DOI: 10.1111/sdi.12740

REVIEW ARTICLE

WILEY Seminars in Dialysis

When the color of peritoneal dialysis effluent can be used as a diagnostic tool

Thomas Dossin[1,2] | Eric Goffin[2]

[1]Department of Nephrology, Centre Hospitalier Universitaire Lapeyronie, Montpellier, France

[2]Department of Nephrology, Université catholique de Louvain, Cliniques Universitaires Saint Luc, Brussels, Belgium

Correspondence
Eric Goffin, Department of Nephrology, Université catholique de Louvain, Cliniques Universitaires St. Luc, Brussels, Belgium.
Email: eric.goffin@uclouvain.be

Abstract

Peritoneal dialysis (PD) effluent is normally transparent. A change in its appearance may be the first indication of an intra- or extraperitoneal abnormality which may or may not be related to the peritoneal dialysis technique itself. What diagnosis should be considered when PD effluent turns on red, orange, cloudy, milky white, green, yellow, purple or black in color? After review of the literature, we propose a differential diagnosis, as well as some management recommendations, for specific abnormal color presentations of the PD effluent.

KEYWORDS
bloody effluent, chyloperitoneum, peritoneal dialysis, peritonitis, rhabdomyolysis

1 | INTRODUCTION

Clinicians who take care of patients on peritoneal dialysis (PD) are often confronted with issues regarding the PD effluent aspect. An unexpected appearance of the effluent usually indicates a complication of the technique and requires quick and accurate management. This can also reveal a problem not directly related to the dialysis technique itself but to another pathological situation that would be unrecognized in patients who are not undergoing peritoneal dialysis exchanges. PD can, thus, be considered as an open window on the abdominal cavity, bringing some additional clues in diagnostic investigations.

After having reviewed the cases reported in the literature, we have developed a list of diagnoses based on the color of the PD effluent, and we here propose a practical approach in the exploration and management of these particular situations.

2 | RED DIALYSATE

Red dialysate usually indicates the presence of red blood cells in the PD effluent (hemoperitoneum), a relatively frequent problem.[1] A quite small amount of blood is enough to change the color of the effluent fluid to a homogenous red (Figure 1). These red blood cells originate from either the peritoneal membrane, from the intraperitoneal organs (organs that are fully encapsulated by the visceral peritoneal membrane), or, more surprisingly, from partially or completely extraperitoneal structures.

The first diagnosis in premenopausal women is blood shedding from the uterine cavity (menstruation) or from one of the ovaries (ovulation).[2] To avoid unnecessary stress, the possibility of these physiological phenomena should be explained to all at risk patients at the beginning of PD.

Once this situation has been excluded, a panoply of situations has to be considered (Table 1). Briefly, hemoperitoneum in PD patients have been reported in situations such as traumatic/mechanical phenomena (postcatheter insertion or manipulation,[1] splenic laceration,[3,4] liver rupture and liver or renal cyst rupture,[5-7] erosion of mesenteric vessel by the Tenckhoff catheter[8] itself, postcolonoscopy,[9,10] postlithotripsy[11]), leakage from an extraperitoneal hematoma,[12-15] infectious/inflammatory diseases (pancreatitis,[16] gangrenous cholecystitis,[11] pseudomembranous colitis,[11] postpelvic irradiation,[17] intra-abdominal connective tissue pouch[11]), bleeding from malignant tumor (renal,[18] gut,[11] liver[19] cancer). Hemoperitoneum can also be seen in patients with coagulation, aggregation or microcirculation issues without any demonstrated focal bleeding (anticoagulant use or low platelets count,[1] abnormal platelet aggregation or adhesiveness,[5] such as in uremic bleeding[5] or scurvy[20]). Recurrent hemoperitoneum in long-term PD patients should raise the possibility of encapsulating peritoneal sclerosis.[21] We also found one case of hemoperitoneum in a PD patient secondary to a ruptured abdominal aortic aneurysm.[22]

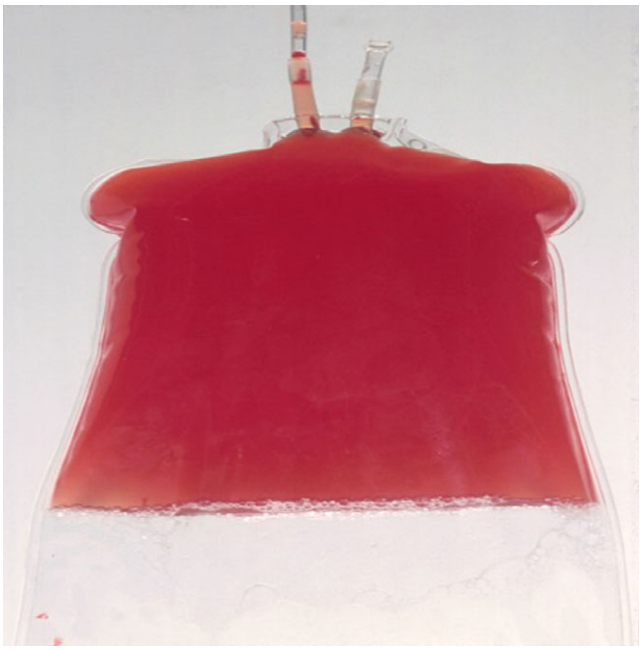

FIGURE 1 Red dialysate. A 38-year-old woman with end-stage renal failure secondary to lupus nephritis on peritoneal dialysis for 4 mo presented with red dialysate effluent on the first morning of her months (hematocrit : 0.3%). Cooling of the dialysate subsequently normalized the effluent aspect

Hemoperitoneum is usually a benign condition, but if bleeding is severe (decreased serum hemoglobin level, dialysate hematocrit > 2%), recurrent or associated with abdominal pain or general symptoms such as fever or altered general condition, investigations must be started with prompt medical or surgical management. Usually physical examination, blood tests, and abdominal imaging (ultrasound or CT scan) are sufficient to make a clear diagnostic and/or rule out severe situations. Gynecologic investigation may be proposed in second-line investigation (in particular to exclude ovarian tumor or endometriosis).

Hemoperitoneum, even recurrent, can sometimes remain without any explanation despite extensive investigations. This situation is not uncommon and does not seem to affect the survival of the dialysis technique.[2]

In addition to the etiological treatment, management is based on the use of rapid flushes and/or use of cooled dialysate.[23] It has also been proposed that unfractionated heparin be added to the dialysate bag (500 UI per liter) to avoid fibrin clot formation and catheter obstruction. Heparin is not absorbed, so it poses no systemic risk. Whether there is a local risk of exacerbating hemoperitoneum is unclear and probably depends on the exact source of bleeding.

3 | ORANGE DIALYSATE

Rifampicin therapy may stain the PD effluent red-orange in addition to discoloring urine, sweat, stool, saliva, and tears (Figure 2). A rusty

TABLE 1 Some causes of hemoperitoneum

Blood origin	Diagnostic considerations
Gynecologic	• Menstruation, ovulation • Ruptured ovarian cyst • Ovarian tumor • Endometriosis
Urologic	• Kidney cysts rupture • Renal angiomyolipoma • Renal cancer
Digestive system	• Postcolonoscopy • Pseudomembranous colitis • Colon cancer • Pancreatitis • Gangrenous cholecystitis • Liver rupture and liver cyst rupture • Hepatoma or hepatic metastasis of solid cancer
Peritoneal membrane	• Sclerosing peritonitis • Peritoneal calcification • Radiation-induced peritoneal injury • After DP interruption
Traumatic	• Postcatheter insertion or manipulation. • Catheter-induced spleen injury • Catheter-induced mesenteric vessel erosion
Miscellaneous	• Anticoagulation therapy • Severe thrombopenia • Uremic bleeding • Scurvy • Leakage from an extraperitoneal hematoma • Ruptured abdominal aortic aneurysm

FIGURE 2 Orange dialysate. Effluent of a 62-year-old man with end-stage renal failure secondary to a diabetic nephropathy on PD for 14 months with persisting dialysate enterococcus infection on rifampicin therapy

aspect of the dialysate effluent has also been described under combination of intravenous iron dextran and rifampicin treatment.[24]

4 | CLOUDY DIALYSATE

Cloudy dialysate in a PD patient most often reflects an increased number of polymorphonuclear neutrophils (PMN) in the effluent fluid, and the first diagnosis to be considered is certainly acute peritonitis (Figure 3). A cell count and a dialysate culture should be performed urgently. ISPD guidelines recommend that PD patients presenting with cloudy effluent be presumed to have bacterial peritonitis and treated as such until the diagnosis can be confirmed or excluded.[25]

If bacterial cultures remain negative despite an increased number of PMN, atypical infections (mycobacteria, fungi, or parasites) have to be considered and adequate cultures have to be performed.[26,27] As reported by Rocklin and Teitelbaum,[26] excessive PMN in PD fluid have also been described in abdominal problems inducing reactive inflammation, such as splenic infarction,[4] ischemic colitis,[28] renal cell cancer,[29] pancreatitis, cholecystitis, appendicitis, hernia, small bowel incarceration,[30] and juxta peritoneal abscess.[31] Abrupt diffuse abdominal pain, excessive PMN in dialysate effluent, and quick favorable evolution without antibiotic treatment are suggestive of an intraperitoneal sterile abscess rupture, usually a remnant of an old peritonitis.[32]

In 1977, two Canadian hospitals experienced an epidemic of 48 episodes of aseptic neutrophilic peritonitis which was found to be secondary to endotoxin contamination of the peritoneal dialysis fluid.[33] A similar story happened again 20 years later in Pennsylvania.[34] This led the authorities to reinforce the sterility requirements for dialysate bags; since then, no other case has been described.

In 2002, a new epidemic of aseptic peritonitis (associated with elevated intraperitoneal monocytes and macrophages) occurred[35] and was subsequently ascribed to a contamination of some icodextrin-solution batches[36] with large amount of peptidoglycans. Again, a reinforcement of quality controls had to be implemented.

Several cases of neutrophilic peritonitis have been described after intraperitoneal infusion of vancomycin[37] and seem to be related to the formulation of the medication.[38] A cloudy appearance of the dialysate can also be related to accumulation of other components, such as eosinophils, malignant cells, or triglycerides (Table 2).

Eosinophilic peritonitis is defined as an absolute eosinophil count of >30/mm^3 in the dialysis fluid.[39] When it is abundant, peritoneal fluid eosinophilia can induce a turbid appearance of the peritoneal fluid, more often found on the nocturnal exchange when CAPD is utilized. Idiopathic eosinophilic peritonitis is usually encountered in the first few months of peritoneal dialysis and is considered a hypersensitivity reaction to certain dialysis system components (catheter or solution),[40] or to air trapped intraperitoneally following catheter placement. It is generally asymptomatic and with a spontaneously favorable outcome over a few days.

Peritoneal fluid eosinophilia can also be related to infection (fungal,[41,42] parasitic,[43] tubercular[44]), and appropriate tests must be performed to rule out these diagnoses in patients at risk. An allergic mechanism has been advanced to explain some cases of eosinophilic peritonitis following intraperitoneal injection of vancomycin[45,46] and streptokinase.[47]

A turbid appearance of the dialysate can also be the consequence of an accumulation of neoplastic or abnormal cells. Such situations have been described in posttransplant lymphoproliferative disorder,[48] Hodgkin's[49] and non-Hodgkin's[50,51] lymphoma, endometrial or prostatic adenocarcinomas.[52] A recurrent turbid appearance of the dialysate, with negative cultures, must prompt consideration of cytological examination to rule out oncological or hematological processes.

Some level of intraperitoneal fibrin formation secondary to persistent low-grade serositis is observed among all PD patients.[53] In some situations such as at dialysis initiation, infectious peritonitis, intraperitoneal bleeding, and encapsulating peritoneal sclerosis,[54] fibrin production may sharply increase. Fibrin overproduction can, by itself, induce a cloudy appearance of the effluent bag, where fibrin filament or fibrin clots can be seen.[55] The main risk of fibrin overproduction is catheter obstruction. Intraperitoneal injection of unfractionated heparin (500 IU/L of dialysate) can prevent intraperitoneal fibrin formation without significant systemic anticoagulant effect[56] and is recommended by some nephrology centers as long as the dialysate appears cloudy.[57]

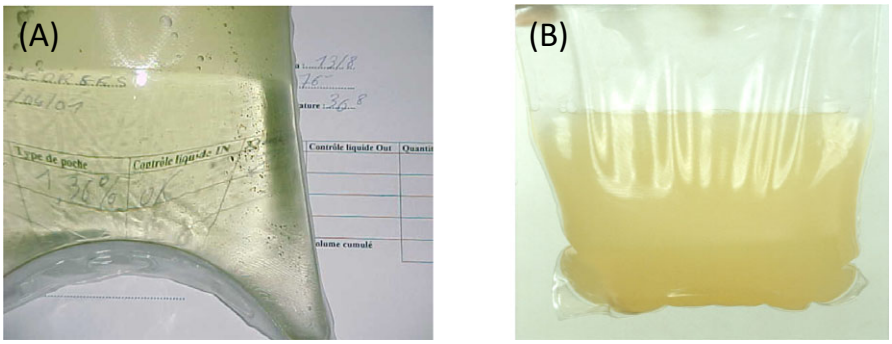

FIGURE 3 Cloudy dialysate. A, Normal CAPD effluent. It is clear enough to allow a text to be read through the dialysate bag. B, Effluent of 47-year-old man with end-stage renal failure secondary to Alport's syndrome admitted with acute peritonitis (white cell count : 7860/μL; 93% of polymorphonuclear neutrophils)

TABLE 2 Causes of cloudy dialysate

Coloring agent	Mechanism	Diagnostic considerations
Polymorphonuclear neutrophils	Fluid infection	• Bacterial • Mycobacterial • Fungal • Parasitic
	Reactional inflammation (negative cultures)	• Splenic infarction • Ischemic colitis • Renal cell cancer • Pancreatitis • Cholecystitis • Abdominal hernia • Small bowel incarceration • Juxta peritoneal abscess • Rupture of an intraperitoneal sterile abscess • Endotoxin contamination of dialysate solution
Eosinophils	"Idiopathic"	• Reaction to the dialysis system constituent (catheter or solution) • Intra-abdominal air after catheter placement • Mechanical irritant effect of repeated significant changes in tension of the peritoneal membrane due to dialysate infusion
	Infection	• Fungal infection • Parasitic infection • Tubercular infection
	Drug induced	• Intraperitoneal injection of vancomycin • Intraperitoneal injection of streptokinase
Neoplastic cells	Hematologic malignancies or solid tumor	• Posttransplantation lymphoproliferative disorder • Hodgkin's lymphoma • Non-Hodgkin's lymphoma • Endometrial adenocarcinoma • Prostatic adenocarcinoma
Fibrin	Serositis	• Spontaneous • At the initiation of PD • Reactional (postperitonitis or posthemoperitoneum) • Encapsulating peritoneal sclerosis

5 | MILKY WHITE DIALYSATE

A milky white appearance of the dialysate suggests a diagnostic of chyloperitoneum, a situation in which triglycerides and chylomicrons accumulate in the peritoneal cavity[58] (Figure 4). After a fatty meal, long-chain triglycerides are incorporated into chylomicrons within enterocytes of the small intestine. Chylomicrons are then secreted via the basolateral membrane to join the lymphatic network and, via the cisterna chyli and the thoracic duct, the central venous system.

Accumulation of chylomicrons and triglycerides in peritoneal cavity occurs whenever there is an interruption of the lymphatic drainage from the gut to the main lymphatic trunks and venous circulation. A milky white appearance of the effluent may, thus, occur after a very high-fat meal as it induces a significant increase in the lymphatic flow.[59] The diagnosis of chyloperitoneum is based on a triglyceride concentration above 110 mg/dL (1.24 mmol/L) in undiluted peritoneal fluid[58] which is a higher level than in plasma.

Multiple etiologies of chyloperitoneum have been reported (Tables 3 and 4). In adults, an obstruction of the lymphatic system mediated by abnormal cell proliferation should rapidly be ruled out. The most common cause is lymphoma, but lymph node extension of cancer from abdominal or pelvic organ[60] and lymphangioleiomyomatosis (sporadic or associated to tuberous sclerosis complex)[61] are also possible.

Infectious diseases such as tuberculosis[62] or lymphatic filariasis[63] are possible causes of lymphatic obstruction, and these diagnoses should be considered if the clinical context is appropriate. Traumatic causes including blunt or penetrating trauma and abdominal (including dialysis catheter placement[64]) or thoracic surgeries can induce lymphatic system damage and secondary chyloperitoneum.

Chyloperitoneum may occur in PD patients with congestive heart failure,[65] constrictive pericarditis,[66] and superior vena cava syndrome.[67] It is best explained by an increase in lymph formation secondary to an excessive pressure in venous capillaries and less effective drainage to the left subclavian vein. Increased lymph production is also the mechanism suggested to explain the rare cases of chylous ascites in cirrhotic patients.[68]

The use of certain dihydropyridine calcium channel blocker (manidipine, benidipine, nisoldipine, nifedipine, and lercanidipine),[69,70] a nondihydropyridine calcium channel blocker (diltiazem),[71] and a direct renin inhibitor (aliskiren)[72] have been associated with chyloperitoneum in CAPD. The mechanism remains unclear.

Chylous ascites has also been described in other situations such as pancreatitis,[73] systemic lupus with mesenteric involvement,[74] nephrotic syndrome,[75,76] sarcoidosis,[77] retroperitoneal fibrosis,[78] and Whipple's disease[79] with pathophysiological mechanisms variably understood. In children, congenital lymphatic abnormalities, obstructive intestinal lesions, lymphangioma, abdominal surgery, and trauma (battered child syndrome) have been described as causes of chyloperitoneum.[80]

The management of chyloperitoneum is based on treating its cause, when possible, bowel rest and parenteral nutrition or, more frequently, enteral nutrition without long-chain triglycerides; medium-chain triglycerides are directly absorbed into the portal system from the gut, bypassing the lymphatic system. PD cessation could accelerate the healing of chylous leak and should be considered when residual renal function or the availability of HD allows for it. Treatment with octreotide seems effective and without any serious adverse events.[81] It blocks somatostatin receptors in the intestine and decreases intestinal fat absorption, intestinal blood flow, and lymph secretion.

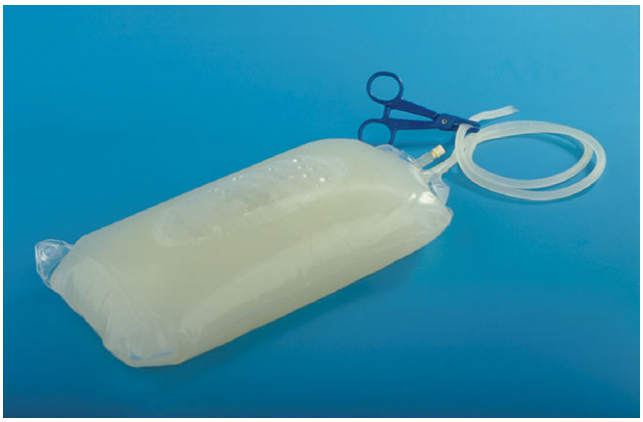

FIGURE 4 Milky dialysate. Overnight effluent of a 46-year-old man with end-stage renal failure secondary to ADPKD on PD for 6 months presenting with milky dialysate after an evening foie-gras rich meal. Subsequent exchanges gradually cleared and thoracoabdominal CT disclosed no abnormality

TABLE 3 Causes of chyloperitoneum

Mechanism	Diagnostic considerations
Obstruction by cell proliferation	• Lymphoma • Lymph node extension of a cancer of an abdominal or a pelvic organ • Lymphangioleiomyomatosis
Obstruction by infections	• Tuberculosis • Lymphatic filariasis
Trauma	• Blunt or penetrating abdominal trauma • Abdominal surgery (including dialysis catheter placement) • Thoracic surgery (thoracic duct trauma)
Excessive lymph production +/− less effective venous drainage	• Congestive heart failure • Constrictive pericarditis • Superior vena cava syndrome • Cirrhosis
Drugs	• Manidipine, benidipine, nisoldipine, nifedipine, lercanidipine • Diltiazem • Aliskiren
Miscellaneous	• Pancreatitis • Lupus disease with mesenteric involvement • Nephrotic syndrome • Sarcoidosis • Retroperitoneal fibrosis • Whipple's disease • Children: congenital anomalies of the lymphatic system, obstructive intestinal lesions, lymphangioma, battered child syndrome

6 | PEACH DIALYSATE

A peach-colored dialysis effluent was reported in the first drain of an urgent-start peritoneal dialysis in a 70-year-old man with diffuse large B-cell lymphoma and tumor lysis syndrome. After analysis, it was shown that this color was the consequence of the combination of chylous ascites (secondary to the lymphoma) and hemoperitoneum (secondary to recent catheter placement and severe thrombocytopenia).[82]

TABLE 4 Other dialysate colors

Color	Diagnostics considerations
Peach	Combinations of chylous ascite and hemoperitoneum
Yellow- Green	Perforated/gangrenous cholecystitis
Brown-black	Hemorrhagic pancreatitis (methemalbumin) Rhabdomyolysis (metmyoglobin)
Fluorescent	Fluorescein angiography
Purple	Interaction between iodine and a particular icodextrin (not present on the market anymore)

7 | YELLOW—GREEN DIALYSATE

A yellow-greenish appearance of the dialysate must suggest the presence of bile in the effluent bag and thus the presence of a communication between the biliary tree and the abdominal cavity (Figure 5). It can be the first and sometimes only symptom of a perforated cholecystitis.[83] Bile leakage can be confirmed by hepatobiliary scintigraphy which shows progressive diffusion of the radiotracer into the dialysate fluid.[84] Surgical management is imperative to avoid infection.

8 | YELLOW—FLUORESCENT DIALYSATE

Fluorescein eye angiography as part of a diabetic retinopathy screening may lead to a yellow-light effluent bag. In the darkness, it emanates a soft fluorescent color (Figure 6).

9 | BROWN-BLACK DIALYSATE

Methemalbumin, an albumin and heme complex, is an indicator of hemolysis. Its accumulation in the dialysate gives it a brown-black color. This has been described in some cases of hemorrhagic pancreatitis where proteolytic pancreatic enzymes cause the destruction of red blood cells. Free heme combines with albumin to form methemalbumin.[85] A similar phenomenon occurs when nonhemorrhagic pancreatitis coexists with intraperitoneal bleeding of other origin.[86]

A brown appearance of the dialysate has also been reported in a patient who has suffered from severe rhabdomyolysis. Peritoneal fluid analysis shows a high concentration of myoglobin and a small number of red blood cells. The brown color persists homogeneously after centrifugation.[87] One hypothesis to explain this particular color is the local formation of metmyoglobin, the oxidized form of the

FIGURE 5 Yellow-green dialysate. Effluent of a 78-year-old man with end-stage renal failure secondary to prostatic hypertrophy on peritoneal dialysis for 46 months presenting with acute cholecystitis

FIGURE 6 Yellow-fluorescent dialysate. Effluent of a 36-year-old male with end-stage renal failure secondary to malignant hypertension one hour after a fluorescein angiography. Note the green-fluorescent color of the dialysate in the darkness

hemeprotein myoglobin. This peculiar aspect of the effluent has been called "coca-cola" or "soy-bean" dialysate[88,89] (Figure 7).

10 | PURPLE DIALYSATE

Between 2001 and 2011, a transient purple discoloration was noted by several patients and clinicians in Europe of the very early

FIGURE 7 Brown-black dialysate. Effluent of a 41-year-old woman with end-stage renal failure secondary to lupus nephritis on peritoneal dialysis for 36 months presenting with acute rhabdomyolysis secondary to drug interactions

drainage phase of a 7.5% icodextrin solution, a starch-derived glucose polymer.[90,91] After careful investigation, it turned out that this phenomenon was secondary to a chemical reaction between the iodine present in the disconnect cap and an icodextrin solution manufactured in Turkey which differed from that produced by other manufacturers by its greater fraction of large polymers.[92] No clinical significance was noted by the authors. Since 2011, this particular icodextrin has not been available.

11 | CONCLUSION

The appearance of PD effluent is a useful and accessible monitoring parameter. It can offer early information about dialysis complications as well as about abdominal or general diseases unrelated to the dialysis technique.

The color of the fluid is by itself a first step toward a diagnosis, which can very often be confirmed by a targeted interrogation, clinical examination and laboratory examination of effluent (cytologic, biochemical, cultures). In some situations, additional examinations such as imaging will be required. Discolored PD effluent can present dramatically and raise urgent questions regarding its source. This review will, we hope, offer answers to such questions.

REFERENCES

1. Greenberg A, Bernardini J, Piraino BM, Johnston JR, Perlmutter JA. Hemoperitoneum complicating chronic peritoneal dialysis: single-center experience and literature review. *Am J Kidney Dis*. 1992;19:252-256.

2. Tse KC, Yip PS, Lam MF, et al. Recurrent hemoperitoneum complicating continuous ambulatory peritoneal dialysis. *Perit Dial Int*. 2002;22:488-491.
3. Van der Niepen P, Sennesael JJ, Verbeelen DL. Massive hemoperitoneum due to spleen injury by a dislocated Tenckhoff catheter. *Perit Dial Int*. 1994;14:90-91.
4. Syed A, Holley JL, Piraino B. Splenic infarct presenting as sterile peritonitis with peripheral embolic phenomena. *Adv Perit Dial*. 1993;9:202-205.
5. Lew SQ. Hemoperitoneum: bloody peritoneal dialysate in ESRD patients receiving peritoneal dialysis. *Perit Dial Int*. 2007;27:226-233.
6. Borras M, Valdivielso JM, Egido R, Vicente de Vera P, Bordalba JR, Fernandez E. Haemoperitoneum caused by bilateral renal cyst rupture in an ACKD peritoneal dialysis patient. *Nephrol Dial Transplant*. 2006;21:789-791.
7. Hammami M, Guirat A, Ksibi H, Azzaza M, Rekik N, Beyrouti MI. Intraperitoneal rupture of renal cyst in autosomal dominant polycystic kidney disease. *N Am J Med Sci*. 2010;2:238-240.
8. Miller R, Denman R, Saltissi D, Healy H, Muller M, Fleming S. Erosion of a mesenteric vessel by a Tenckhoff catheter. *Perit Dial Int*. 1996;16:528-529.
9. Nace GS, George AL, Stone WJ. Hemoperitoneum: a red flag in CAPD. *Perit Dial Int*. 1985;5:42-44.
10. Luni FK, Bawany MZ, Khan AR, Nawras A, Vetteth S. Haemoperitoneum post colonoscopy in a continuous ambulatory peritoneal dialysis patient. *Arab J Gastroenterol*. 2014;15:159-160.
11. Bargman JM. Noninfectious Complications of Peritoneal Dialysis. In: Khanna R, Krediet RT, Nolph KD, eds. *Nolph and Gokal's Textbook of Peritoneal Dialysis*, 3rd ed. New York, NY: Springer; 2009:571-609.
12. Fernandez GF, Hermosilla SF, Paralle AM, Gonzalez MJ. Hemoperitoneum in peritoneal dialysis secondary to retroperitoneal hematoma. *Perit Dial Int*. 1996;16:644.
13. Campisi S, Cavatorta F, De LE. Iliopsoas spontaneous hematoma: an unusual cause of hemoperitoneum in CAPD patients. *Perit Dial Int*. 1992;12:78.
14. Lew SQ. Persistent hemoperitoneum in a pregnant patient receiving peritoneal dialysis. *Perit Dial Int*. 2006;26:108-110.
15. Balafa O, Koundouris S, Mitsis M, Siamopoulos KC. An unusual case of hemoperitoneum: spontaneous rectus sheath hematoma. *Perit Dial Int*. 2014;34:134-135.
16. Pannekeet MM, Krediet RT, Boeschoten EW, Arisz L. Acute pancreatitis during CAPD in The Netherlands. *Nephrol Dial Transplant*. 1993;8:1376-1381.
17. Hassell LH, Moore J Jr, Conklin JJ. Hemoperitoneum during continuous ambulatory peritoneal dialysis: a possible complication of radiation induced peritoneal injury. *Clin Nephrol*. 1984;21:241-243.
18. Rinaldi S, Sera F, Verrina E, et al. The Italian registry of pediatric chronic peritoneal dialysis: a ten-year experience with chronic peritoneal dialysis catheters. *Perit Dial Int*. 1998;18:71-74.
19. Fine A, Novak C. Hemoperitoneum due to carcinomatosis in the liver of a CAPD patient. *Perit Dial Int*. 1996;16:181-183.
20. Kittisakmontri K, Swangtrakul N, Padungmaneesub W, Charoenkwan P. Gingival bleeding and bloody dialysate: a case report of scurvy in a child with end-stage renal disease receiving peritoneal dialysis. *J Ren Nutr*. 2016;26:407-411.
21. Gernone G, Pepe V, Giannattasio M. Hemoperitoneum after dropout from peritoneal dialysis. *G Ital Nefrol*. 2014;31. pii: gin/31.1.5. [Article in Italian]
22. Yap DY, Yip TP, Lui SL, Lo WK. Ruptured abdominal aortic aneurysm as a cause of spontaneous hemoperitoneum in a patient on peritoneal dialysis. *Perit Dial Int*. 2011;31:600-602.
23. Goodkin DA, Benning MG. An outpatient maneuver to treat bloody effluent during continuous ambulatory peritoneal dialysis (CAPD). *Perit Dial Int*. 1990;10:227-229.
24. Carter TB, Garris AG, Ullian ME. Rusty peritoneal dialysis fluid after intravenous administration of iron dextran. *Am J Kidney Dis*. 1996;27:147-150.
25. Li PK, Szeto CC, Piraino B, et al. ISPD peritonitis recommendations: 2016 update on prevention and treatment. *Perit Dial Int*. 2016;36:481-508.
26. Rocklin MA, Teitelbaum I. Noninfectious causes of cloudy peritoneal dialysate. *Semin Dial*. 2001;14:37-40.
27. Waness A, Al SS. Tuberculous peritonitis associated with peritoneal dialysis. *Saudi J Kidney Dis Transpl*. 2012;23:44-47.
28. Prischl FC, Wallner M, Schauer W, Balon R, Kramar R. An important differential diagnosis in CAPD patients with sudden onset of fever, vomiting, abdominal pain, and cloudy dialysate. *Perit Dial Int*. 1999;19:81-84.
29. Streather CP, Carr P, Barton IK. Carcinoma of the kidney presenting as sterile peritonitis in a patient on continuous ambulatory peritoneal dialysis. *Nephron*. 1991;58:121.
30. Steiner RW, Halasz NA. Abdominal catastrophes and other unusual events in continuous ambulatory peritoneal dialysis patients. *Am J Kidney Dis*. 1990;15:1-7.
31. Bistrup C, Biegel E, Pedersen RS. Sterile peritonitis in a CAPD patient caused by a juxtaperitoneal abscess. *Nephrol Dial Transplant*. 1995;10:406-407.
32. Alpert BE, Folkert VW, Longnecker RE, Sherman RA. Acute sterile peritonitis. *Nephron*. 1982;30:187-189.
33. Karanicolas S, Oreopoulos DG, Izatt S, et al. Epidemic of aseptic peritonitis caused by endotoxin during chronic peritoneal dialysis. *N Engl J Med*. 1977;296:1336-1337.
34. Mangram AJ, Archibald LK, Hupert M, et al. Outbreak of sterile peritonitis among continuous cycling peritoneal dialysis patients. *Kidney Int*. 1998;54:1367-1371.
35. Tintillier M, Pochet JM, Christophe JL, Scheiff JM, Goffin E. Transient sterile chemical peritonitis with icodextrin: clinical presentation, prevalence, and literature review. *Perit Dial Int*. 2002;22:534-537.
36. Toure F, Lavaud S, Mohajer M, et al. Icodextrin-induced peritonitis: study of five cases and comparison with bacterial peritonitis. *Kidney Int*. 2004;65:654-660.
37. Freiman JP, Graham DJ, Reed TG, McGoodwin EB. Chemical peritonitis following the intraperitoneal administration of vancomycin. *Perit Dial Int*. 1992;12:57-60.
38. Charney DI, Gouge SF. Chemical peritonitis secondary to intraperitoneal vancomycin. *Am J Kidney Dis*. 1991;17:76-79.
39. Piraino BM, Silver MR, Dominguez JH, Puschett JB. Peritoneal eosinophils during intermittent peritoneal dialysis. *Am J Nephrol*. 1984;4:152-157.
40. Ejaz AA, Fitzpatrick PM, Durkin AJ, et al. Pathophysiology of peritoneal fluid eosinophilia in peritoneal dialysis patients. *Nephron*. 1999;81:125-130.
41. Sridhar R, Thornley-Brown D, Kant KS. Peritonitis due to Aspergillus niger: diagnostic importance of peritoneal eosinophilia. *Perit Dial Int*. 1990;10:100-101.
42. Ampel NM, White JD, Varanasi UR, Larwood TR, Van Wyck DB, Galgiani JN. Coccidioidal peritonitis associated with continuous ambulatory peritoneal dialysis. *Am J Kidney Dis*. 1988;11:512-514.
43. Basyigit S, Sapmaz F, Bora F. Eosinophilic peritonitis caused by *Echinococcus granulosus* in a patient receiving maintenance peritoneal dialysis. *Ther Apher Dial*. 2016;20:92-93.
44. Wang HH, Yang LY, Chang JW, Hung YT, Lee TY, Tang RB. Eosinophilic peritonitis: an unusual manifestation of tuberculous peritonitis in peritoneal dialysis patient. *J Chin Med Assoc*. 2011;74:322-324.
45. Rosner MH, Chhatkuli B. Vancomycin-related eosinophilic peritonitis. *Perit Dial Int*. 2010;30:650-652.
46. Deweese R, Slavens J, Barua A, Sutton J. Vancomycin-induced eosinophilic peritonitis. *Am J Health Syst Pharm*. 2016;73:e243-e246.

47. Rathi M. Intraperitoneal streptokinase use-associated eosinophilic peritonitis. *Saudi J Kidney Dis Transpl*. 2015;26:128-131.
48. Kolb A, Gibson P, O'Sullivan ED. Post-transplant lymphoproliferative disorder presenting as cloudy peritoneal dialysate. *Perit Dial Int*. 2017;37:585-586.
49. Viray P, Setyapranata S, Holt SG. Hodgkin's lymphoma diagnosed from peritoneal effluent. *Perit Dial Int*. 2016;36:350-351.
50. Delgado-Cordova M, Penaloza JC, Fuentes A, Coronel F. Non-Hodgkin lymphoma mimicking peritonitis in a patient on peritoneal dialysis. *Nefrologia*. 2014;34:686-687.
51. Bargman JM, Zent R, Ellis P, Auger M, Wilson S. Diagnosis of lymphoma in a continuous ambulatory peritoneal dialysis patient by peritoneal fluid cytology. *Am J Kidney Dis*. 1994;23:747-750.
52. Talwar M, Self SE, Ullian ME. Prostate cancer metastatic to the peritoneum in a peritoneal dialysis patient. *Perit Dial Int*. 2012;32:570-572.
53. Dobbie JW. Serositis: comparative analysis of histological findings and pathogenetic mechanisms in nonbacterial serosal inflammation. *Perit Dial Int*. 1993;13:256-269.
54. Braun N, Fritz P, Ulmer C, et al. Histological criteria for encapsulating peritoneal sclerosis - a standardized approach. *PLoS ONE*. 2012;7:e48647.
55. Vanderperren B, Hammer F, Malaise J, Goffin E. Poor ultrafiltration shortly after peritoneal dialysis initiation. *Nephrol Dial Transplant*. 2002;17:2265-2267.
56. Gries E, Paar D, Graben N, Bock KD. Intraperitoneal heparin in peritoneal dialysis and its effect on fibrinopeptide A in plasma and dialysate. *Haemostasis*. 1989;19:21-25.
57. Voinescu CG, Khanna R. Peritonitis in peritoneal dialysis. *Int J Artif Organs*. 2002;25:249-260.
58. Kjeldsberg C, Knight J. *Body Fluids: Laboratory Examination of Cerebrospinal, Seminal, Serous and Synovial Fluids*, 3rd ed. Chicago, IL: American Society of Clinical Pathology Press; 1993.
59. Unger SW, Chandler JG. Chylous ascites in infants and children. *Surgery*. 1983;93:455-461.
60. Poux JM, Benevent D, Guiserix J, Le MY, Lagarde C, Leroux-Robert C. [Chylous ascites in 12 patients undergoing peritoneal dialysis]. *Nephrologie*. 1994;15:201-205.
61. Gupta R, Kitaichi M, Inoue Y, Kotloff R, McCormack FX. Lymphatic manifestations of lymphangioleiomyomatosis. *Lymphology*. 2014;47:106-117.
62. Huang CH, Chen HS, Chen YM, Tsai TJ. Fibroadhesive form of tuberculous peritonitis: chyloperitoneum in a patient undergoing automated peritoneal dialysis. *Nephron*. 1996;72:708-711.
63. Palumbo E. Filariasis: diagnosis, treatment and prevention. *Acta Biomed*. 2008;79:106-109.
64. Garcia FT, Rodriguez-Carmona A, Perez FM, et al. Complications of permanent catheter implantation for peritoneal dialysis: incidence and risk factors. *Adv Perit Dial*. 1994;10:206-209.
65. Hurley MK, Emiliani VJ, Comer GM, Patel A, Navarro C, Maiki CO. Dilated cardiomyopathy associated with chylous ascites. *Am J Gastroenterol*. 1989;84:1567-1569.
66. Riza AM, Avsar S, Yanik S. Chylous ascites and chylothorax due to constrictive pericarditis in a patient undergoing haemodialysis. *Neth J Med*. 2004;62:59-61.
67. Rector WG Jr. Spontaneous chylous ascites of cirrhosis. *J Clin Gastroenterol*. 1984;6:369-372.
68. Al-Busafi SA, Ghali P, Deschenes M, Wong P. Chylous ascites: evaluation and management. *ISRN Hepatol*. 2014;2014:240473.
69. Yoshimoto K, Saima S, Nakamura Y, et al. Dihydropyridine type calcium channel blocker-induced turbid dialysate in patients undergoing peritoneal dialysis. *Clin Nephrol*. 1998;50:90-93.
70. Yang WS, Huang JW, Chen HW, Tsai TJ, Wu KD. Lercanidipine-induced chyloperitoneum in patients on peritoneal dialysis. *Perit Dial Int*. 2008;28:632-636.
71. Ram R, Swarnalatha G, Pai BS, Rao CS, Dakshinamurty KV. Cloudy peritoneal fluid attributable to non-dihydropyridine calcium channel blocker. *Perit Dial Int*. 2012;32:110-111.
72. Saka Y, Tachi H, Sakurai H, et al. Aliskiren-induced chyloperitoneum in a patient on peritoneal dialysis. *Perit Dial Int*. 2012;32:111-113.
73. Perez FM, Pombo F, Soto A, Perez Fontan FJ, Rodriguez-Carmona A. Chylous ascites associated with acute pancreatitis in a patient undergoing continuous ambulatory peritoneal dialysis. *Nephron*. 1993;63:458-461.
74. Lee CK, Han JM, Lee KN, et al. Concurrent occurrence of chylothorax, chylous ascites, and protein-losing enteropathy in systemic lupus erythematosus. *J Rheumatol*. 2002;29:1330-1333.
75. Lindenbaum J, Scheidt SS. Chylous ascites and the nephrotic syndrome. Report of a case, associated with renal vein thrombosis. *Am J Med*. 1968;44:830-836.
76. Sujan SS, Ruiz SR, Cobelo C, Campos CT. Chyloperitoneum: is secondary amyloidosis a possible cause? *Perit Dial Int*. 2009;29:582-583.
77. Cappell MS, Friedman D, Mikhail N. Chyloperitoneum associated with chronic severe sarcoidosis. *Am J Gastroenterol*. 1993;88:99-101.
78. Gilkeson GS, Allen NB. Retroperitoneal fibrosis. A true connective tissue disease. *Rheum Dis Clin North Am*. 1996;22:23-38.
79. Isenberg JI, Gilbert SB, Pitcher JL. Ascites with peritoneal involvement in Whipple's disease. Report of a case. *Gastroenterology*. 1971;60:305-310.
80. Soylu A, Alaygut D, Yesilirmak D, et al. Resolution of chyloperitoneum in a preterm with octreotide, diet and cessation of dialysis. *Pediatr Nephrol*. 2010;25:363-366.
81. Lee PH, Lin CL, Lai PC, Yang CW. Octreotide therapy for chylous ascites in a chronic dialysis patient. *Nephrology*. 2005;10:344-347.
82. Hu SL, Kerns ES. Peach-colored effluent in an urgent-start PD patient. *Perit Dial Int*. 2015;35:603.
83. Chao CT, Huang JW. Asymptomatic green dialysate. *Am J Med Sci*. 2012;344:227.
84. Babin J, Langlois S, Friede J, Guay G, Agharazii M. Green dialysate: asymptomatic perforated cholecystitis without peritonitis. *Nephrol Dial Transplant*. 2006;21:1121-1122.
85. Connacher AA, Stewart WK. Pancreatitis causes brownish-black peritoneal dialysate due to the presence of methaemalbumin. *Nephrol Dial Transplant*. 1987;2:45-47.
86. Ling EK, Lin BS, Chiang SS, Tsai MH. Cola-colored dialysate as a gastrointestinal sequelae of cardiac surgery in a patient who underwent peritoneal dialysis. *Ren Fail*. 2012;34:1160-1162.
87. Lai MY, Yang WC, Chen JY, Lin CC, Ng YY. Hemoperitoneum in a woman with acute paraplegia. *Kidney Int*. 2006;69:639.
88. Shiao CC, Leu SC, Kao JL, Chang YM, Tsai SC. 'Soybean sauce' peritoneal dialysate. *NDT Plus*. 2011;4:75-76.
89. De SN, Wittebole X, Van den Bergh P, Haufroid V, Goffin E, Hantson P. Severe rhabdomyolysis associated with simvastatin and role of ciprofloxacin and amlodipine coadministration. *Case Rep Nephrol*. 2015;2015:761393.
90. Robertson S, Huxtable H, Blakemore C, Williams G, Donovan KL. The icodextrin black line sign. *Perit Dial Int*. 2001;21:621-623.
91. Sekercioglu N, Jassal SV. Early morning blues–a complication of icodextrin. *Perit Dial Int*. 2004;24:197-199.
92. Abrahams AC, Rutherford P, Boer WH. Purple icodextrin: Turkish delight? *Perit Dial Int*. 2013;33:445-447.

How to cite this article: Dossin T, Goffin E. When the color of peritoneal dialysis effluent can be used as a diagnostic tool. *Semin Dial*. 2018;00:1–8. https://doi.org/10.1111/sdi.12740