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Diagnosis by peritoneal scintigraphy of peritoneal dialysis–associated hydrothorax in an infant

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ACUTE HYDROTHORAX COMPLICATING PERITONEAL DIALYSIS

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♦ *Aim:* **To determine whether gradually increasing the peritoneal dialysate fill volume from 10 to 40 mL/kg over 6 days, rather than commencing at 40 mL/kg, prevents hydrothorax in children and reverses it if present.**
♦ *Methods:* **A review of children peritoneally dialyzed in a single center.**
♦ *Results:* **During the 20 years beginning June 1985, 416 children were peritoneally dialyzed, of which 327 (79%) had acute and 89 had end-stage renal failure. Among 253 children who had gradually increasing fill volumes, none developed acute hydrothoraces, but 13/163 (8%) who began with 40 mL/kg cycles did ($p < 0.000$, Fisher's exact test). These were diagnosed after a median (range) of 48 (6 – 72) hours and were predominantly right sided. Initially, we readily abandoned peritoneal dialysis; 2 were changed to hemodialysis. Subsequently, we found that peritoneal dialysis could be continued by using small volumes with the patients sitting up; cycle volumes were then gradually increased again. One pre-term baby died soon after developing an acute hydrothorax. One patient on chronic peritoneal dialysis developed an acute hydrothorax after forceful vomiting, but recovered after being dialyzed sitting up with low fills.**
♦ *Conclusion:* **Acute hydrothorax can be prevented and treated using graduated cycle volumes, and is not a contraindication for peritoneal dialysis.**

Acute hydrothorax complicates peritoneal dialysis (PD) in about 2% – 4% of adults and children (1–4). It may lead to treatment being compromised or abandoned, either by embarrassing respiration or by limiting ultrafiltration or dialysis efficiency. Suggested treatments include surgery to diaphragmatic communi-

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cations (5), low volume exchanges (4), sometimes with the patient sitting upright (6,7), and pleurodesis with tetracycline (8), talcum powder (9), or autologous blood (10,11). However, the ideal treatment has not been determined. We therefore reviewed our experience of this problem in our pediatric nephrology unit over 20 years to determine a successful management strategy.

PATIENTS AND METHODS

We reviewed the notes from all the children we peritoneally dialyzed in our regional pediatric nephrology unit over 20 years from June 1985. Our catheter insertion and fluid management protocols gradually changed during this time. Initially, catheters were placed percutaneously for acute and surgically for end-stage renal failure, but subsequently we inserted them all surgically, in common with international practice. Throughout, we gradually increased fluid cycle volumes for surgically inserted catheters, starting with continuous 10 mL/kg cycles for 2 days, followed by overnight cycling with 20 and 30 mL/kg for 2 days each, and 40 mL/kg thereafter. Early in the period under study, percutaneously inserted catheters were cycled with 40 mL/kg immediately after placement, but later, following the high incidence of acute hydrothorax, percutaneous catheters were also cycled using the graduated regimen. All dialysis was carried out using automated machines initially with dialysate containing 1.36% glucose solutions, and with initial 45-minute cycles with 30-minute dwell periods.

Acute hydrothorax was diagnosed clinically when children retained dialysis fluid and developed new areas of dullness to chest percussion and reduced air entry, and was always confirmed on chest radiography.

We also report a case of a boy who developed an acute unilateral hydrothorax after 2 years of home PD.

Comparisons of proportions were tested statistically with Fisher's exact test, which remains robust even when some groups are small.

RESULTS

During the 20 years, we peritoneally dialyzed 416 children, of whom 327 (79%) were treated for acute and 89 for end-stage renal failure. During this time, 163 (39%) patients commenced on 40 mL/kg cycles and 253 (including all those with chronic renal failure) had graduated fills. Thirteen children (3% of the total) developed acute hydrothoraces (Table 1) after commencement of dialysis. None of these cases had either pneumonia with effusion or hypoalbuminemia. However, all 13 had been commenced on 40 mL/kg cycles, whereas none of those cycled with the graduated regimen developed a hydrothorax (8.0% vs 0%, $p < 0.000$). The hydrothoraces were either solely or predominantly right-sided in 11 cases, and approximately equal in just 2. Neither sex was more likely to develop an acute hydrothorax. All children with hydrothoraces had their catheters placed in a standard position and were cycled at standard frequency.

Acute hydrothoraces were significantly more common in children with diarrhea-positive hemolytic–uremic syndrome (D+HUS), where 11/211 were affected compared to 2/205 with other diagnoses ($p = 0.02$). However, all 11 children with D+HUS that developed hydrothorax did so while being commenced on 40 mL/kg fills, whereas no child with D+HUS that received graduated fills developed this complication (11/73 vs 0/138, $p < 0.000$).

In the 12 older children, acute hydrothoraces took a median (range) of 48 (14 – 72) hours to become clini-

cally manifest. In 10, we continued to use PD with the patient sitting upright and using about half-volume cycles, which resulted in the volume of the hydrothorax reducing, with complete resolution over several days. In 4 children, renal recovery meant that they were still being dialyzed with reduced cycle volumes when treatment was discontinued. In the other 6, we gradually increased the cycle volume back up to 40 mL/kg, without any recurrence of a hydrothorax, and continued dialyzing at full volume for as long as remained necessary.

In 2 initial cases, we continued PD at full cycle volumes in the supine position, which caused worsening respiratory distress. They had central venous lines inserted for hemodialysis.

A 31-weeks' gestation baby that developed acute renal failure secondary to septicemia on day 6 suffered an acute circulatory collapse, with poor air entry at both lung bases 4 hours after commencing PD. A right-chest drain drained 20 mL of serous fluid. He died 7 hours later despite maximum ventilatory and inotropic support and using only half-volume dialysis cycles. At post-mortem, there was 20 mL of dialysis fluid in each pleural cavity, but no anatomical diaphragmatic defect could be detected.

We also report a 2.7-year-old boy who suddenly developed an acute hydrothorax after having been peritoneally dialyzed overnight since 3 weeks of age. He had undergone a successful fundoplication for persistent vomiting at 2 years of age, and about 6 months later suddenly began to cough forcefully and retch as part of a

TABLE 1
Patients Who Developed an Acute Hydrothorax

Patient	Cause of ARF	Age (years)	Sex	Time to symptoms (hours)	Subsequent treatment	Subsequent cycle volume (mL/kg)	Subsequent PD (days)	Acute hydrothorax Right	Left
1	HUS	7.2	M	12	HD	—	—	++	—
2	HUS	3.0	F	72	PD	22	19	+++	—
3	HUS	3.0	M	24	CAVH	—	—	+++	—
4	HUS	3.0	F	48	PD	24	14	+++	—
5	Septicemia	6 days	M	4	Died	20	7 hours	40 mL	20 mL
6	HUS	7.0	M	14	PD	25	15	+	+
7	Nephrolithotomy	2.0	F	36	Recovered function		0	+++	+
8	HUS	6.5	F	72	PD	18	7	+++	++
9	HUS	3.7	F	72	PD	20	15	+++	—
10	HUS	2.3	M	48	PD	18	11	+++	—
11	HUS	2.0	F	72	PD	22	15	+++	+
12	HUS	1.6	M	64	PD	22	20	++	—
13	HUS	2.1	F	36	PD	9	9	+++	+++

ARF = acute renal failure; HUS = hemolytic–uremic syndrome; HD = hemodialysis; PD = peritoneal dialysis; CAVH = continuous arteriovenous hemodialysis.

viral illness, and managed to vomit despite his fundoplication. He became acutely dyspneic and had a marked reduction in his apparent ultrafiltrate. Clinically, he had a large right pleural effusion, which was confirmed radiologically. Sitting upright through the day reduced his hydrothorax volume and he was dialyzed comfortably the next night on half-volume exchanges. The cycles were increased back to 40 mL/kg over 2 weeks without any recurrence of hydrothorax, and he remained on PD until he received a transplant 2.1 years later.

DISCUSSION

A major factor in the development of an acute hydrothorax with PD appears to be starting to cycle with 40 mL/kg volumes, whereas increasing to this volume from initial 10 mL/kg cycles over 6 days prevents hydrothorax. It is difficult to know precisely what physical forces are acting to cause this typically right-sided (1,6,12) reversible leak. However, we recommend using this graduated approach, except where extremely abnormal biochemistry results demand urgent dialysis clearances. Although this is a single-center observational study rather than a randomized controlled trial, the strength of the association leaves little doubt about its validity.

It is interesting that we have found children D+HUS to be more likely to develop hydrothoraces, as has been described before (4). However, we have also shown that, even among this apparently vulnerable group, this complication can be prevented by using graduated fills.

Clearly, in small pre-term babies, greater caution has to be applied to continuing dialysis. However, outside the neonatal period, an acute hydrothorax should not be a reason to abandon PD. Instead, low-volume exchanges should be tried with the child sat or propped upright. In the 2 children in whom we abandoned peritoneal and changed to hemodialysis, we had not tried this maneuver, which may well have worked. Changing dialysis modality has the disadvantage of requiring another access procedure. In our cases, it was not simply that we were able to manage dialysis with the children sitting up, but we were able to gradually increase the fill volumes back to the normal maximum of 40 mL/kg in those children that required longer periods of treatment. This implies that whatever route the fluid tracked through the diaphragm to reach the pleural space was not a permanent channel, but one that appeared to "reseal" in time. This is important if the possibility of PD in the future is not to be lost inappropriately in children who first present with this complication. It is interesting that others that made similar reductions in the cycle volumes (to a mean of 23 mL/kg) but did not sit the children up, do not describe such good results (4). We did not measure intra-abdominal pressure when sitting the children up, but presume from the clinical improvements we saw that this maneuver must have reduced it.

Our child who developed an acute hydrothorax after 6 months on dialysis, presumably secondary to diaphragmatic crural trauma generated by a severe cough, demonstrates that large acute leaks can suddenly occur later, and that these can subsequently completely resolve.

Many authors have speculated about the underlying anatomical cause of the leak, generally assuming that it represents an "inherent" or congenital defect (which will presumably prevent PD in the future) (5,13,14). Although anatomical factors may contribute to the propensity to leak fluid, our data suggest that such components alone are insufficient to matter clinically and must be anatomically subtle. Despite a careful search at postmortem, we were unable to find an anatomical defect in the diaphragm of our pre-term baby whose acute hydrothorax contributed to his pulmonary compromise and death. Radionuclide studies have limited value in detecting pleuroperitoneal communications (15) and are not required if the hydrothoraces heal spontaneously without recurrence when smaller dialysis volumes are used.

We recommend commencing PD with 10 mL/kg cycles on the majority of occasions that the child's biochemistry allows this, and only increasing the cycle volume to 40 mL/kg over days to try to prevent acute hydrothoraces occurring. If they develop despite that (which we have not seen), we would try dialyzing with small volumes and with the child sitting up before abandoning this modality, except in very small neonates where great caution is required. We do not believe that an acute hydrothorax should be assumed to represent a permanent anatomical problem that might prohibit the use of full-volume effective PD.

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