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Lanthanum deposition from oral lanthanum carbonate in the upper gastrointestinal tract

Raza S. Hoda, M.D.,
Department of Pathology, Massachusetts General Hospital, Boston, MA

Soma Sanyal, M.D.,
Department of Pathology, SUNY Upstate Medical University, Syracuse, NY

Jerrold L. Abraham, M.D.,
Department of Pathology, SUNY Upstate Medical University, Syracuse, NY

Jamie M. Everett, M.D.,
Department of Pathology, Massachusetts General Hospital, Boston, MA

Gregory L. Hundemer, M.D., M.P.H.,
Department of Nephrology Service of Medicine, Massachusetts General Hospital, Boston, MA

Eric Yee, M.D.,
Department of Pathology, Beth Israel Beth Israel Deaconess Medical Center, Boston, MA

Gregory Y. Lauwers, M.D.,
Department of Pathology, Massachusetts General Hospital, Boston, MA

Nina Tolkoff-Rubin, M.D., and
Department of Nephrology Service of Medicine, Massachusetts General Hospital, Boston, MA

Joseph Misdraji, M.D.
Department of Pathology, Massachusetts General Hospital, Boston, MA

Address correspondence to: Joseph Misdraji, M.D., Massachusetts General Hospital, Department of Pathology, 55 Fruit Street, Boston, MA 02114, jmisdraji@partners.org.
Current affiliation: Dr. Jamie M. Everett, Department of Pathology and Laboratory Medicine, The University of Texas Health Science Center, Houston, TX
Current affiliation: Dr. Eric Yee, Department of Pathology, University of Oklahoma Medical System, Oklahoma City, OK
Current affiliation: Dr. Gregory Y. Lauwers, H. Lee Moffitt Cancer Center, Department of Anatomic Pathology, 12902 USF Magnolia Drive, MCC Lab, Tampa, FL 33612

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Raza S. Hoda, M.D.:, Manuscript writing, selection of figures,
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Jamie M. Everett M.D.: Collection of cases, writing of initial draft.
Gregory L. Hundemer, M.D., Nina Tolkoff-Rubin, M.D.: Clinical information, clinical context, clinical portion of manuscript
Eric Yee, M.D.: Contribution of cases, clinical information from outside institution, editing manuscript
Gregory Y. Lauwers, M.D.: Initial diagnosis of lanthanum, editing manuscript
Joseph Misdraji, M.D.: Supervised project, final editing and writing of manuscript

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Abstract

Aims—Lanthanum carbonate is used an alternative to calcium-based phosphate binders to manage hyperphosphatemia in patients with renal failure. The deposition of lanthanum within gastroduodenal mucosa of patients treated with the medication has been described, but given the relative novelty of this entity, the histiocytic deposits in the gastroduodenal mucosa can be confused with a variety of other processes, including infections and other drug-induced forms of injury.

Methods and Results—We describe 5 cases of lanthanum phosphate deposition in upper gastrointestinal tract biopsies. Three cases were confirmed with scanning electron microscopy and energy dispersive x-ray analysis, including one unique patient, status post renal transplant for polycystic kidney disease, who had last taken lanthanum 7 years prior to biopsy.

Conclusion—Lanthanum deposition in the upper GI tract is a mimic of other drug related forms of gastrointestinal injury, including iron pill related gastropathy. The key to making this diagnosis is a thorough drug history and awareness of the histologic features.

Keywords

Lanthanum carbonate; histiocytosis; stomach; duodenum

INTRODUCTION

In 2004, lanthanum carbonate, an orally-administrated non-calcium based phosphate-binder, was approved by the United States' Food and Drug Administration (FDA) for treatment of hyperphosphatemia in patients on dialysis. Lanthanum carbonate has been reported to be effective with decreased pill burden, fewer adverse effects, and potentially decreased risk of mortality compared to calcium-based phosphate binders.[1]

Lanthanum carbonate therapy is not, however, without complications. Various adverse gastrointestinal effects have been described in patients on lanthanum carbonate therapy, particularly dysphagia, nausea, vomiting, and reflux.[1–3] Endoscopic findings include the presence of non-specific gastritis, gastric erosion, gastric ulceration, gastric polyps, and duodenal ulceration.[4–9] Biopsies taken from gastric and duodenal lesions have shown accumulations of histiocytes filled with granular material within the lamina propria. These deposits have been characterized as lanthanum phosphate by scanning electron microscopy and energy dispersive x-ray analysis.[4–10]

Despite several reports of deposition of lanthanum in tissue, the histopathologic features associated with lanthanum carbonate are not well recognized. Herein, we describe our experience with 5 patients with gastrointestinal changes secondary to lanthanum carbonate therapy, including one with only a remote history of lanthanum carbonate treatment. Scanning electron microscopy and Energy Dispersive X-ray Spectroscopy (EDS) were used to confirm the presence of lanthanum and phosphorus in these deposits in three cases. The study was approved by the Massachusetts General Hospital IRB committee (November 16, 2015, protocol 2015P002424). Informed, written consent was waived. The research was performed in accordance with the Declaration of Helsinki.

MATERIALS AND METHODS

Three upper gastrointestinal tract biopsies that had accumulation of histiocytes with coarsely granular, amphophilic material within the lamina propria were seen in routine surgical pathology biopsy material at Massachusetts General Hospital, Boston, Massachusetts over an interval of two months. Two cases were seen in routine biopsy material at the Beth Israel Deaconess Medical Center, Boston, Massachusetts over the same period.

To confirm the presence of lanthanum in the deposits, the freshly-cut surface of paraffin blocks of three cases (including one patient no longer taking lanthanum) were examined directly with a scanning electron microscope (SEM), using the variable pressure system of the FEI (previously ASPEX) SEM, as previously described.[11] The operating conditions were 20 keV accelerating voltage, 15-to 16-mm working distance, 0.15 torr pressure in the specimen chamber. Back-scattered electron imaging revealed relatively high atomic number inorganic materials in a low atomic number organic matrix (tissue). The EDS spectra collected from individual features of micrometer dimensions were compared with standard reference spectra of chemical elements, thus demonstrating the elemental composition of each feature.

In order to determine if other cases had been missed, a computer search for lanthanum, histiocytosis, gastric mucosal calcinosis, medication-induced gastritis, and iron therapy-related gastritis was performed, and all cases diagnosed at Massachusetts General Hospital over a 2-year period, from July 2013 to July 2015, were retrieved and reviewed. No additional cases were found, although this may reflect the difficulty of uncovering cases using language searches.

RESULTS

The clinical features are summarized in Table 1. The three men and two women ranged in age from 29–66 years (mean 53 years). Four patients had end-stage renal disease at the time of biopsy. Three patients were on peritoneal dialysis, and 1 was on hemodialysis. Total daily doses of lanthanum carbonate varied from 3,000 mg to 6,000 mg per day. One patient (case 5) had received a successful deceased donor renal transplant 7 years prior and had not taken lanthanum carbonate since the transplant. The most common gastrointestinal symptoms that lead to upper endoscopy were nausea and reflux. Endoscopic abnormalities most often were found in the gastric antrum and included erythematous mucosal changes.

Lanthanum deposits were found in 3 gastric antral biopsies, 2 gastric fundic biopsies, and 1 duodenal biopsy. One patient had both duodenal and antral deposits and this patient also had biopsies of an esophageal ulcer with non-specific histology presumably related to reflux. In all cases, the deposits consisted of histiocytes within the lamina propria, in some cases expanding the lamina propria; the histiocytes contained amphophilic granular material, together with coarser brown to deep purple material (Figure 1). The material was refractile but not birefringent under polarized light, indicating amorphous rather than crystalline material. The histiocytes were pale and vaguely epithelioid but in only one case did they form vaguely rounded aggregates reminiscent of poorly formed granulomas (Figure 1). All

cases had at least rare histiocytes that were binucleated or multinucleated. The antral biopsies had either an atrophic appearance or an appearance of reactive gastropathy with foveolar hyperplasia and focal corkscrewing of the pits. The fundic biopsies had less dramatic changes, with foveolar hyperplasia in one case only. The duodenal biopsy showed dramatic expansion of the lamina propria with histiocytes, congestion of the villous tips, and focal foveolar metaplasia.

Von Kossa stains were performed in all six biopsies and were negative in all cases. Iron stains were performed on all cases, and showed faint staining of the largest particles in most cases (Figure 2). One case had faint staining of the more fine granular deposits, and one case was negative. Periodic acid Schiff (PAS) with diastase digestion was performed in two cases and showed faint staining of the granular material. Two cases were assessed with acid fast and GMS stains, and were negative. One case was assessed with Fite stain, Brown-Hopps stain, PCR for Whipple's disease, and immunohistochemistry for H. pylori, and these studies were negative.

All cases were signed out initially as non-specific. In several cases, a differential was offered of drug ingestion, ingestion of exogenous substances, and in two cases the possibility of infection was suggested as well. One pathologist also suggested mucosal calcinosis as a possibility.

On dark field imaging, the granular material within histiocytes was strongly refractile. SEM/EDS analysis showed the intracellular deposits contained lanthanum, phosphorus, and calcium (Figure 3). The relative peak heights for La, P and Ca were similar in the 3 cases, indicating that the deposits are not pure lanthanum phosphate but a compound that also contains calcium.

DISCUSSION

Hyperphosphatemia is an important risk factor for the development of secondary hyperparathyroidism, metabolic bone disease, vascular calcification, and cardiovascular events in patients with chronic renal failure. Consequently, the management of hyperphosphatemia is an important therapeutic target for this patient population. Treatment of high serum phosphorus includes phosphate binders. The utility of aluminum- and calcium-based phosphate binders has been limited by their side effect profile. Lanthanum carbonate has emerged as a safer calcium-free alternative to traditional phosphate-binding therapies.[4, 12, 13] Lanthanum is a rare alkaline earth metal that binds with phosphate in the gastrointestinal tract and is mostly excreted in the feces.[4, 14, 15] Despite a poor absorption rate, lanthanum accumulates in bone, liver, mesenteric lymph nodes, and plasma in humans, albeit at low levels.[16–20]

Histologic recognition of lanthanum deposition in human tissues was first reported in 2009 in a mesenteric lymph node found during post-mortem examination of a 38-year-old woman with nephrogenic systemic fibrosis from gadolinium and end-stage renal disease secondary to IgA nephropathy.[16] Five years later, lanthanum phosphate deposition in gastrointestinal mucosa was described in a 63-year-old woman with end-stage renal disease, who had

undergone peritoneal dialysis and had taken lanthanum carbonate for nearly 4 years.[4] Since then, multiple reports have described lanthanum carbonate deposition in gastrointestinal biopsies. In a case series of 13 patients, Goto et al. described histologic evidence of lanthanum deposition in gastric, duodenal, and colonic biopsies in 68% of patients who had taken lanthanum carbonate.[6] Most reported cases, and all of the cases in our series, demonstrate lanthanum deposition in the upper gastrointestinal tract, specifically the stomach. Lanthanum deposition appears as prominent accumulation of histocytes within the lamina propria, just beneath the surface epithelium.[6–8, 21, 22] The histocytes are filled with material that has been described in the various reports as either fine or coarse and ranging from colorless to amphophilic.[6–8, 21, 22] The lanthanum accumulation within the histocytes has been found to stain positively for PAS, PAS after diastase digestion, and Prussian blue in some cases.[4, 5]

Lanthanum deposition in the upper gastrointestinal tract may cause a variety of clinical symptoms, including dysphagia, nausea, vomiting, and reflux,[5] although these are common and may not be related to the lanthanum. Endoscopic findings are usually nonspecific, as they were in our cases, and include gastritis, gastric erosion, gastric erythema, gastric ulceration, polypoid gastric mucosa, and duodenal ulceration.[4–9, 17–19, 21, 22] Lanthanum deposition has been detected as early as 3 months after administration.[18] Several reports suggest that the process is reversible after cessation of lanthanum carbonate use.[20] In one case report, Rothenberg et al.[8] found that after three months of cessation of lanthanum carbonate therapy, there was no histologic evidence of lanthanum in the stomach, marked decrease of the histocytosis in the duodenum, and resolution of the patient's symptoms, indicating that the lanthanum deposition decreases after cessation of therapy and may be reversible given time. However, there have been experiences that raise questions about the reversibility of lanthanum deposition. Davis and Abraham described persistent granular lanthanum deposition within histiocytes of a mesenteric lymph node of a patient who had not received lanthanum carbonate for 3 years.[16] In our cohort, a 52-year-old man with end-stage renal disease secondary to autosomal dominant polycystic kidney disease was found to have lanthanum deposition in a gastric biopsy 7 years after discontinuation of lanthanum carbonate. The consequences of lanthanum deposition in tissue, particularly the long-term consequences in patients in whom the process does not seem to be reversible, are unknown.

Another rare earth metal that may serve as a cautionary tale is gadolinium, an element used as a contrast agent with magnetic resonance imaging. Gadolinium-containing contrast agents are almost exclusively cleared by the kidney and the half-life increases dramatically with advanced or end stage kidney disease.[23] With prolonged exposure due to poor renal clearance, gadolinium deposits in multiple organs, including the skin.[23–26] Affected individuals develop a debilitating condition known as nephrogenic systemic fibrosis (NSF) where they develop thickened, fibrotic skin which can cause pain and severely restricted mobility.[27–29] No proven medical therapy exists for NSF and current practice is to avoid gadolinium exposure in patients with advanced or end stage kidney disease. Notably, the association between gadolinium exposure and NSF did not come to light until 18 years after gadolinium had been approved by the FDA. It is also worth noting that the ratios of Gd:P:Ca seen in the insoluble deposits in NSF cases are similar to the La:P:Ca ratios seen in our cases.[30]

The presence of histiocytes with granular material within the lamina propria may not be recognized as lanthanum, and in fact, none of our cases was initially recognized as being lanthanum deposition. Although the possibility of drug-related GI tract injury was entertained in several cases, there was a broad differential diagnosis in these cases, and numerous stains were applied in an attempt to define the process. The differential diagnoses of the granular material considered in our series included gastric mucosal calcinosis, iron-therapy-induced gastric injury, and infection. The fact that some of these deposits variably stain with Prussian blue stain may cause confusion with iron-therapy related gastric injury, although in that condition, the deposits usually appears as yellow to brown coarse extracellular deposits in the superficial lamina propria that stain strongly with Prussian blue stain.[31] Interestingly, despite the variable staining with iron stains, EDS did not demonstrate iron, possibly related to the sensitivity of EDS or the state of the iron. Gastric mucosal calcinosis typically shows coarse basophilic deposits in the superficial lamina propria that stain strongly with von Kossa stains for calcium.[32]

In conclusion, we believe that lanthanum deposition is readily recognizable by pathologists, once they are aware of the entity. Nephrologists should alert pathologists to the patient's drug history. The identification of lanthanum deposits in these patients may be important, as the long-term consequences of lanthanum in tissue are unknown, the use of this medication is increasing, and other options to manage hyperphosphatemia exist in patients with end stage renal failure. We have also shown that lanthanum deposition may not be reversible in some patients, as evidenced by our transplant patient who had discontinued lanthanum 7 years prior and now has normal renal function. The long-term effects of lanthanum carbonate are still to be determined.

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References

1. Jamal SA, Vandermeer B, Raggi P, et al. Effect of calcium-based versus non-calcium-based phosphate binders on mortality in patients with chronic kidney disease: an updated systematic review and meta-analysis. Lancet. 2013; 382:1268–1277. [PubMed: 23870817]
2. Yamada S, Yoshida H, Taniguchi M, et al. Effectiveness of lanthanum carbonate treatment used in combination with other phosphate binders in peritoneal dialysis patients. Intern Med. 2012; 51:2097–2104. [PubMed: 22892485]
3. Rombola G, Londrino F, Corbani V, et al. Lanthanum carbonate: a postmarketing observational study of efficacy and safety. J Nephrol. 2012; 25:490–496. [PubMed: 22476966]
4. Makino M, Kawaguchi K, Shimojo H, et al. Extensive lanthanum deposition in the gastric mucosa: the first histopathological report. Pathol Int. 2015; 65:33–37. [PubMed: 25413959]
5. Haratake J, Yasunaga C, Ootani A, et al. Peculiar histiocytic lesions with massive lanthanum deposition in dialysis patients treated with lanthanum carbonate. Am J Surg Pathol. 2015; 39:767–771. [PubMed: 25602800]
6. Goto K, Ogawa K. Lanthanum Deposition Is Frequently Observed in the Gastric Mucosa of Dialysis Patients With Lanthanum Carbonate Therapy: A Clinicopathologic Study of 13 Cases, Including 1

Case of Lanthanum Granuloma in the Colon and 2 Nongranulomatous Gastric Cases. Int J Surg Pathol. 2016; 24:89–92. [PubMed: 26490721]

7. Khurram M, Montgomery E. Lanthanum carbonated-associated injury to the small bowel. Diagnostic Histopathology. 2015; 21:452–454.
8. Rothenberg ME, Araya H, Longacre TA, et al. Lanthanum-Induced Gastrointestinal Histiocytosis. ACG Case Rep J. 2015; 2:187–189. [PubMed: 26157959]
9. Mancano MA. Gastrointestinal Nodules and Bleeding with Long-Term Lanthanum Use; DRESS and Hepatotoxicity Due to Rivaroxaban; Thrombocytopenia Induced by Pentoxifylline; Amlodipine-Induced Schamberg's Disease; Varenicline-Induced Acute Liver Injury. Hosp Pharm. 2016; 51:284–287. [PubMed: 27303074]
10. Tonooka A, Uda S, Tanaka H, et al. Possibility of lanthanum absorption in the stomach. Clin Kidney J. 2015; 8:572–575. [PubMed: 26413283]
11. Nasr MR, Savici D, Tudor L, et al. Inorganic dust exposure causes pulmonary fibrosis in smokers: analysis using light microscopy, scanning electron microscopy, and energy dispersive X-ray spectroscopy. Arch Environ Occup Health. 2006; 61:53–60. [PubMed: 17649956]
12. Graff L, Burnel D. A possible non-aluminum oral phosphate binder? A comparative study on dietary phosphorus absorption. Res Commun Mol Pathol Pharmacol. 1995; 89:373–388. [PubMed: 8680806]
13. Finn WF, Joy MS, Hladik GA, et al. Results of a radnomized dose-ranging, placebo controlled study of lanthanum carbonate for reduction of serum phosphate in chronic renal failure patients receiving hemodialysis. J Am Soc Neprhol. 1999; 10:261A.
14. Hutchinson AJ. Calcitriol, lanthanum carbonate, and other new phosphate binders in the management of renal osteodystrphy. Perit Dial Int. 1999; 19(Suppl 2):S408–412. [PubMed: 10406555]
15. Hutchinson AJ, Wilson RJ, Garafola S, et al. Lanthanum carbonate: safety data after 10 years. Nephrology (Carlton). 2016 epub ahead of print.
16. Davis RL, Abraham JL. Lanthanum deposition in a dialysis patient. Nephrol Dial Transplant. 2009; 24:3247–3250. [PubMed: 19625369]
17. Iwamuro M, Kanzaki H, Tanaka T, et al. Lanthanum phosphate deposition in the gastric mucosa of patients with chronic renal failure. Nihon Shokakibyo Gakkai Zasshi. 2016; 113:1216–1222. [PubMed: 27383105]
18. Yabuki K, Shiba E, Harada H, et al. Lanthanum deposition in the gastrointestinal mucosa and regional lymph nodes in dialysis patients: Analysis of surgically excised specimens and review of the literature. Pathol Res Pract. 2016
19. Valika AK, Jain D, Jaffe PE, et al. A Nodular Foreign Body Reaction in a Dialysis Patient Receiving Long-term Treatment With Lanthanum Carbonate. Am J Kidney Dis. 2016; 67:128–132. [PubMed: 26385816]
20. Spasovski GB, Sikole A, Gelev S, et al. Evolution of bone and plasma concentration of lanthanum in dialysis patients before, during 1 year of treatment with lanthanum carbonate and after 2 years of follow-up. Nephrol Dial Transplant. 2006; 21:2217–2224. [PubMed: 16595583]
21. Iwamuro M, Tanaka T, Urata H, et al. Lanthanum phosphate deposition in the duodenum. Gastrointest Endosc. 2016 In press.
22. Yasunaga C, Haratake J, Ohtani A. Specific Accumulation of Lanthanum Carbonate in the Gastric Mucosal Histiocytes in a Dialysis Patient. Ther Apher Dial. 2015; 19:622–624. [PubMed: 26420000]
23. High WA, Ayers RA, Chandler J, et al. Gadolinium is detectable within the tissue of patients with nephrogenic systemic fibrosis. J Am Acad Dermatol. 2007; 56:21–26. [PubMed: 17097388]
24. High WA, Ayers RA, Cowper SE. Gadolinium is quantifiable within the tissue of patients with nephrogenic systemic fibrosis. J Am Acad Dermatol. 2007; 56:710–712. [PubMed: 17289213]
25. Boyd AS, Zic JA, Abraham JL. Gadolinium deposition in nephrogenic fibrosing dermopathy. J Am Acad Dermatol. 2007; 56:27–30. [PubMed: 17109993]
26. Schroeder JA, Weingart C, Coras B, et al. Ultrastructural evidence of dermal gadolinium deposits in a patient with nephrogenic systemic fibrosis and end-stage renal disease. Clin J Am Soc Nephrol. 2008; 3:968–975. [PubMed: 18385397]

27. Galan A, Cowper SE, Bucala R. Nephrogenic systemic fibrosis (nephrogenic fibrosing dermopathy). Curr Opin Rheumatol. 2006; 18:614–617. [PubMed: 17053507]

28. Marckmann P, Skov L, Rossen K, et al. Nephrogenic systemic fibrosis: suspected causative role of gadodiamide used for contrast-enhanced magnetic resonance imaging. J Am Soc Nephrol. 2006; 17:2359–2362. [PubMed: 16885403]

29. Grobner T. Gadolinium–a specific trigger for the development of nephrogenic fibrosing dermopathy and nephrogenic systemic fibrosis? Nephrol Dial Transplant. 2006; 21:1104–1108. [PubMed: 16431890]

30. George SJ, Webb SM, Abraham JL, et al. Synchrotron X-ray analyses demonstrate phosphate-bound gadolinium in skin in nephrogenic systemic fibrosis. Br J Dermatol. 2010; 163:1077–1081. [PubMed: 20560953]

31. Abraham SC, Yardley JH, Wu TT. Erosive injury to the upper gastrointestinal tract in patients receiving iron medication: an underrecognized entity. Am J Surg Pathol. 1999; 23:1241–1247. [PubMed: 10524525]

32. Greenson JK, Trinidad SB, Pfeil SA, et al. Gastric mucosal calcinosis. Calcified aluminum phosphate deposits secondary to aluminum-containing antacids or sucralfate therapy in organ transplant patients. Am J Surg Pathol. 1993; 17:45–50. [PubMed: 8447508]

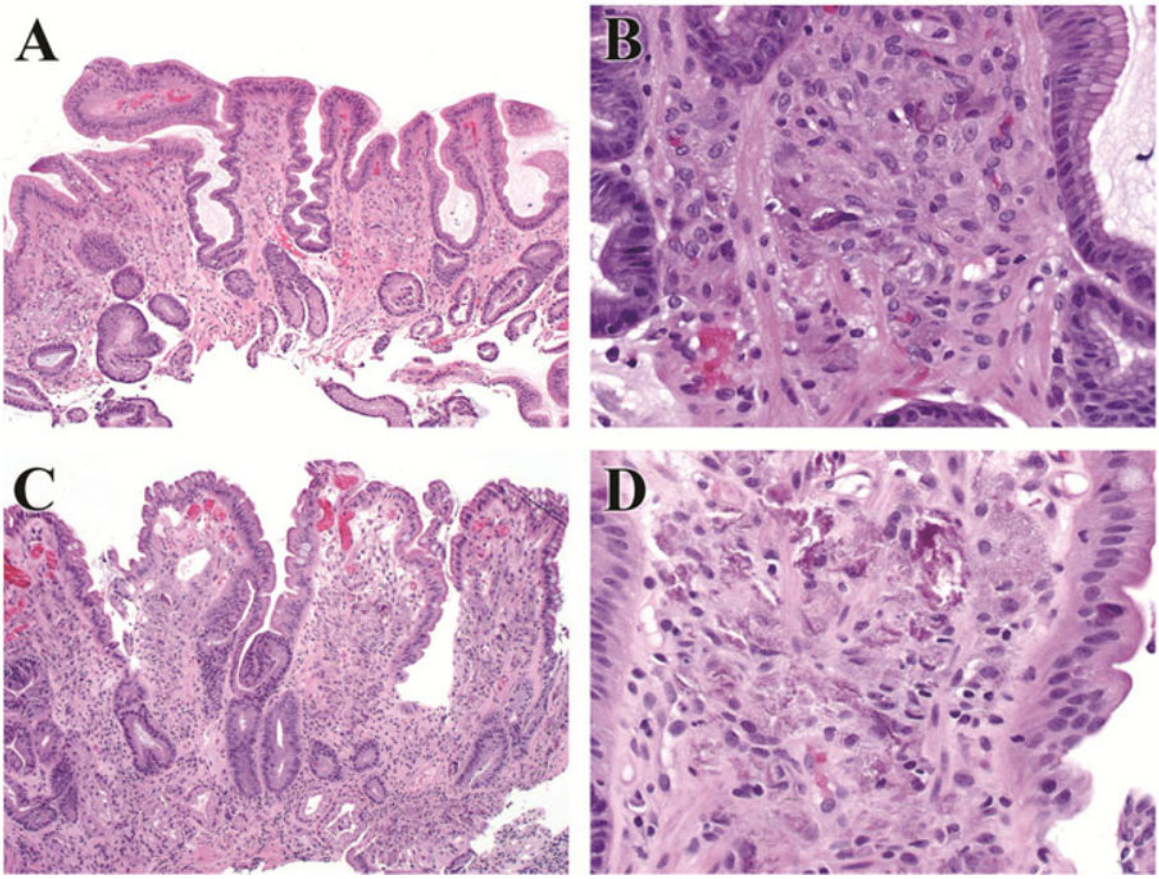

Figure 1.

Lanthanum deposits in macrophages in stomach and duodenum. A) Case 2. Low power view of the gastric antrum shows expansion of the lamina propria with histiocytes and corkscrewing of the gastric pits. B). High power view of the same case shows histiocytes forming a vaguely round aggregate. C). Case 3. Low power view of a duodenal biopsy shows marked expansion of the lamina propria with histiocytes. D). High power view of the biopsy in C showing the granular amorphous material in histiocytes that appears light brown to purple.

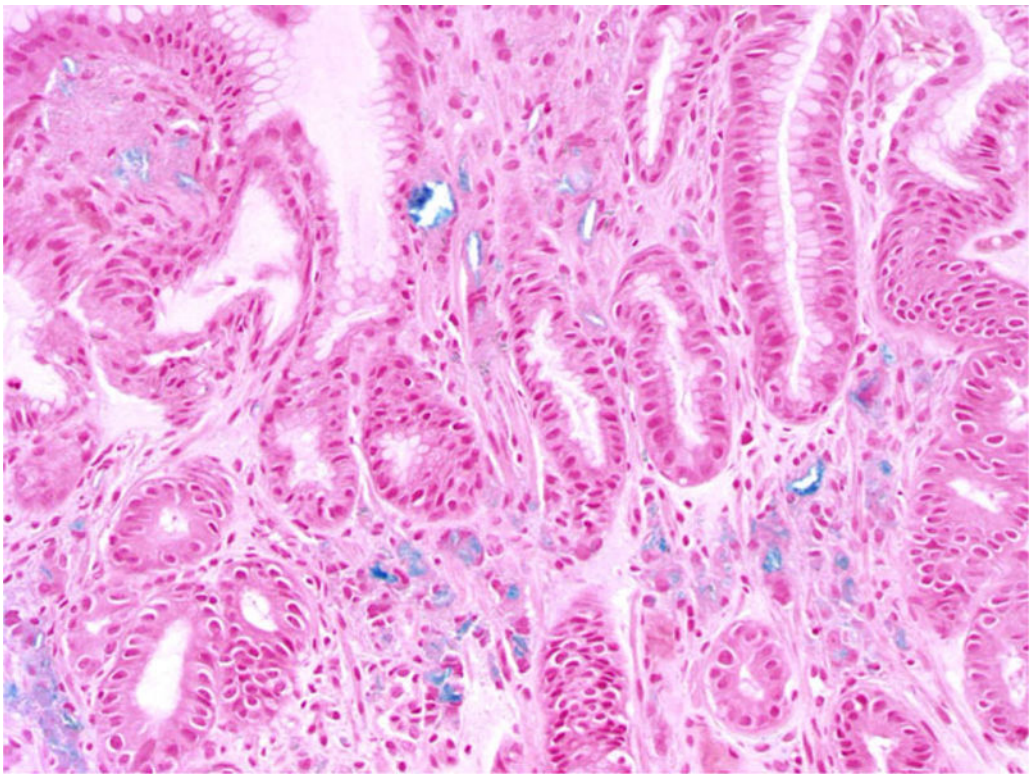

Figure 2.
Case 3. Prussian blue stain of the gastric biopsy showing staining of the coarser deposits.

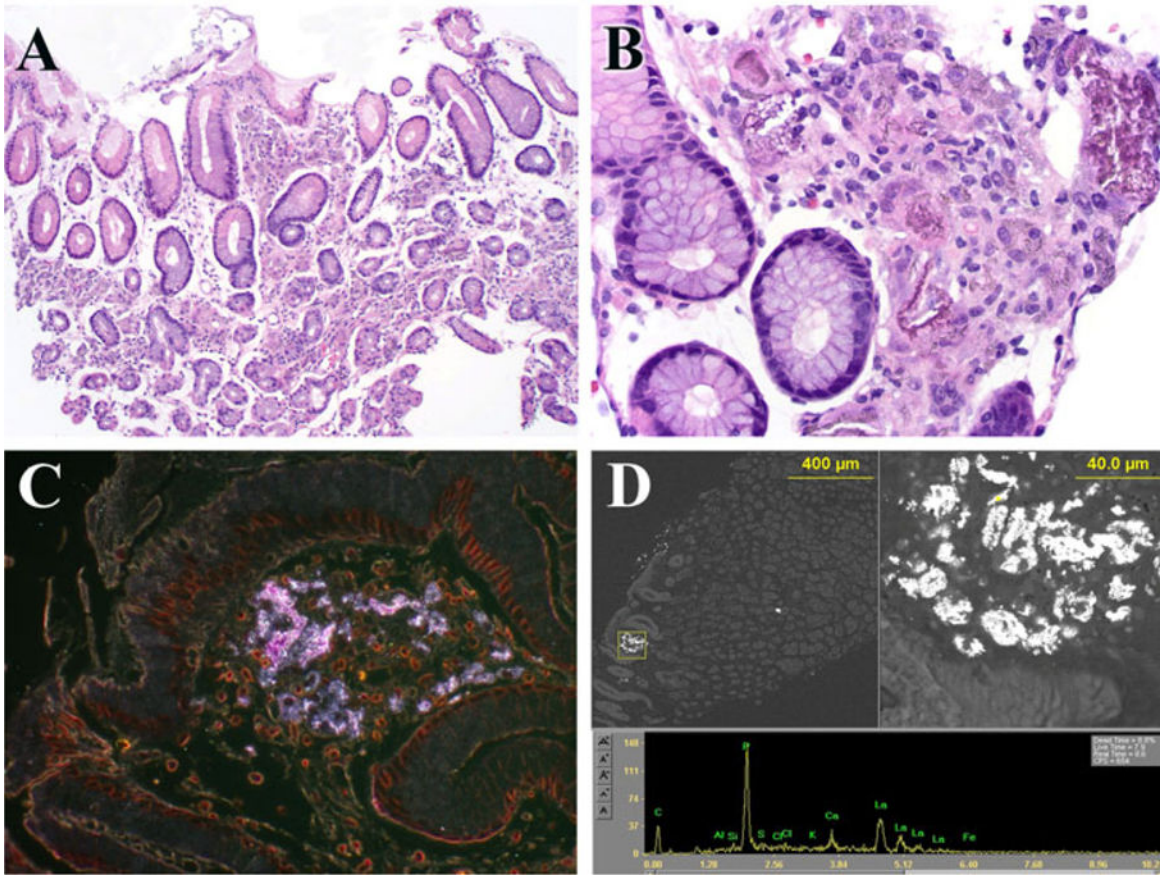

Figure 3.

Case 5. A. Low power view of gastric fundus shows histiocytes in the lamina propria in a patient 7 years after taking lanthanum carbonate. B. High power view of the gastric fundus in this patient shows that the histiocytes contain granular amorphous material with coarse, brown to purple material. C: Darkfield LM image of the deposits, showing brightly refractile deposits in histiocytes. D. SEM with back-scattered electron images indicating high atomic number composition of deposits, and EDS spectrum confirming composition of La, P, Ca.

Table 1

Clinical features of patients with lanthanum carbonate mucosal deposition

Case	Sex (M/F)	Age (yrs)	Duration of lanthanum carbonate therapy (yrs)	Site(s) involved	Clinical symptoms	Endoscopic findings	Etiology of renal disease
1	M	66	3	Antrum	Dysphagia, reflux, and nausea	Erythema of gastric mucosa	Hypertension
2	M	63	1	Antrum	Foreign body ingestion	Erythema in the gastric antrum.	IgA Nephropathy
3	F	53	6	Antrum Duodenum	Epigastric burning sensation and reflux	Granular and micronodular mucosa in the gastric antrum and duodenum	Diabetes Mellitus
4	F	29	2	Fundus	Early satiety, nausea, and vomiting	Two superficial, non-bleeding ulcers	Lupus nephritis
5	M	52	3 (7 years prior to biopsy)	Fundus	Melena	Gastric mucosal lymphangiectasia	ADPKD (status post renal transplant)

ADPKD: Autosomal dominant polycystic kidney disease