

Gastrointestinal-Predominant Alpha-Gal Syndrome with Severe Eosinophilia in a Child: A Case Report from Sri Lanka

Tharindi Suriapperuma

svtp.nadeesha@kln.ac.lk

Faculty of Medicine

Nadisha Badanasinghe

Faculty of Medicine

Gihani Dineshika

Colombo North Teaching Hospital

Vindya Millavithana

Colombo North Teaching Hospital

Thakshila Vipulanayake

Colombo North Teaching Hospital

Sachith Mettananda

Faculty of Medicine

Case Report

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Abstract

Background

Alpha-gal syndrome (AGS) is an emerging IgE-mediated allergy caused by sensitization to galactose- α -1,3-galactose following tick bites. Although AGS typically presents with delayed urticaria or anaphylaxis after ingestion of mammalian meat, isolated gastrointestinal manifestations are increasingly recognized and may lead to delayed diagnosis. Reports in children, particularly from South Asia, remain limited.

Case presentation

A previously healthy 4-year-old Sri Lankan girl presented with recurrent abdominal pain for three months and vomiting undigested food particles with associated gastrointestinal manifestations. Investigations revealed marked peripheral eosinophilia ($8,506/\text{mm}^3$), while abdominal ultrasonography and serological investigations for parasitic infections were unremarkable. Symptoms persisted despite empirical anti-helminthic therapy and symptomatic treatment. Further history revealed an episode of delayed anaphylaxis after pork ingestion and multiple tick bites. Her diet was rich in dairy. Skin prick testing showed strong sensitization to red meat, gelatine, and cow's milk, while alpha-gal-specific IgE was strongly positive (29 kUA/L), confirming AGS. Following elimination of alpha-gal containing foods her gastrointestinal symptoms resolved completely with normalization of eosinophil counts within one month.

Conclusions: This case highlights AGS as an important differential diagnosis in children with unexplained recurrent gastrointestinal symptoms and eosinophilia, particularly when there is a history of tick exposure.

Background

Alpha-gal syndrome (AGS) is an emerging form of food allergy characterised by immunoglobulin E (IgE) antibodies directed against the oligosaccharide galactose- α -1,3-galactose (alpha-gal), a carbohydrate found in non-primate mammalian meat and related products. Sensitisation typically occurs following bites from certain tick species, which introduce alpha-gal-containing molecules and trigger the development of specific IgE antibodies. Unlike classical food allergies, AGS is notable for its delayed onset of symptoms, usually occurring 3–6 hours after ingestion of mammalian meat such as beef, pork, or lamb. AGS has emerged as an increasingly recognised food allergy worldwide and is now being reported with growing frequency among both adults and children (1).

AGS most commonly manifests with urticaria, angioedema, and anaphylaxis, and presentation with predominant gastrointestinal symptoms is rare. The gastrointestinal phenotype of AGS could include recurrent abdominal pain, vomiting, diarrhoea, nausea, and bloating, often without obvious cutaneous or

respiratory features. Such presentations can mimic other gastrointestinal disorders and may delay recognition of the underlying allergy (2).

Although AGS has been increasingly reported worldwide, paediatric cases remain relatively uncommon, and the gastrointestinal phenotype in children is particularly underrecognized. In some patients, gastrointestinal symptoms may be accompanied by peripheral eosinophilia, suggesting an allergic inflammatory process involving the gastrointestinal tract. However, reports describing AGS with prominent gastrointestinal manifestations and peripheral eosinophilia are limited, especially in children (3). Here, we report a child with AGS presenting primarily with gastrointestinal symptoms and marked eosinophilia, highlighting the diagnostic challenges and the need for greater awareness of this atypical presentation.

Case presentation

A four-year-old previously well Sri Lankan girl presented with recurrent episodes of abdominal pain of three months' duration. The abdominal pain was in the central and epigastric area, which was aching in nature. It recurred on almost all days and lasted several hours per day. She also complained of frequent episodes of vomiting, consisting of small amounts of undigested food particles several times per day over the past three weeks. She had early satiety and poor weight gain over the preceding three months. There was no change in bowel habits. She had been on symptomatic treatment with omeprazole, domperidone, and antacids; however, there was no improvement.

On examination, her weight was 17.1 kg (between median and +1SD), height was 110 cm (between +1SD and +2SD), and weight-for-height was between median and - 1SD. She was not pale or icteric and did not have ankle oedema or lymphadenopathy. Her abdomen was soft and non-tender, and there were no masses or organomegaly. Her cardiovascular, respiratory and nervous system examinations were normal.

Her full blood count revealed leucocytosis (21,590/mm³), marked eosinophilia (8,506/mm³) with normal haemoglobin and platelet counts. Peripheral blood smear demonstrated severe eosinophilia with otherwise normal cellular morphology. The abdominal ultrasonography was normal. Her C-reactive protein was 3.3 mg/dL, and erythrocyte sedimentation rate was 15 mm in the first hour. Serological tests for *Toxoplasma*, *Toxocara*, and filarial infections were negative. Upper gastrointestinal endoscopy was arranged but could not be performed due to limited resources.

Due to the presence of severe eosinophilia, she was treated empirically with albendazole for five days while continuing symptomatic management with omeprazole, domperidone, and antacids. Persistence of abdominal pain despite treatment led to the commencement of a trial of mebeverine for possible abdominal pain-predominant irritable bowel syndrome. However, she continued to experience daily abdominal pain and vomiting, with only a minimal reduction in eosinophil count two weeks after completion of albendazole therapy.

Further detailed history revealed that she had previously experienced an episode of urticaria, cough, and shortness of breath approximately three hours after ingestion of pork. This episode was managed as anaphylaxis with intramuscular adrenaline, nebulised adrenaline and salbutamol, and an intravenous bolus of 0.9% sodium chloride. She had previously consumed pork at around two years of age without any adverse reactions, although red meat was not a regular component of her diet. Her diet was rich in dairy products, including fresh milk, cheese, and yoghurt. Her mother also recalled multiple events of tick bites in the past.

In view of the history of delayed anaphylaxis following red meat ingestion despite previous tolerance and the background history of multiple tick bites, AGS was suspected as the cause for recurrent abdominal pain. Dairy products may have contributed to symptom persistence because they formed a substantial component of the child's diet. A skin prick test was arranged, which showed strong positive reactions to red meat, gelatine, and cow's milk. The wheal size was larger with raw meat compared with commercial allergen extracts, a finding highly supportive of AGS. The alpha-gal-specific IgE test was strongly positive with a titre of 29 kUA/L, confirming the diagnosis of AGS.

Following diagnosis, an elimination diet excluding red meat, dairy products, and gelatine was initiated. There was a dramatic improvement in her abdominal pain and vomiting, and her early satiety resolved. One month after dietary modification, peripheral eosinophilia completely resolved (Table 1).

Table 1
Trend of peripheral blood counts during evaluation and management

Parameter	At presentation	2 weeks after elimination diet	1 month after elimination diet
Total WBC (cells/mm ³)	21,590	12,790	6,010
Neutrophils (cells/mm ³)	4,426	3,290	3,140
Lymphocytes (cells/mm ³)	7,664	6,580	2,610
Eosinophils (cells/mm ³)	8,506	2,270	260

Discussion

AGS was first described in 2009 and is increasingly recognised as an important cause of food allergy worldwide. Despite growing awareness, AGS remains under-recognised, and delayed diagnosis is common particularly in children. AGS commonly presents with urticaria, angioedema, and anaphylaxis and presentation with predominant gastrointestinal symptoms without classical allergic features is rare. Such presentations in children could lead to diagnostic delays, as the symptom profile closely mimics common gastrointestinal disorders (2).

Several studies have reported that patients sensitized to alpha-gal may present with isolated gastrointestinal symptoms, including abdominal pain, nausea, vomiting, and diarrhoea, without accompanying urticaria or anaphylaxis however, most of them are adults. McGill et al. reported that isolated gastrointestinal distress occurred in approximately 40% of patients who developed symptoms during oral food challenge with mammalian meat (3). Similarly, another study described 75 patients with a “GI-variant” of AGS, who presented predominantly with gastrointestinal symptoms and were found to have alpha-gal sensitization. Many of these patients experienced significant clinical improvement following elimination of alpha-gal-containing foods (4).

Further evidence supporting this phenotype was provided by Wilson et al., who reported that 32.3% of patients presenting with unexplained abdominal pain, diarrhoea, nausea, or vomiting had detectable alpha-gal specific IgE. Among these patients, 82% experienced improvement in gastrointestinal symptoms after avoiding mammalian food products. These findings highlight the importance of considering AGS in patients presenting with unexplained chronic gastrointestinal symptoms (5).

The patient described in this case report presented with recurrent abdominal pain followed by frequent vomiting of undigested food particles. These symptoms lead to the initial differential diagnoses of gastroesophageal reflux disease, irritable bowel syndrome, and functional gastrointestinal disorders. However, the persistence of symptoms and the presence of severe peripheral eosinophilia prompted further investigations to identify the underlying cause. The history of delayed anaphylaxis following pork ingestion, together with a background history of tick bites, positive skin prick test, high alpha-gal specific IgE and complete resolution of gastrointestinal symptoms following elimination of alpha-gal containing foods, confirmed the diagnosis.

Dairy products and gelatine may have contributed to symptom persistence in this patient because they were consumed regularly and contain alpha-gal. Although AGS is classically associated with reactions to mammalian meat, alpha-gal is also present in dairy products, and approximately 10% of patients with AGS may react to dairy (1).

Our patient had marked peripheral eosinophilia with GI manifestations, raising the possibility of eosinophil-associated gastrointestinal inflammation (6, 7). Sensitisation to alpha-gal commonly occurs following tick bites, which induces the production of alpha-gal-specific IgE antibodies. Upon re-exposure, alpha-gal triggers mast cell activation and the release of inflammatory mediators, promoting eosinophil recruitment and activation through interleukin (IL)-4, IL-5, and IL-13, raises the possibility of eosinophil-rich inflammation within the gastrointestinal tract (1).

The main limitation in this report is that endoscopic and histopathological evaluation was not performed to confirm EGID, as the patient improved soon after dietary modification.

Management of AGS primarily involves strict avoidance of mammalian meat, meat-derived products, dairy products, and gelatine and take all precautions to prevent tick bites (1). A progressive decline in eosinophil count was observed after initiation of the elimination diet in our patient, with normal levels

reached within 1 month. This clear temporal relationship between dietary modification and haematological improvement strongly supports an allergic aetiology consistent with AGS.

To our knowledge, this is among the first reported paediatric cases of gastrointestinal-predominant alpha-gal syndrome from Sri Lanka. This case showcased several important clinical points, including an atypical presentation of gastrointestinal phenotype, the potential role of dairy as the triggering allergen, and the association with peripheral eosinophilia in AGS.

Conclusion

This case report highlights the importance of considering AGS in children presenting with unexplained gastrointestinal symptoms and peripheral eosinophilia, even in the absence of typical allergic manifestations. Recognition of the gastrointestinal phenotype of AGS may help reduce diagnostic delays and prevent unnecessary investigations.

Declarations

Ethics approval and consent to participate: Written informed consent from the mother.

Consent for publication: Written informed consent for publication of this case and the accompanying image was obtained from the mother. The copies of the written consent forms would be available on request for review by the Editor-in-Chief of this journal.

Availability of data and material: Not applicable

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Author's contributions: TS, GD, VM, TV and NB participated in making the diagnosis and management of the children. TS, GD, VM, TV, NB and SM wrote the manuscript. All authors reviewed and approved the final manuscript.

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References

1. Commins SP. Diagnosis & management of alpha-gal syndrome: lessons from 2,500 patients. *Expert Rev Clin Immunol.* 2020;16(7):667-677.
2. Propst SBH, Thompson DK. Alpha-gal syndrome and the gastrointestinal reaction: a narrative review. *Front Allergy.* 2025;24; 6:1535103.
3. McGill SK, Levin ME, Shaheen NJ, Cotton CC, Platts-Mills TA, Commins SP. Gastrointestinal-isolated Distress is Common in Alpha-gal Allergic Patients on Mammalian Meat Challenge. *J Clin*

Gastroenterol. 2024. 1;58(1):80-84.

4. Richards NE, Makin TA, Smith AR, Platts-Mills TAE, Richards RD, Wilson JM. The α -Gal mammalian meat allergy as a cause of isolated gastrointestinal symptoms. *Front. Gastroenterol.* 2022;1:987713.
5. Richards NE, Richards RD Jr. Alpha-Gal Allergy as a Cause of Intestinal Symptoms in a Gastroenterology Community Practice. *South Med J.* 2021;114(3):169-173.
6. Amil-Dias J, Oliva S, Papadopoulou A, Thomson M, Gutiérrez-Junquera C, Kalach N, Orel R, Auth MK, et al. Diagnosis and management of eosinophilic esophagitis in children: An update from the European Society for Paediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN). *J Pediatr Gastroenterol Nutr.* 2024;79(2):394-437.
7. Papadopoulou A, Amil-Dias J, Auth MK, Chehade M, Collins MH, Gupta SK, Gutiérrez-Junquera C, Orel R, et al. Joint ESPGHAN/NASPGHAN Guidelines on Childhood Eosinophilic Gastrointestinal Disorders Beyond Eosinophilic Esophagitis. *J Pediatr Gastroenterol Nutr.* 2024;78(1):122-152.

Figures



Figure 1

Image showing the result of the skin prick test of the child. N- negative control (0.9%NaCl), RP- raw pork, RB- raw beef, PE- pork extract (commercial), G- gelatine, M- milk, P- positive control (1% histamine).