S., SKG Shettigar, Manikchandra T., Dr. S. Gujral, Dr. R.K. Kulkarni, BARC Hospital & Tata Memorial Hospital.

Journal: *Medical Technology, window on Science and Technology* 2010; Vol.XX-2010: 15-17.



Rev. Fr. P. Thangasamy, Director, Sagamatha Hospital Pullambadi Trichy awarding 2nd best paper presentation award to Smt. Shagufta Shaikh

Paper Presentation

Title: Bull's Multirule Algorithm, an excellent means of internal quality control for hematology analyzers.

Authors: Shagufta S., SKG Shettigar, Manikchandra T., Dr. S. Gujral, Dr. R.K. Kulkarni, BARC Hospital & Tata Memorial Hospital.

Presented by: Shagufta S

Conference: AIIMT 20th Congress of Biomedical Laboratory Science

Venue: Jubilee Hall, St. Joseph's College, Tiruchirapalli, Tamil Nadu.

Date: 18 -19th December 2010.

This paper was adjudged as the Second Best Paper Presentation Award.

Title: Primary Signet Ring Carcinoma of Breast- A Case Report

Authors: Dr. V. Jayalakshmi, Dr. Susan Cherian, Dr. R.K. Kulkarni, Dr. Uma Chaturvedi

Presented by: Dr. V. Jayalakshmi

Venue: Maharashtra Association of Pathologists Chapter Conference (MAPCON) GMC, Nagpur

Date: 24-26 Sept. 2010.

Title Using Genetic Information in Managing Type 2 Diabetes: a Possibility of Early Intervention?

Authors: Dr. R.K. Kulkarni, S.K.G. Shettigar, Namrata P. Londhe, C. Shailaja, M. Seshadri

Presented by: Dr. R.K. Kulkarni.

Venue: 8th DAE Medimeet, Kaiga.

Date: 9-10 December 2010

Pathology and ENT

Publication

Topic: Epithelial-myoepithelial carcinoma of hard palate-a case report

Authors: Dr. Susan Cherian, Dr. R.K. Kulkarni, Dr. Nalini Bhat.

Name of Journal: *Acta Cytologica*, 2010, Vol.54; 835-839.

Surgical

Publication

Topic: Where there are no emergency medical services – Prehospital care for the injured in Mumbai, India.

Authors: Roy N., Murlidhar V., Chowdhury R., Patil S.B., Supe P.A., Vaishnav P.D., Vatar A.

Journal: *Prehosp Disaster Med* 2010; 25(2): 145-151.

Topic: The Medical Council of India guidelines on industry – physician relationship: Breaking the conspiracy of silence.

Authors: Nagral S., Roy N.

Journal: *National Medical Journal of India* Mar-Apr 2010 Vol.23 (2): 69

Topic: Conflict resolution in the healthcare environment

Authors: Roy N.

Journal: *Indian J Med Ethics*.2010 Jul-Sep; 7(3).

Topic: Genetic variants in the distal enhancer region of the PSA gene and their implication in the occurrence of advanced prostate cancer.

Authors: Chavan SV, Maitra A, Roy N, Patwardhan S, Chavan PR.

Journal: *Molecular Medicine Reports* 3: 837 -843, 2010.

Paper Presentations

Title: Surgical Site Infections

Presented by: Dr. Bhakti Sarang

Venue: ASICON 2010, AIIMS, New Delhi

Title: Benign Breast Diseases

Authors: Dr. A. Gadgil, Dr. Samiksha Mehare.

Presented by: Dr. Samiksha Mehare

Venue: ASICON 2010, AIIMS, New Delhi.

Achievements

Dr. N. Roy, Head Surgical Department, was invited as National Faculty for Training Program on 'GIS Application in Public Health Mapping (GISPM) from September 27th to October 1st 2010, at the Tata Institute of Social Sciences.

Dispensaries

Deonar (West)

Paper Presentations

Title: Study of Prevention and Early Reversal of Early Atherosclerotic changes in Syndrome X Individuals.

Authors: Dr. K.R. Bantwal, Dr. S. Anand, Dr. A.V. Kulkarni, et al.

Presented by: Dr. K.R. Bantwal

Venue: 8th DAE Medimeet, Kaiga

Date: 8th December 2010

Mandala

Paper Presentations

Title: Multifactorial aetiology of obesity: Its prevention and consequences on health.

Authors: Dr. Indrani Haldar, Dr. A.V. Kulkarni

Presented by: Dr. Indrani Haldar

Venue: 8th DAE Medimeet, Kaiga.

Date: 9th December 2010.

Title: Spectrum of Thyroid Disorders with regard to Clinical Pattern, Management & Follow Up in a closed community

Authors: Dr. Debjani Pal, Dr. A.V. Kulkarni

Presented by: Dr. A.V. Kulkarni

Venue: 8th DAE Medimeet, Kaiga.

Date: 9th December 2010.

Title: Multidimensional Approach and Preventive Care Guide Lines in Elderly

Authors: Dr. S. Rajeswari

Presented by: Dr. S. Rajeswari

Venue: 8th DAE Medimeet, Kaiga.

Date: 9th December.

Title: Study of underweight children (1-10 yrs.) with regards to etiology, prevention management and comparison with maternal nutritional status

Authors: Dr. Anita Patil, Dr. A.V. Kulkarni

Presented by: Dr. Anita Patil

Venue: 8th DAE Medimeet, Kaiga.

Date: 10th December 2010

Trombay Occupational Health Unit

Publication

Topic: Immune responses to novel allergens and modulation of inflammation by vitamin K3 analogue: A ROS dependent mechanism

Authors: Vineet Kohli, Deepak Sharma, Santosh K. Sandur, Shweta Suryavanshi, Krishna B. Sainis

Name of the Journal: *International Immunopharmacology*, 2011, Vol.11; 233-243.

Vashi and Dombivili

Paper Presentation

Topic: Association of increased waist circumference and BMI in patients with HT and DM

Authors: Dr. Santosh Wattal, Dr. Rajesh Jha, Dr. Uday Thakre

Presented by: Dr. Santosh Wattal

Venue: 8th DAE Medimeet 2010, Kaiga.

Date: 10th December 2010.

Dental awareness programme



Dental Lab Technician Team



हिंदी विज्ञान साहित्य परिषद द्वारा "दंत रोग-समस्यायें, सावधानियाँ एवं निदान" विषय पर एक दिवसीय संघोष्ठ का आयोजन 19 फरवरी 2011 को किया गया था।

The Hindi Vigyan Sahitya Parishad BARC, headed by Dr. K.B. Sainis had organized an one day seminar as a part of 'Manav Swasth Sangosthi Srinkhala' on "Dental disease, precaution and solution on dental problems", at Multipurpose hall, Training school hostel, Anushakti Nagar on 19th February, 2011.

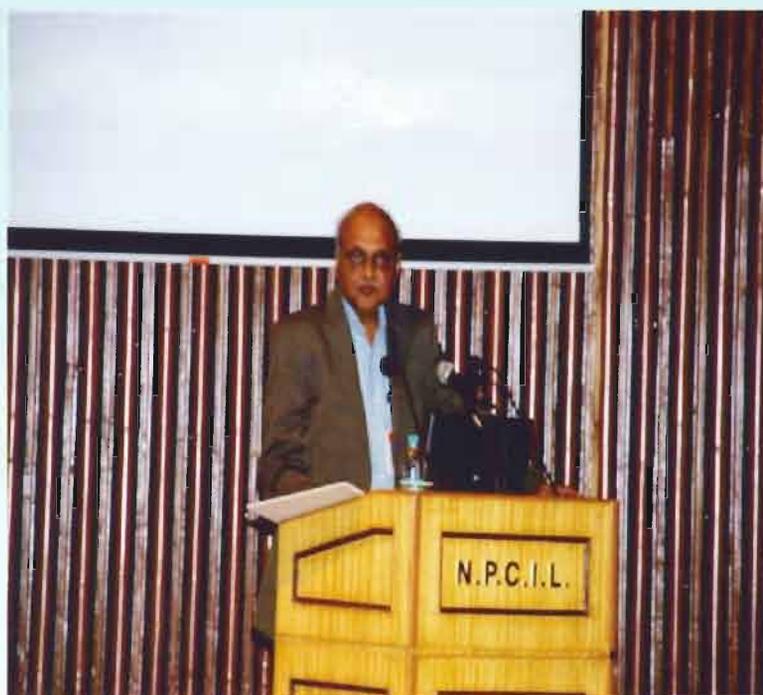
A Dental technician team from BARC hospital presented the topic: "Role of dental technicians in dentistry." The presentation was prepared by Ms. Arachana Dongre, Mr. Anil Kunjan & Mr. Abhay Deokar under the guidance of Dr. M.M. Atre.

The intention of the presentation was to create awareness about the role played by dental technicians to prepare dental prosthesis like removable & fixed appliances.

The programme was organized by Mr. Jayaprakash Tripathi and was inaugurated by Director, Bio-Medical Group Dr. K.B. Sainis and other Honorable invitees including Dr. Tulsi Mukherjee. The seminar was co-ordinated by Head of the Department Dr. M.M. Atre.

The following topics were covered:

Topic	Speaker
1. Periodontal Disease	Dr. Ajay Kakkad
2. Root canal Treatment	Dr. Mona Kakkad
3. Dental Awareness	Dr. M.M. Atre
4. Pediatric Dentistry	Dr. Julli Bajaj
5. Implant Dentistry	Dr. Rajib Ghoshal
6. Dental Laboratory Procedure	Mr. Abhay Deokar



Dr. R.K. Sinha, Director, BARC addressing at the 35th Annual Medical Division Day held on January 12, 2011



Dr. K.B. Sainis, Director, Bio-Medical Group participating in the Walkathon on January 13, 2011

Chief Editor

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