Journal of
Medical Case Reports

CASE REPORT

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# Incidental diagnosis of gastric antral vascular ectasia in a case of chronic kidney disease from Nepal: a case report

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## Abstract

**Introduction** Gastric antral vascular ectasia is an uncommon clinical disease that affects elder people and is characterized by severe chronic upper gastrointestinal bleeding mainly affecting the gastric antrum. It is generally unusual among patients undergoing maintenance hemodialysis for chronic kidney disease.

**Case presentation** Here, we aim to present an uncommon case of incidental diagnosis of the gastric antral vascular ectasia and erosive gastritis in a 71-year-old Hindu male patient belonging to the Gurung ethnicity of Nepal undergoing maintenance hemodialysis due to chronic kidney disease. The patient presented with a history of melena and fatigue. On investigation, a low hemoglobin level of 7.3 gm% was used for blood transfusion. The patient was on regular hemodialysis after admission at our institution. Upper gastrointestinal bleeding was suspected after analyzing patient's history and investigations. Therefore, an upper gastrointestinal endoscopy was performed that showed linear ectatic punctuate lesions radiating from the body of the stomach to the antrum, and hence, an incidental diagnosis of the gastric antral vascular ectasia was made. Initial fluid resuscitation, iron therapy, and a triple regimen were administered. Proper management with argon plasma coagulation therapy was scheduled at another institution due to lack of respective facilities in our institution.

**Discussion** Gastric antral vascular ectasia is an unusual cause of upper gastrointestinal bleeding, primarily affecting the gastric antrum and pylorus with rare cases affecting the duodenum, jejunum, and gastric fundus. It is generally associated with other chronic disease conditions. Several hypotheses have been proposed for the pathogenesis of gastric antral vascular ectasia, especially its association with chronic kidney disease, as in our case, which is considered to be rare. Management varies from medical to endoscopic interventions to even surgery.

**Conclusion** Prompt proper diagnosis and treatment for the gastric antral vascular ectasia should be sought, as it is frequently misdiagnosed or missed during upper gastrointestinal endoscopy. Our case report presents a case of gastric antral vascular ectasia in chronic kidney disease undergoing maintenance hemodialysis, which is quite uncommon, as literature has suggested the same point.

**Keywords** CKD, Case report, Gave, Hemodialysis

## Introduction

Gastric antral vascular ectasia (GAVE) is an uncommon clinical disease that is associated with upper gastrointestinal bleeding and mainly affects the elderly population in their seventies [1]. First reported by Rider *et al.* [2] in 1953, GAVE was characterized by telangiectatic blood vessels involving the gastric antrum and pylorus. The

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term "watermelon stomach" was coined by Jabbari *et al.* [3] and is characterized by longitudinal rugal folds with visible columns of convoluted vessels resembling watermelon stripes. It has also been reported to be associated with other chronic diseases of the liver, kidney, and connective tissue disorders. The disease also often shows association with autoimmune and collagen vascular diseases, such as systemic sclerosis and atrophic gastritis, along with even hypothyroidism [4, 5]. Here, we intend to report a case of a 71-year-old Hindu male with a diagnosis of chronic kidney disease (CKD) who was receiving hemodialysis and presented at our institution where an incidental diagnosis of GAVE was made. This case is the second of its kind to be reported from Nepal, the first being reported by Lageju *et al.* [6].

This case has been reported in line with the 2020 SCARE guidelines [7].

## Case presentation

A 71-year-old male from Kathmandu having chronic kidney disease (CKD) who underwent repetitive hemodialysis via an A-V fistula came to our institution with a history of generalized weakness and fatigability for 5 months. The disease was gradual in onset and associated with episodes of shortness of breath progressing from being present during exertion to even resting. He had a history of passing through black tarry stool for 3 months.

The patient had no history of similar complaints. He had started treatment and hemodialysis for the CKD 8 years before he was admitted at our institution and is still under treatment. His last hemodialysis session prior to presentation was 3 days back.

He had a history of diabetes and hypertension for 10 years, which was controlled by medication, and had no significant family history. His past surgical history revealed appendectomy.

At the time of presentation, the patient was alert, well oriented to time, place, and person and was not in a toxic state of health. He had signs of pallor with bilateral pitting edema. Vitals showed an elevated blood pressure of 180/100 mmHg.

On further investigation, the hemoglobin level was 7.3 gm%, with a total count of 3840 cells/mm$^3$ and a platelet count of 152,000 cells/mm$^3$. His prehemodialysis serum urea level was 159 mg/dl, and his serum creatinine level was 11.58 mg/dl. He underwent one session of hemodialysis of 3.5 hours duration after admission, that removed 3 liters of ultrafiltrate. His posthemodialysis serum urea concentration was limited to 54 mg/dl and his serum creatinine concentration to 4.63 mg/dl. The liver function test depicted a total bilirubin of 0.94 mg/dl, with direct bilirubin of 0.17 mg/dl and indirect bilirubin of 0.77 mg/dl. Serum enzymes of alanine aminotransferase (ALT) was 17 U/l and aspartate aminotransferase (AST) 27.4 U/l. The concentration of serum albumin was 2.8 g/dl, along with a total protein concentration of 7.7 g/dl.

The patient's coagulation panel revealed prothrombin time of 16.3 seconds with an international normalized ration (INR) of 1.2. Similarly, the bleeding time was 3 minutes while the clotting time was 9 minutes.

A peripheral blood smear showed anisocytosis and microcytic, hypochromic cells mixed with normocytic normochromic cells in the presence of pencil cells. The stool was positive for occult blood. A consultation with a gastroenterologist was made to plan for an upper gastrointestinal endoscopy. The endoscopy revealed multiple linear erosions with polypoidal tissues, suggesting gastropathy with linear ectatic punctate lesions radiating from the body to the antrum of the stomach, as shown in Fig. 1a and b.

A diagnosis of the GAVE and erosive gastritis (Fig. 2) was made. Initial resuscitation with two pints of packed red blood cells was performed, and a triple regimen for

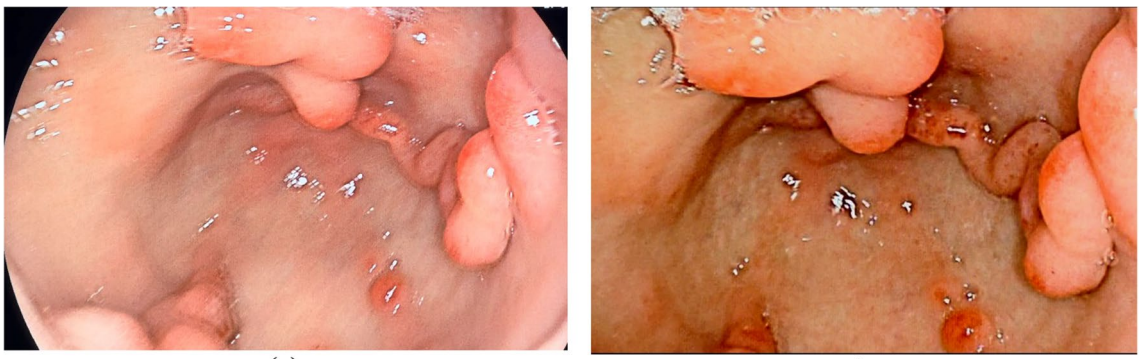


**Fig. 1** **a**, **b** Linear punctate lesion resembling a watermelon stomach

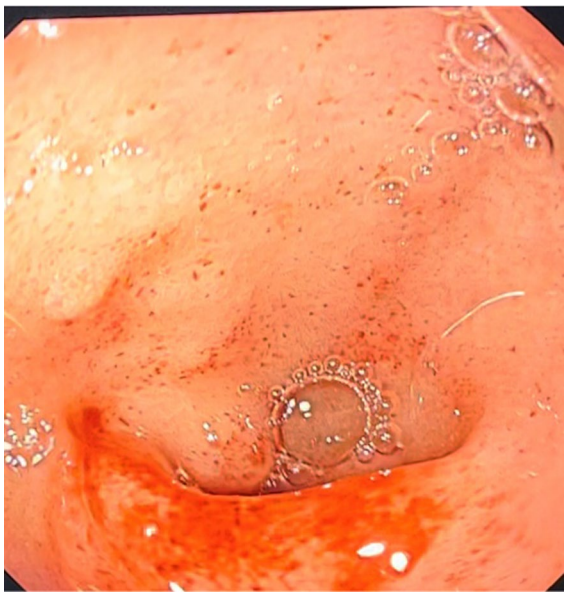

**Fig. 2** Erosive gastritis

the erosive gastritis with iron therapy was started. The patient was not under any anticoagulant and antiplatelet medications at the time of presentation and management at our institution.

Since the patient was financially burdened and we lack various facilities, he was scheduled for argon plasma coagulation at another institution.

## Discussion

GAVE is an uncommon but serious cause of upper gastrointestinal bleeding. Although typically found in the antrum, in rare cases, it may involve the duodenum, jejunum, and rectum [8]. A cohort study performed by Dulai *et al.* revealed that 26 out of 744 (4%) patients with nonvariceal gastrointestinal bleeding had a watermelon stomach [9]. Their study revealed that upper gastrointestinal bleeding occurred more extensively in patients having portal hypertension with a greater number of antral lesions causing more gastric bleeding than in those without portal hypertension [9]. The exact pathogenesis and cause of GAVE remain unclear, with studies suggesting that mechanical stress from strong gastric peristalsis causes prolapse, trauma, and intermittent obstruction of the mucosal blood vessels of the antrum, leading to ectasia [10]. As described earlier, GAVE is associated with underlying chronic medical conditions. It shows its association with end stage liver disease (ESLD) or cirrhosis, but the exact prevalence still needs to be sought out. Ward, *et al.* reported a prevalence of the disease to be 2.3% in patients with ESLD by undertaking a thorough esophagogastroduodenoscopy prior to having an orthotropic liver transplantation [11].

A study performed by Quintero *et al.* showed that high levels of serum gastric with consequent low levels of pepsinogen I were observed in cirrhotic patients with gastric mucosal ectasia [12]. A peculiar assessment of our patient showed that GAVE was incidentally reported during an endoscopy in a patient with the CKD. A combination of both CKD and GAVE in our patient may probably be the main reason behind anemia, as evidenced by low hemoglobin levels. Several hypotheses suggest the role of CKD in the pathogenesis of GAVE; one theory being that increased mechanical stress in uremia-induced weakening of gastric emptying leads to increased capillary pressure causing ectasia. Another theory states that decreased renal excretion of vasoactive mediators such as prostaglandin E2 and gastrin in patients with CKD results in GAVE [13].

Histological features revealed fibromuscular hypertrophy and capillary ectasia with microvascular thrombosis of the lamina propria [12]. Endoscopy is the investigation of choice for diagnosing GAVE, which typically shows two characteristic patterns: the punctate type observed more often in cirrhotic patients and the striped type observed in our patient [14].

GAVE can often be confused with antral gastritis and portal hypertensive gastropathy (PHG). PHG and GAVE can be distinguished by their specific locations; the PHG affects the fundus and has a mosaic pattern, while GAVE affects the antrum [15]. Similarly, GAVE can be differentiated from antral gastritis by its characteristic histological features of fibrin thrombus and ectasia [16].

The management of GAVE can be divided into medical, endoscopic, and surgical approaches. Medical management has been used to treat GAVE, but its long-term effectiveness is still debated. Some drugs that are used include corticosteroids, hormone-based therapy (estrogen-progesterone), octreotide, tranexamic acid, and thalidomide [17]. Endoscopic treatment with argon plasma coagulation (APC) has been used in the recent years to treat GAVE. Multiple treatment sessions are needed for APC to avoid recurrence. RFA is also regarded as an alternative to APC [18]. The surgical approach of an antrectomy provides no recurrence of postoperative bleeding [8]. Iguchi *et al.* reported that GAVE in patients with CKD can progress to remission through hemodialysis, and the recurrence of GAVE after hemodialysis is a rare event [13]. Our patient received medical treatment comprising of iron therapy and a triple regimen and was planned for APC.

## Strength and limitations of our study

This case report is the second reported case from Nepal despite challenges encountered due to lack of various facilities for the management of GAVE. Due to patient's financial strain, further diagnostic evaluation could not be performed. Argon plasma coagulation (APC) could not be performed due to lack of respective facilities, and hence, the patient was scheduled at an another institution, which is one of the limitations of our study. Therefore, we bring attention to the proper work up and management of GAVE by bringing forth the required resources and facilities.

## Conclusion

GAVE is an uncommon but serious cause of upper gastrointestinal bleeding in the elderly population. Its exact pathogenesis has not been fully elucidated; although, several hypotheses have been proposed in various literature. It is often misdiagnosed and can be incidentally found during upper gastrointestinal endoscopy. Although APC via endoscopy is the best treatment modality, GAVE can be symptomatically managed through medical approach by applying iron therapy and other drugs, as in our patient.

**Acknowledgements**
We thank the patient and his family for allowing us to study this case.

**Author contributions**
All the authors contributed equally for the preparation of this case report. Authors AA and KB were involved in conceptualization, study design, previous literature review, collection of all the information from the patient with endoscopy photographs, laboratory reports, requisition of written consent, and preparation of manuscript and editing. AB performed endoscopy and editing of the draft. All the authors individually did final proofreading of the manuscript before submission.

**Funding**
There are no sources of funding.

**Availability of data and materials**
Not applicable.

## Declarations

**Ethics approval and consent to participate**
Not applicable.

**Informed consent**
Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal upon request.

**Competing interests**
There are no competing of interest.

Received: 30 December 2023 Accepted: 8 August 2024
Published online: 15 October 2024

**References**

1. Stefanidis I, Liakopoulos V, Kapsoritakis AN, *et al*. Gastric antral vascular ectasia (watermelon stomach) in patients with ESRD. Am J Kidney Dis. 2006;47(6):e77–82. https://doi.org/10.1053/j.ajkd.2006.02.185.
2. Rider JA, Klotz AP, Kirsner JB. Gastritis with veno-capillary ectasia as a source of massive gastric hemorrhage. Gastroenterology. 1953;24(1):118–23. https://doi.org/10.1016/S0016-5085(53)80070-3.
3. Jabbari M, Cherry R, Lough JO, Daly DS, Kinnear DG, Goresky CA. Gastric antral vascular ectasia: the watermelon stomach. Gastroenterology. 1984;87(5):1165–70. https://doi.org/10.1016/S0016-5085(84)80080-3.
4. Alkhormi AM, Memon MY, Alqarawi A. Gastric Antral Vascular Ectasia: a case report and literature review. J Transl Int Med. 2018;6(1):47–51. https://doi.org/10.2478/jtim-2018-0010.
5. Calamia KT, Scolapio JS, Viggiano TR. Endoscopic YAG laser treatment of watermelon stomach (gastric antral vascular ectasia) in patients with systemic sclerosis. Clin Exp Rheumatol. 2000;18(5):605–8.
6. Lageju N, Uprety P, Neupane D, *et al*. Gastric antral vascular ectasia (Watermelon stomach); an unusual cause of upper gastrointestinal bleeding in elderly: a case report. Ann Med Surg (Lond). 2022;82:104733. https://doi.org/10.1016/j.amsu.2022.104733.
7. Agha RA, Franchi T, Sohrabi C, Mathew G, Kerwan A, SCARE Group. The SCARE 2020 guideline: updating consensus surgical CAse REport (SCARE) guidelines. Int J Surg. 2020;84:226–30. https://doi.org/10.1016/j.ijsu.2020.10.034.
8. Fuccio L, Mussetto A, Laterza L, Eusebi LH, Bazzoli F. Diagnosis and management of gastric antral vascular ectasia. World J Gastrointest Endosc. 2013;5(1):6–13. https://doi.org/10.4253/wjge.v5.i1.6.
9. Dulai GS, Jensen DM, Kovacs TO, Gralnek IM, Jutabha R. Endoscopic treatment outcomes in watermelon stomach patients with and without portal hypertension. Endoscopy. 2004;36(1):68–72. https://doi.org/10.1055/s-2004-814112.
10. Unnikrishnan R, Vijayakumar P, Senan S, Paul G. Gastric antral vascular ectasia: an uncommon cause of anemia in elderly individuals. Dubai Med J. 2019;2(3):99–101. https://doi.org/10.1159/000501795.
11. Ward EM, Raimondo M, Rosser BG, Wallace MB, Dickson RD. Prevalence and natural history of gastric antral vascular ectasia in patients undergoing orthotopic liver transplantation. J Clin Gastroenterol. 2004;38(10):898–900. https://doi.org/10.1097/00004836-200411000-00013.
12. Quintero E, Pique JM, Bombi JA, *et al*. Gastric mucosal vascular ectasias causing bleeding in cirrhosis. A distinct entity associated with hypergastrinemia and low serum levels of pepsinogen I. Gastroenterology. 1987;93(5):1054–61. https://doi.org/10.1016/0016-5085(87)90569-5.
13. Iguchi A, James Kazama J, Komatsu M, *et al*. Three cases of gastric antral vascular ectasia in chronic renal failure. Case Rep Nephrol Urol. 2011;1(1):15–9. https://doi.org/10.1159/000332832.
14. Ito M, Uchida Y, Kamano S, Kawabata H, Nishioka M. Clinical comparisons between two subsets of gastric antral vascular ectasia. Gastrointest Endosc. 2001;53(7):764–70. https://doi.org/10.1067/mge.2001.113922.
15. Gostout CJ, Viggiano TR, Ahlquist DA, Wang KK, Larson MV, Balm R. The clinical and endoscopic spectrum of the watermelon stomach. J Clin Gastroenterol. 1992;15(3):256–63. https://doi.org/10.1097/00004836-199210000-00019.
16. Gilliam JH 3rd, Geisinger KR, Wu WC, Weidner N, Richter JE. Endoscopic biopsy is diagnostic in gastric antral vascular ectasia. The "watermelon stomach." Dig Dis Sci. 1989;34(6):885–8. https://doi.org/10.1007/BF01540274.
17. Hsu WH, Wang YK, Hsieh MS, *et al*. Insights into the management of gastric antral vascular ectasia (watermelon stomach). Ther Adv Gastroenterol. 2018;11:175628317747471. https://doi.org/10.1177/1756283X17747471.
18. Peng M, Guo X, Yi F, *et al*. Endoscopic treatment for gastric antral vascular ectasia. Ther Adv Chronic Dis. 2021;12:20406223211039696. https://doi.org/10.1177/20406223211039696.

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