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COMMENTARIES

PERITONEAL DIALYSIS IN INFANTS: NEVER LOSE SIGHT OF—AND FROM—ARTERIAL HYPOTENSION!

In this volume of *Peritoneal Dialysis International*, Di Zazzo *et al.* report on 7 children who developed sudden blindness due to bilateral anterior ischemic optic neuropathy (AION) while undergoing chronic peritoneal dialysis (PD) (1). Their cohort adds to 10 pediatric cases previously reported in the literature. Arterial hypotension is the primary causative mechanism leading to AION. The posterior ciliary arteries are particularly vulnerable to decreases in perfusion pressure, leading to ischemic damage of the optic nerve head and peripapillary area (2).

Young infants appear at greatest risk for this complication. Blood pressure physiologically increases by more than 30 mmHg from neonatal to adolescent age (3,4). Pediatric nephrologists are usually well aware of this age dependence and sensibly use age-, sex- and height-specific percentiles to diagnose hypertension in dialyzed children. Much less attention tends to be paid to the lower end of the blood pressure range, which is equally age dependent. Hypotension is defined as a systolic blood pressure lower than 60 mmHg in neonates and lower than 70 mmHg in infants less than 1 year, lower than 70 + (2 × age) mmHg from 1 to 10 years, and lower than 90 mmHg from age 10 years onward (5).

The physiological pressure autoregulation in the terminal arterioles maintains a constant perfusion pressure to central nervous tissues from 60 to 150 mmHg systemic blood pressure (6). With their physiologically lower blood pressure, infants can be assumed to have a reduced autoregulatory reserve in case of acute and chronic dehydration, explaining their increased vulnerability.

Why are infants on chronic PD prone to hypovolemia? Malformations of the kidneys and urinary tract are the most common cause of end-stage renal disease in this age group (7). These disorders are characterized by impaired tubular function as a consequence of renal dysplasia, causing polyuria and sodium depletion even in end-stage renal disease.

Furthermore, the large energy demands due to rapid body growth during infancy require a high fluid intake relative to body size. In oligoanuric infants, this fluid load must be compensated by high dialytic ultrafiltration rates and will lead to large sodium losses. During automated PD, about 80 mmol of sodium are removed per liter of ultrafiltrate (8). Hence, an anuric 5 kg infant with 300 mL daily ultrafiltration will lose almost 5 mmol/kg sodium per day, more than twice the daily urine losses of a healthy child. If the child receives 500 mL standard formula milk per day, sodium intake will only be around 3 – 10 mmol (9). Therefore, nutritional sodium supplementation is mandatory, and intense monitoring of sodium and fluid balance is required in this population (10).

In addition to meticulous attention to salt and fluid balance, care must be given to avoid hypotensive states related to acute illness or antihypertensive medication. Bilateral nephrectomy, frequently performed in infants with polycystic kidney disease or congenital nephrotic syndrome, is another risk factor for chronic hypotension.

Several circumstances may prevent the timely diagnosis of gradually developing dehydration in infants on chronic PD. Whereas hypotonic dehydration is readily diagnosed by hyponatremia, isotonic dehydration may go unnoticed for extended periods of time. Since in infants on automated PD ultrafiltration occurs exclusively at nighttime, dehydration and arterial hypotension may initially be limited to the early morning hours and thus escape detection in the outpatient setting. Therefore, regular measurements of blood pressure and heart rate upon disconnection from the cycler are essential to diagnose subacute dehydration.

The prognosis of AION in children on chronic PD is grim. In a reported series of 14 children, 9 remained permanently blind while 5 regained some visual function (11). In line with the pathophysiology of the condition, immediate aggressive vascular refilling is the only apparently efficacious therapeutic approach: All 5 of the latter patients received fluid bolus therapy, in contrast to only 1 of the 9 children whose blindness persisted. Intravenous steroids, administered to reduce optic nerve inflammation and disc edema, were not effective.

Di Zazzo *et al.* observed a 1% event rate for AION in the Italian Pediatric PD Registry. If this figure is correct and representative of the global pediatric PD population, dozens of young children must turn blind each year, most of them irreversibly, as a consequence of inadequate fluid and blood pressure management. Heightened awareness of the risk

of hypotension on automated PD will be key to prevent this catastrophic complication in the pediatric—as well as in the adult—PD population.

DISCLOSURES

The author has no financial conflicts of interest to declare.

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