Prescribing Pattern of Zinc and Antimicrobials in Acute Diarrhea

IAP Task Force on Management of Acute Diarrhea recommends 20mg of elemental zinc to be given to all children with acute diarrhea older then 6 months and 10 mg to all children aged 2 to 6 months for 14 days(1). We studied the pattern of prescriptions of zinc in acute diarrhea and its relationship to the antimicrobial prescribing patterns in a private tertiary care children's hospital. We carried out a retrospective analysis of case records of children aged 1 month-5 years who were admitted for acute watery diarrhea between June 2004 to May 2007, at Kanchi Kamakoti CHILDS Trust Hospital, a private tertiary care hospital for children. These case records were analyzed for prescriptions of zinc, their formulation, dosage and the details of prescriptions of antibiotics. During this period, 1700 children were hospitalized for acute diarrhea out of 37,296 (4.5%) admissions. Of these 1700 children, 120 were aged 2 to 6 months, 728 between 6 months to 1 year and 852 children between 1 and 5 years of age. Of these, 163(9.5%) children had features of severe dehydration and 24 (1.4%) had grade IV protein energy malnutrition (as per IAP Classification). Zinc was prescribed in 1,111(65%) out of 1,700 children. Over the period of 3 years, prescriptions of zinc increased gradually from 51% in 2004 to 75% in 2007. Amongst the various formulations of zinc (syrup, tablet and capsules), syrup formulation was commonly prescribed in 59%. Of the 1,700 children, 712 (41.8%) received antimicrobials. Ceftriaxone was the most commonly used antimicrobial in 25.8% followed by amikacin in 25%. Persistence of fever >38ºC and loose stools for more than 48 hours was noted to be the common parameter for antimicrobial prescriptions. The usage of antimicrobial significantly dropped from 54.9% in 2004 to 28% in 2007, thus suggesting that zinc use might have resulted in reduced usage of antimicrobials in acute diarrhea. However, other variables which could have influenced the decline in antimicrobial use were not studied. Our findings reinforce the need to propagate the use of zinc which might reduce the indiscriminate antimicrobial usage in acute diarrhea.

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Situs Inversus with Autosomal Recessive Polycystic Kidney Disease

Situs inversus totalis and autosomal recessive polycystic kidney disease occur with an incidence of 1/10000 in the general population(1) and 1 in 20,000 live births, respectively. Association of the two conditions is rare; a medline search revealed only two such cases(2,3). Recent studies have revealed ciliary dysfunction as a cause of both conditions(4,5).

A 10-month-old boy (birthweight 1650 g), born of nonconsanguineous parents, was admitted with history of abdominal distension since birth and fever of 10 days duration. The baby was delivered at 34 weeks of gestation by induction of labor due to severe oligohydramnios. The postnatal period was unevent-ful. On examination, he was alert, with stable vital signs except for a blood pressure of 140/90 mm Hg. There were no dysmorphic features or limb anomalies. Abdomen was distended, with bilateral renal masses. Liver was palpable 2 cm

below the left costal margin. Cardiovascular examination suggested the heart to be on the right side. Ophthalmic fundus was normal.

Hemoglobin, total and differential leucocyte count and platelet count were normal. Blood urea was 18 mg/dL, serum creatinine 0.7 mg/dL and alanine aminotransferase 30U/L. Urine culture revealed significant growth of *E.coli*. Ultrasonogram revealed bilateral enlarged kidneys (right 10.3 × 4.8 × 4.8 cm; left: 9.8×4.7×4.9 cm). Kidneys revealed diffuse increase in parenchymal echogenicity and loss of corticomedullary differentiation suggestive of poly-cystic kidney disease. There were a few sparsely distributed micro cysts of 3-6 mm. The calyceal systems and ureters were undilated. Bladder showed normal wall and mucosa. There was no residual urine. A normal liver was imaged on the left side and sonographically looking normal spleen on the right side. No free fluid was noted in the abdomen.

An *X*-ray chest and abdomen confirmed situs inversus with dextrocardia. ECG suggested mirror image dextrocardia. Echocardiogram ruled out structural anomalies of heart. Ultrasonogram of the parents did not reveal renal cysts. The baby was treated with parenteral antibiotics for urinary tract infection and hypertension was controlled with enalapril. A micturating cystourethrogram done later revealed no vesicoureteric reflux.

Autosomal recessive polycystic kidney disease is caused by mutations in the polycystic kidney and hepatic disease 1 (PKHD1) gene. Several proteins that are encoded by genes associated with polycystic kidney disease have been identified in primary cilia in renal tubular epithelia. These findings have suggested that abnormalities in cilia formation and function may play a role in the pathogenesis of PKD. Kif3, a protein on chromosome 5 is required for the maintenance of primary cilia and the loss of cilia in the kidney produces renal cysts(4). Studies in mice with mutation involving disruption of cilia structure (Tg737, Kif3a, and Kif3b) or function (Pkd2) have demonstrated the connection between situs determination and cilia(5). These studies have shown the role of embryonic cilia in establishing left-right axis determination. The present case illustrates the unusual association of two cilia related diseases.

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