



# **PEDIATRIC SURGERY UPDATE ©**

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### **TPNIC**

TPN induced cholestatic (TPNIC) jaundice is an uncommon conjugated hyperbilirubinemia in infant which carries serious implications. Infectious, metabolic, and structural causes of cholestasis should be rule-out. The incidence of TPNIC is around 30-50% of infants receiving prolonged TPN. Very low birth weight and prematures are more at risk than term infants. No single etiologic factor has been identified, although large amounts of amino acids associated to low amounts of fat in the TPN solution can cause sludge and stone formation in the gallbladder. The most important factor in the hepato-biliary dysfunction is the patient underlying disease state and severity of his illness. TPNIC is a predisposing factor for sepsis in surgical neonates since it leads to impaired non-specific cellular immunity. Cholestasis is associated with intracranial hemorrhage, PDA, sepsis, and GI conditions requiring surgery. This infants will show markedly elevated liver enzymes, no excretion of Tc labeled HIDA and increased GGT levels. Liver histology correlates with the chronologic progression of the disease process and TPN use as follows: by 5 days of TPN you can find steatosis with prominent eosinophils in portal tracts, by 10 days there is canalicular cholestasis, by 21 days bile duct proliferation, by 90 days portal fibrosis and after 5 months micronodular cirrhosis. Phenobarbital which increase bile flow has not been found beneficial in TPNIC. In most cases the degree of dysfunction is self-limiting and reversible upon cessation of TPN.

### **GER and NI**

Feeding disturbances, vomiting, anticonvulsant drug non-compliance, and recurrent chest infections in neurologically impaired (NI) children often request the effort of surgical options to improve growth, nutrition, quality of life and reduce seizure activity. Options are enteral tube feeding, percutaneous endoscopic gastrostomy (PEG), and open gastrostomy with or without antireflux surgery (ARS). Whenever a NI child is referred for feeding gastrostomy a expeditious search for gastroesophageal reflux (GER) is achieved and ARS considered. ARS in NI has a higher rate of complication than in non-NI children (4:1). They include: wrap herniation into chest (entire

or paraesophageal), disruption and recurrent preop GER symptoms (vomiting and pneumonia are the most troublesome). The etiology of NI offers little help in predicting success of ARS. Theories explaining this high rate of failure are: supine position, swallowing incoordination, esophageal dysmotility, spasticity, seizures, delayed gastric emptying, chronic constipation and scoliosis (increase intrabdominal pressure). Not every NI child requires ARS in the presence of GER. Improving nutrition can decrease GER symptoms. PEG can help improve nutrition and serve as a screening tool for those pts. with continuing GER related problems who will eventually need ARS. Ballantine permanent roux-en-y jejunostomy is another alternative in pts. with persistent feeding intolerance after ARS.

### **WT and HA**

In 1967 hyaluronic acid (HA), a glycosaminoglycan is detected in the serum and urine of a patient with a Wilm's Tumor (WT). HA has also been found in children with WT to: cause serum hyperviscosity , suppress the formation of humoral precipitating antibodies to certain major classes of proteins (interfering with elicitation of a complete antibody response), and produce platelet dysfunction presenting as coagulopathy suggesting Acquire von Willebrand disease. The group from UCSF School of Medicine postulated that since the fetal kidney is the source of circulating HA stimulating activity and WT is an embryonal tumor, HA could serve as a WT tumor marker. A cooperative study started in 1990 gathered data for 105 pts proved that: HA urine levels in WT pts are high preop, decrease with tumor resection and persist high with tumor persistence or relapse (APSA 94).

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\*Edited by: **Humberto L. Lugo-Vicente, MD, FACS, FAAP**

P.O. Box 10426, Caparra Heights Station, San Juan, Puerto Rico 00922-0426.

Tel (787)-786-3495 Fax (787)-720-6103

E-mail: titolugo@coqui.net Internet Address:<http://home.coqui.net/titolugo>