Dialysis-Associated Telangiectasia: Case Report and Review of the Literature

Patients with end-stage renal disease (ESRD) are at an increased risk of bleeding. We report the case of a 40-year-old female, on peritoneal dialysis for ESRD, who presented with profound anemia; she was later found to have a bleeding jejunal dialysis-associated telangiectasia. We review the literature published to date on dialysis-associated telangiectasia and discuss the possible underlying etiologies.

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KEY WORDS: Dialysis-associated telangiectasia; end-stage renal disease; peritoneal dialysis.

End-stage renal disease (ESRD) patients on dialysis are known to have significantly higher rates of gastrointestinal (GI) bleeding than normal (1). Several causes have been suggested including the use of anticoagulants, as well as platelet aggregation defects secondary to accumulating toxic products. Of the less common causes suggested is dialysis-associated telangiectasia (DAT). Dialysis-associated telangiectasia has been vaguely described in pathology literature. Telangiectasiae refer to dilated small blood vessels (i.e. arterioles, capillaries, and venules) with or without abnormal architecture. In DAT, these appear as small flattened reddish vessels with fernlike margins in the subserosa (2).

To date, there have only been 2 case series reported of DAT (total $n = 17$) in the literature (3,4). Interestingly, all patients reported were receiving hemodialysis. Here we report a case of jejunal telangiectasiae in a patient receiving continuous ambulatory peritoneal dialysis (CAPD).

CASE REPORT

A 40-year-old woman presented with a 2-day history of epigastric and hypogastric abdominal pain. She had a background history of ESRD due to reflux nephropathy, for which she received CAPD. She described the pain as intermittent,

and it was relieved by moving her bowel. The pain radiated to both flanks. She had associated postural lightheadedness, and described her bowel movements as dark and soft. She denied any weight loss, vomiting, difficulty swallowing, rectal bleeding, or post-prandial pain.

Of note, she was a smoker and had shown poor compliance to medications in the past. She was on vitamin D and calcium supplements only. She denied any over-the-counter medication use. At the time of presentation, she was afebrile and hemodynamically stable. She did, however, look pale and had cold peripheries. On examination, she had a soft abdomen with mild epigastric tenderness. Her initial hemoglobin was 44 g/L, and she received intravenous omeprazole and blood transfusions.

She was investigated with gastroscopy, which was normal, and then had a colonoscopy, which showed bleeding from a source proximal to the terminal ileum. Her hemoglobin continued to drop despite multiple transfusions (she required 15 units in total). A computed tomography angiogram was normal, and following this, she had a capsule endoscopy which showed bleeding in the proximal jejunum. She went on to have a balloon push enteroscopy which found the likely site of the bleeding, but the endoscopist was unable to intervene. The section of the jejunum was tattooed for later intra-operative identification.

She proceeded to laparotomy and the tattoo was identified about 140 cm from the duodenojejunal flexure and 250 cm from the ileocecal valve. No obvious lesion was found in the small bowel. Approximately 40 cm of small bowel was then resected: 10 cm proximal to the tattoo and 30 cm distal.

Four days post-laparotomy, she was clinically much better and her hemoglobin was stable. She was discharged to return for dialysis and hemoglobin check the following day. Pathological examination of the resected specimen revealed numerous tortuous dilated blood vessels, predominantly venous but with thickened walls, within the submucosa, muscularis propria, and mesenteric fat.

DISCUSSION

The pathogenesis of DAT is not well-defined and is likely to be multifactorial. Intermittent overhydration secondary to water and sodium retention could contribute to venous hypertension and subsequent venous dilatation. In addition, hemodialysis requires long-term use of aluminium hydroxide gel to control hyperphosphatemia. Chronic exposure to aluminium has been implicated in the development of telangiectasia (5). Trauma from the peritoneal catheter and/or pressure from the dialysis fluid has been theorized to induce the formation of intra-abdominal aneurysms and other vascular defects, all of which could potentially contribute to the formation of telangiectasiae (6).

Dialysis-associated telangiectasia can result in multiple telangiectasiae throughout the GI tract. Most patients with DAT reported to date had gastric telangiectasia with or without involvement of other sites in the GI tract (3,4). In a recent endoscopic study, 75 patients with known GI telangiectasiae

TABLE 1
Key Clinico-Pathological Differences Between Telangiectasia and Angiodysplasia

	Telangiectasia	Angiodysplasia
Origin	Typically congenital	Typically acquired
Etiology	Abnormal direct communication between arteries and veins	Abnormally dilated blood vessels
Morphology	Complex clusters of tortuous dilated vascular channels and vessels that are intermediate between arteries and veins	Dilated tortuous veins and venules with associated dilated capillaries
Histological site(s)	Typically external to the muscularis propria (i.e. subserosal)	Submucosal (with or without mucosal involvement)
Gross site(s)	Throughout the gastrointestinal tract (especially stomach)	Cecum and ascending colon
May be associated with	1. Hereditary hemorrhagic telangiectasia (8) 2. DAT 3. von Willebrand's disease (9) 4. CREST syndrome (10) 5. Turner's syndrome (11) 6. Cutaneous telangiectasia (12)	Aortic stenosis (Heyde's syndrome) (13)

DAT = dialysis-associated telangiectasia; CREST = calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasia.

underwent oesophagogastroduodenoscopy, capsule endoscopy, and colonoscopy. Of the study population, 5 patients (7%) had ESRD, although the study does not allude to their use of dialysis (7).

It is interesting to note that telangiectasias seen in DAT differ from the more common angiodysplasias often seen in the colon. It is important to distinguish them, as each might be associated with different clinical syndromes. Table 1 shows the different characteristics of GI telangiectasias and angiodysplasias. Management of DAT depends on the patient's symptomatology. Cited options include medical therapy (e.g., estrogens, danazol, or aminocaproic acid), therapeutic endoscopy, or surgical excision (3,4).

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The authors have no financial conflicts of interest to declare.

Yassar Alamri^{1*}
James Tietjens²
Christopher Wakeman³

Department of Medicine¹
University of Otago, Dunedin, New Zealand
Department of General Surgery²
Capital and Coast District Health Board
Wellington, New Zealand
Department of General Surgery³
Christchurch Public Hospital, Christchurch, New Zealand

*email: yassar.alamri@nzbri.org

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