



PEDIATRIC SURGERY Update © **Vol. 31 No. 05 NOVEMBER 2008**

Reperfusion Injury

Hypovolemic shock whether hemorrhagic (trauma) or septic is the leading cause of morbidity and mortality in intensive care units. Reperfusion injury commences after physicians reoxygenate tissue that has undergone ischemic insults caused by hypoxia. The problem starts in the endothelial cells bed, since these cells are very sensitive to hypoxia and manifest damage by increasing cellular volume, loss of cytoskeletal organization, loss of adherence to the basement membrane, decreases in membrane fluidity and adherence of activated leukocytes. The location of this endothelial damage occurs at the level of arterioles, capillaries and venules. Specifically this last (venules) are the most common sites of inflammatory response due to reperfusion causing leukocyte-endothelial cell adhesion, transendothelial migration, platelet-leukocyte aggregation and enhanced oxidant production. Leukocytes use binding proteins to attach themselves to the endothelium and promotes damages liberating oxidative substances. The consumer of oxygen in the cell is the mitochondria.

There is an inability for the mitochondria to use oxygen during reperfusion leaving the cell in a cytopathic hypoxia (inability to produce ATP via oxidative phosphorylation). Endothelium is also damage in reperfusion injury due to oxidative radicals (superoxide and hydrogen peroxide). One of the most dramatic examples of reperfusion injury occurs during the development of necrotizing enterocolitis of babies.

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Mesenteric Vascular Occlusion

Mesenteric vascular occlusion causing infarction of the bowel is a very devastating disease rarely seen in the pediatric age group. Most cases are caused by mesenteric venous thrombosis. In children, mesenteric vascular thrombosis may occur both in idiopathic form or associated with a predisposing disease. Predisposing diseases includes thrombotic

disorders causing a hypercoagulable state, cardiac diseases, diabetes mellitus, vasculitis (polyarteritis nodosa), artificial surfaces, trauma and surgery. Major difficulty is in establishing a prompt diagnosis since symptoms and signs mimic many disorders and bowel necrosis is already present when surgery is performed. Abdominal pain, distension, rigidity and tenderness are usually present. Persistent metabolic acidosis is a warning sign of bowel ischemia. Likewise imaging is nonspecific and can present with air-fluid levels, pneumatosis intestinalis, portal vein air, and thickened bowel loops. Selective SMA angiography is the most reliable diagnostic procedure in suspected cases and thrombolysis using urokinase or streptokinase is an alternative therapy. With failed patent arterial or venous visualization laparotomy is the next step in management.

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Polyarteritis Nodosa

Polyarteritis nodosa (PAN) is an autoimmune mediated necrotizing vasculitis affecting principally medium and small sized arteries which become swollen and damaged from attacks by rogue immune cells. PAN affects principally the skin and kidney, but almost every organ in the body is involved. In both the glomeruli and blood vessels, endothelial injury and subendothelial fibrin deposition are the earliest detectable ultrastructural changes. Boys and girls seem to be equally affected, with a peak at the age of ten years. Clinical symptoms vary depending on site of vascular involvement. Clinically children manifest fever, abdominal pain, vomiting, diarrhoea, weight loss, joint pains and skin rash. For surgeons, PAN can produce acute mesenteric vascular occlusion from venous thrombosis causing bowel ischemia. Unfortunately severe gastrointestinal involvement in PAN is usually fatal despite aggressive therapy. It can also produce acute cholecystitis. Cutaneous PAN can appear in children and has a benign and chronic course. Skin biopsy will make the diagnosis. The diagnosis of polyarteritis nodosa is difficult and often delayed. Management consists of steroids (prednisone) and immunosuppression (cyclophosphamide).

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