

2. Despite extensive spinal cord lesions and infiltration of aorta and complete resolution of lymphoma can be achieved and normal kidney function can be maintained.

EP-150 ACUTE KIDNEY INJURY IN PREMATURE INFANTS WITH NECROTIZING ENTEROCOLITIS

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Introduction: Neonatal acute kidney injury (AKI) is an important contributing factor to morbidity and mortality of critically ill neonates. Prematurity itself is an independent risk factor for AKI as a result of incomplete nephrogenesis and low nephron number. Neonatal disease caused by perinatal hypoxia is also strongly associated with AKI.

Material and methods: We analyze the frequency of acute kidney injury in premature infants with necrotizing enterocolitis (NEC) and gestational age less than 32 weeks who died. The study involved 21 of premature infants with necrotizing enterocolitis and gestational age less than 32 weeks who died. Comparison group - 25 infants who survived with similar stages of NEC. Statistical processing of the data obtained was carried out on a personal computer using STATISTICA 6.1 and IBM SPSS.

Results: The average body weight of children was 1371.2 ± 70.5 g, gestational age 29.0 ± 0.5 weeks of gestation, predominantly boys (66.7%). 12 children were diagnosed with stage III of necrotizing enterocolitis, 9 children - stage II of necrotizing enterocolitis. Acute kidney injury developed in 15 (71.4%) premature infants with NEC who died. The levels of creatinine (114.3 to 426 $\mu\text{mol/l}$) and urea (15.4 to 46.7 mmol/l) in serum of premature infants with NEC who died was significantly increased ($p < 0.01$). The level of cystatin C in the blood serum of children who died was significantly increased (Me 3.45 [2.86; 4.52] ng/ml, $p < 0.05$). Acute kidney damage manifested itself with anuria - in 10 (66.7%) infants, and oliguria developed in 5 infants. In addition, 13 premature infants (61.9%) were confirmed to have an intrauterine infection, 6 (28.6%) premature infants had sepsis, and 18 (85.7%) premature infants had been diagnosed with hypoxic damage of the brain. The odds ratio (OR) of lethal outcome in premature infants with gestational age less than 32 weeks with NEC and development of acute kidney injury (OR = 12,364; 95% CI: 3,415–44,768) and χ^2 Pearson ($\chi^2 = 17,578$, $p < 0.001$).

Conclusions: Acute kidney injury significantly associated with lethal outcome in preterm infants with gestational age less than 32 weeks and necrotizing enterocolitis.

EP-151 NUTRITIONAL MANAGEMENT OF PAEDIATRIC NEPHROTIC SYNDROME

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Introduction: Nephrotic syndrome is a relatively common glomerular disease during childhood, characterized by the clinical triad of proteinuria, hypoalbuminaemia and oedema. Nutritional management is an important part of nephrotic syndromes overall management and its aims are managing signs and symptoms, replacing the nutrients losses through the urine, improving nutritional status and reducing the risk of greater renal damage.

Material and methods: A Pubmed search was conducted up to September 2021. The literature search was performed using the terms

"nephrotic syndrome", "children", "paediatric" and "nutritional management". 23 studies were eligible for the review.

Results: Although high-protein diets might cause glomerular hypertrophy and hyperfiltration and worsen proteinuria, adequate dietary energy and protein intake is of great importance, in order to reduce the risk of protein-energy malnutrition. 100–130 kcal/kg/day and 0.8g of protein/kg/day seem to be adequate. Increase of protein intake by 1 gram for every gram of protein lost through the urine is suggested. Dietary saturated fat must be limited, while emphasis should be given on mono-unsaturated and poly-unsaturated fatty acids. Supplemental administration of cholecalciferol and calcium is suggested in cases of low levels of 25-OH-D3 and/or ionized calcium and/or elevated levels of PTH. In case of anemia, supplemental administration of vitamin B12 and iron is suggested. Supplemental administration of 10 mg/day zinc may reduce nephrotic syndromes relapse rate, whereas supplemental administration of magnesium reduces thrombosis risk. Dietary sodium intake should not exceed 35mg/kg/day, while water consumption should not exceed urine output plus insensible losses. Many clinical studies have found a relationship between food hypersensitivity and nephrotic syndromes clinical manifestations severity, while various mechanisms explaining this relationship have been proposed.

Conclusions: Dietary management of nephrotic syndrome in children should be individualized, due to the great heterogeneity of its clinical manifestations. A dietitian is an important member of the multidisciplinary team that manages children with nephrotic syndrome.

EP-152 MEGACYSTIS IN TWO FEMALE FOETUSES

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Introduction: We are presenting two case histories of female newborns, diagnosed prenatally with a megacystis and hydronephrosis. Foetal megacystis usually indicates a lower urinary tract obstruction (LUTO); in males most commonly due to posterior urethral valves. A LUTO in females is rare, with a urethral stenosis being the leading differential.

Material and methods: Patient A was diagnosed in week 12 of pregnancy and underwent further investigations including a detailed ultrasound scan and genetic analysis. Patient B was found to have a megacystis, hydronephrosis and dilated bowel loops prenatally, no additional investigations were performed. Both patients had a vesico-amniotic shunt placed, despite normal amniotic fluid volumes.

Results: Patient A was born by caesarean section at 37+4 weeks. She was noted to have a sinus urogenitalis, a ventrally displaced anus and a uterus duplex. Renal function was normal, the hydronephrosis resolved following suprapubic catheter insertion. A micturating cysto-urethrogram showed grade II–III vesicoureteral reflux. The infant was diagnosed with an anorectal/urogenital malformation syndrome; a correction operation was performed at the age of 7 months.

Patient B was born by vaginal delivery at 39+4 weeks. On day three, she developed an ileus requiring surgery. An enlarged bladder and bowel malrotation were seen intraoperatively and a stoma was formed. Two further operations were required due to repeated episodes of ileus. Enteral feeding was not tolerated because of severe hypoperistalsis of the gastrointestinal tract. In week five she developed peritonitis, leading to SIRS and ultimately death. Genetic analysis confirmed a diagnosis of megacystis microcolon intestinal hypoperistalsis syndrome (MMIHS).