A Potencial Experimental Model For the Study of Osteopenia in CCl4 Liver Cirrhotic Rats*

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Abstract of:

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In order to search for an experimental model to further investigate the osteopenia associated to liver cirrhosis (LC), this study has been focused on investigating the occurrence of bone disorders in male rats to which LC histologically confirmed was induced through the validated procedure of CCl_4 inhalation. Length, anteroposterior and lateromedial diameters, densitometry, mechanical stress resistance, hydroxyproline (OHprol) and calcium and phosphate contents were measured in femurs from control (n=10) and liver cirrhosis rats (n=10). It has been found that femurs from liver cirrhosis rats showed a significant reduction ($p < 0.01$) in bone weight (0.254 ± 0.003 vs 0.230 ± 0.004 g/100 g b.w), anteroposterior (4.08 ± 0.06 vs 3.69 ± 0.05 mm) and lateromedial (5.33 ± 0.05 vs 5.08 ± 0.04 mm, $p < 0.05$) diameters, resistance to mechanical stress (405.8 ± 9.5 vs 332.5 ± 9.1 N) and total densitometry (0.416 ± 0.005 vs 0.381 ± 0.004 g/cm^2). However, no significant differences were observed in bone length, calcium, OHprol and phosphate (all expressed as mg/100 mg fresh bone tissue) contents. Therefore, the proteins matrix to mineral contents ratio was not altered. These results indicate that in this model of experimental liver cirrhosis there is osteopenia characterized by bone frailty and reduced thickness, and it could offer an experimental model to stududy bone changes associated to liver cirrhosis.

Avascular Necrosis of the Distal Fibular Epiphysis: A New Condition?

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A 5-year-old boy was seen because of a right ankle sprain; radiographs showed a marginal anterior avulsion of the distal fibular epiphysis. After plaster immobilization, the patient continued with pain and subjective instability. New radiographs showed a lytic area in the same zone of the avulsion with surrounding sclerosis and a detached bone fragment. Biopsy showed bone necrosis. Two years later, without any traumatic event the patient began to have a similar pain in the left ankle. Radiographs showed fragmentation of the anterior portion of the distal fibular epiphysis with surrounding sclerosis. Surgical treatment was perfomed by curettage and cancellous bone grafting; biopsy showed connective tissue, mucoid degeneration, and necrotic bone. After excluding any general disease, we believe that this bone necrosis could be considered an epiphyseal idiopathic avascular necrosis in children. Key Words: Avascular necrosis–Fibula–Osteochondroses.

Outer Membrane Differences between Pathogenic and Environmental *Yersinia enterocolitica* Biogroups Probed with Hydrophobic Permeants and Polycationic Peptides

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Sensitivities to polycationic peptides and EDTA were compared in *Yersinia enterocolitica* pathogenic and environmental biogroups. As shown by changes in permeability to the fluorescent hydrophobic probe *N*-phenylnaphthylamine (NPN), the outer membranes (OMs) of pathogenic and environmental strains grown at 26ºC in standard broth were more resistant to poly-L-lysine, poly-L-ornithine, melittin, cecropin PI, polymyxin B, and EDTA than *Escherichia coli* OMs. At 37ºC, OMs of pathogenic biogroups were resistant to EDTA and polycations and OMs of environmental strains were resistant to EDTA whereas *E. coli* OMs were sensitive to both EDTA and polycations. Similar results were found when testing deoxycholate sensitivity after polycation exposure or when isogenic pairs with or without virulence plasmid pYV were compared. With bacteria grown without Ca++ available, OM permeability to NPN was drastically increased in pathogenic but not in enviromental strains or *E. coli*. Under these conditions, OMs of pYV- cells showed small differences in NPN permeability but differences in polycation sensitivity could not be detected by fluorimetry. O:1,6 (environmental type) lipopolysaccharide (LPS), but not O:3 or O:8 LPS, was markedly rough at 37ºC, and this could explain the differences in polycation sensitivity. LPSs from serotypes O:3 and O:8 grown at 37ºC were more permeable to NPN than O:1,6 LPS, and O:8 LPS was resistant to polycation-induced permeabilization. These data suggest that LPSs relate to some but not all the OM differences described. It is hypothesized that the different OM properties of environmental and pathogenic biogroups reflect the adaptation of the latter biogroups to pathogenicity.

Decreased Anion Exchanger 2 Immunoreactivity in the Liver of Patients With Primary Biliary Cirrhosis

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Chloride-bicarbonate anion exchanger 2 (AE2) is expressed in a variety of tissues, including the liver and salivary glands, where it may participate in the generation of hydroionic fluxes into secretions. We have previously reported decreased hepatic levels of AE2 messenger RNA in patients with primary biliary cirrohosis (PBC), a cholestatic condition frequently associated with pluriglandular exocrine failure. Here we investigated the expression of AE2 protein in the liver of PBC patients. Using a monoclonal antibody against an AE2 peptide, immunohistochemistry was performed on liver biopsy specimens from subjects with normal liver (n=7), patients with PBC (n=13), and patients with cirrhosis or cholestasis other than PBC (n=17 and 11, respectively). Immunostaining was graded from 0 to 7, according to its intensity and distribution. AE2 immunoreactivity was observed in normal livers, as previously reported, and in many pathological liver biopsy specimens, being mainly restricted to canaliculi and the luminal membrane of terminal and interlobular bile ducts. Canalicular and ductular scores were significantly reduced in the PBC group compared with each control group (normal liver and cirrhosis or cholestasis other than PBC), whereas no differences in immunoreactivity scores were observed among control groups. When four patients with primary sclerosing cholangitis (PSC) were analyzed, they also differed from those with PBC. These results suggest that PBC is characterized by diminished expression of AE2 in the liver. Reduced levels of this transporter protein might be involved in the pathogenesis of cholestasis in PBC.