12. Zondek LH, Zondek T. Horseshoe kid- and associated congenital anoma-Urol Int 1964,18: 347-349.

13. Whitaker RH, Hunt GM. Incidence and distribution of renal anomalies in patients with neural tube defects. Eur Urol 1987,13: 322-325.

14. Bauer SB, Perlmutter AD, Retik AB. Anomalies of upper urinary tract. *In:* Campbell's Urology, 6th edn. Eds. Walsh PC, Retik AB, Stamey TA, Vaughan ED. Philadelphia, W.B. Saunders Company, 1992, pp 1376-1381.

15. Gay BB Jr, Dawes RK, Atkinson GO Jr, Ball TI Jr. Wilms' tumor in horseshoe kidneys: Radiologic diagnosis. Radiology 1983,146: 693-679.

16. Pilotti S, Rilke F, Alasio L, Garbagnati F. The role of fine needle aspiration in the assessment of renal masses. Acta Cytol 1988, 32:1-10.

17. Quijano G, Drut R. Cytologic characteristics of Wilms' tumor in fine needle aspirates. A study of ten cases. Acta Cytol 1989, 33: 263-266.

18. Dey P, Radhika S, Rajwanshi A, *et al.* Aspiration cytology of Wilms' tumor. Acta Cytol 1993, 37: 477-482.

Fungal Peritonitis Complicating Peritoneal Dialysis

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Peritonitis is the most frequent serious complication in patients requiring multiple peritoneal dialysis (PD). The causative organisms are usually bacteria(l). Fungi are uncommonly implicated, .being responsible for only 2-10% of all peritonitis episodes associated with PD(2). The majority of the published experience deals with fungal peritonitis in patients undergoing continuous ambulatory peritoneal dialysis, the incidence being 0.2 to 1.7 episodes per 12 patient months of dialysis(3). Fungal peritonitis, if not detected early and treated appropriately, is associated with increased morbidity (development of peritoneal adhesions and inability to resume PD) and mortality. We describe the case of a 21 month old girl with hemolytic uremic syndrome who needed multiple peritoneal dialysis and developed fungal peritonitis, which was not suspected antemortem.

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Case Report

A 21-months-old girl with dysentery

of 5 days duration presented with altered sensorium and anuria for 24 hours. Examination showed an euhydrated, comatose child with severe pallor, acidotic breathing and a blood pressure of 110/80 mm of Hg. The serial investigations are summarized in *(Table I),* The initial blood counts revealed a hemoglobin of 5.7 g/dl and a platelet count of 1.0 x 10^9/l. The peripheral blood film showed features of a microangiopathic hemolytic anemia. A diagnosis of hemolytic uremic syndrome was made. She remained anuric despite a fluid challenge and low dose dopamine (2 µg/kg/min) infusion. Since investigations showed severe acidosis, azotemia and hyperkalemia, she was taken up for her first peritoneal dialysis 12 hours after admission.

The initial stool culture grew *Salmonella typhimurium* and she was started on parenteral ciprofloxacin. Over the next 20 days she underwent a total of 6 peritoneal dialysis for persistent anuria and azotemia and received two packed red cell transfusions. Though urine output was gradually established and azotemia was corrected, she started deteriorating from the 30th hospital day when she developed abdominal distension and hypothermia. Peritoneal tap was suggestive of peritonitis and the fluid grew *Escherichia coli* Simultaneous blood culture grew *Escherichia coli* and *Streptococcus fecalis.* She received intravenous ceftazidime, metronidazole and supportive measures but there was no improvement. Preterminally she developed extensive bleeding manifestations which culminated in death on her 35th hospital day. A repeat culture of her peritoneal fluid (sample sent on the day of her demise) revealed fungal peritonitis caused by *Torulopsis glabrata.*

Discussion

Peritonitis complicating PD is usually bacterial in origin. Coagulase negative *Staphylococcus* and *Staphylococcus aureus* are the most common organisms, followed by *Streptococcus* and *Enterococcus. Pseudomonas aeuruginosa, Escherichia coli* and *Klebsiella pneumoniae* are responsible for most Gram-negative peritonitis(l). On the other hand, fungal peritonitis in patients receiving PD is rare and the most frequent fungus is *Candida* species(3). *Candida albicans, C. tropicalis* and C. *parapsilosis* account for 90% of all cases of fungal peritonitis. *Torulopsis glabrata (Candida glabrata)* which was grown from the peritoneal fluid in our patient, is*a rare cause of peritonitis in patients undergoing PD, accounting for only 3-5% cases of fungal etiology(3,5).

Fungi enter peritoneal cavity through touch contamination of dialysis tubing or by direct extension of the infection from the catheter exit site. The possibility of fungal peritonitis should be considered in patients undergoing PD who have one or more of the following risk factors(2): *(i)* previous episode of bacterial peritonitis within one month or co-existing bacterial peritonitis; *(ii)* antecedent antibiotic usage in the preceding month; *(iii)* prolonged indwelling catheter; *(iv)* co-existing extraperitoneal site of fungal infection; and *(v)* use of immunosuppressive drugs. In addition, when PD is used as an emergency measure for the management of acute renal failure (ARF), the procedure is associated with an in-

TABLE I—*Serial Investigations in the Subject.*

	Hospital day				
	Day 1	Day 7	Day 20	Day 30	Day 35
Hemoglobin (g/dl)	5.7	8.2	11.0	6.0	8.5
TLC ($\times 10^9$/l)	5.1	6.2	7.0	3.2	2.0
Platelets ($\times 10^9$/l)	1.0	0.2	1.6	0.8	0.2
Peripheral smear	Suggestive Microangio-pathic hemo-lytic anemia	Suggestive Microangio-pathic hemo-lytic anemia	Microcytic anemia	Microcytic anemia	Dimor-phic anemia
S. Na^+ (mmol/l)	136	128	130	120	126
S. K^+ (mmol/l)	6.4	3.3	3.0	2.0	3.8
B. urea (mg/dl)	90	110	60	40	100
S. creatinine (mg/dl)	2.0	2.4	1.2	1.0	1.8
Blood culture					
(*a*) Aerobic	Sterile	ND	ND	*Escherichia coli/Strept faecalis*	Sterile
(*b*) Anaerobic	Sterile	ND	ND	Sterile	Sterile
(*c*) Fungal	ND	ND	ND	ND	Sterile
Peritoneal fluid					
(*a*) Cells/mm^3	ND	20	No cell	1000 (90% polymorphs)	2500 (75% polymor-phs)
(*b*) Gram stain	ND	Negative	Negative	Negative	Negative
(*c*) KOH smear	ND	ND	ND	ND	Negative
(*d*) Bacterial culture	ND	Sterile	Sterile	*Esch.coli*	Sterile
(*e*) Fungal culture	ND	ND	ND	ND	Torulopsis glabrata
Coagulogram	ND	ND	ND	ND	-
(*a*) PTI/PT					42%/75"
(*b*) PTTK					75"(C-32")
(*c*) FDP					ND

ND—Not done

creased risk of fungal peritonitis. In the series of 55 patients reported by Eisenberg, 16 had early fungal peritonitis and all of them had received PD for ARF(4).

Fungal and bacterial peritonitis cannot be differentiated on the characteristics of PD fluid alone. In a series of 9 patients with fungal peritonitis(5), KOH smear was positive in only 2 cases. Therefore, the clinician should be aware of all the factors which increase the risk of fungal peritonitis in patients undergoing PD for ARF, specially when the catheters are placed under suboptimal conditions and the patients are receiving broad spectrum antibiotics. Our patient received multiple peritoneal dialysis for acute renal failure, had *Escherlchia coli* bacterial peritonitis and received broad spectrum antibiotics for a prolonged duration. Thus, she had most of the risk factors for fungal peritonitis listed above. An early suspicion of fungal peritonitis and institution of appropriate treatment could, probably, have prevented her death.

There is no unified strategy for treatment of fungal peritonitis. Nagappan *et al*.(6) managed most of their cases just by early catheter removal. Antifungal therapy was instituted in those cases whose symptoms and signs did not improve within.48 hours after catheter removal or in the cases who were seriously ill. Most authorities(5,7,8) agree that the cathether should be removed and an antifungal agent should be administered. Amphotericin B (0.5-1 mg/kg/day IV) either alone or in combination with flucytosine (30-100 mg/kg/day IV) or oral ketoconazole (4-6 mg/kg/day) for 2-6 weeks has been recommended^). Some authors have advocated insertion of a temporary catheter for peritoneal lavage with antifungal agents(9).

Fungal infections are fast emerging as a major pathogen in our clinical practice. The propositus illustrates the need for surveillance for detection of fungi by culturing cathether tip and peritoneal dialysis effluent in all cases undergoing peritoneal dialysis. This would be especially relevant in those with high risk factors. Early detection of fungal peritonitis would lead to institution of appropriate therapy.

REFERENCES

1. Vas SI. Infections of continuous ambulatory peritoneal dialysis catheters. Infect Dis Clin North Am 1989, 3: 301-328. .v
2. Fergie JE, Fitzwater DS, Einstein P, Leggiadro RJ. Mucor peritonitis associated with acute peritoneal dialysis. Pediatr Infect Dis J1992,11: 498-500.
3. Johnson R, Rmsey PG, Gallhagar N, *et al*. Fungal peritonitis in patients on peritoneal dialysis: incidence, clinical features and prognosis. Am J Nephrol 1985, 5:169-175.
4. Eisenberg ES, Leviton L, Soeiro R. Fungal peritonitis in patients receiving peritoneal dialysis: Experience with 11 patients and review of literature. Rev Infect Dis 1986, 8: 309-321.
5. Enriquez JL, Kalia A, Travis LB. Fungal peritonitis in children on peritoneal dialysis. J Pediatr 1990, 117: 830-832.
6. Nagappan R, Collins JF, Lee WT. Fungal peritonitis in continuous ambulatory peritoneal dialysis—The Auckland

Experience. Am J Kidney Dis 1992, 20: 492-496.

?. Oh SH, Conley SB, Rose GM, *et al.* Fungal peritonitis in children undergoing peritoneal dialysis. Pediatr Infect Dis J 1985, 4: 62-66.

8. Agarv/al S. Goodman NL, Malluche HH. Peritonitis due to *Exophila jeanselmei* in a patient undergoing continuous ambulatory peritoneal dialysis. Am J Kidney Dis 1993, 21: 673-675.

9. Rodriguez-Perez JD. Fungal peritonitis in CAPD: which treatment is best? Contrib Nephrol 1987, 57:114-121.

Unusual Complications of Rickets

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Hypocalcemia occurs when the homeostatic mechanisms responsible for the maintenance of physiological serum concentrations of ionized calcium fail. Hypocalcemia whether transient, acute or chronic, should be regarded as a sign only; therefore, an underlying patho-physiology should be sought at the same time that the treatment is initiated for all but the mildest symptoms(l).

We describe an interesting case of rickets associated hypocalcemia manifesting as seizures and later high dose Vitamin D therapy to the child leading to coma and cardiac tetany (hungry bone phenomenon)(2). The case is presented because after extensive search of literature and to the best of our knowledge a similar presentation could not be found.

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Case Report

A one-year-old female Muslim child weighing 8 kg presented with fever, cough and respiratory distress since 8 days with 6 episodes of convulsions of generalized tonic-clonic type since one day. Between convulsions, the child was irritable without impairment of consciousness. There was a past history of six episodes of generalized tonic-clonic convulsions not associated with fever since the age of seven months, the frequency of which had increased since the last one month, in which the child had four episodes. The child was receiving anticonvulsant carbamazepine in inadequate dose with no response. The developmental and natal history was normal; however, the child had been exclusively breastfed with no weaning. On examination, the child had evidence of anemia and rickets and the neurological examination was normal.