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CASE REPORT

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Alpha-gal syndrome—Food or drug allergy: A case report

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Key Clinical Message

Alpha-gal syndrome is an immunoglobulin E-mediated hypersensitivity characterized by delayed allergic reactions to ingested products containing alpha-gal carbohydrate. We present a patient with recurrent urticaria and suspected repaglinide hypersensitivity, who was eventually diagnosed with alpha-gal syndrome, wanting to emphasize possible drug allergy misdiagnosis and required caution with the medication choice.

KEYWORDS

alpha-gal, case report, drug hypersensitivity, food hypersensitivity, red meat, tick bite

1 | INTRODUCTION

Alpha-gal syndrome is an immunoglobulin E (IgE)-mediated hypersensitivity to galactose-alpha-1,3-galactose (alpha-gal), a carbohydrate found in all nonprimate mammalian tissues.[1,2] Tick bites sensitize people to alpha-gal in tick saliva. After years of tolerating mammalian meat, the initial onset is usually in adulthood.[2–4] Most patients have cutaneous (itching, urticaria, and angioedema) and/or gastrointestinal symptoms, although anaphylaxis is also frequent.[2,5] Unlike other food allergies, responses to mammalian meat products (beef, pork, and lamb) are delayed 3–6 hours and often occur at night. Intraindividual variability causes inconsistent reactions to an exposure; hence, patients can sometimes tolerate red meat.[6] Thus, the illness is hard to identify and might be mistaken as unexplained anaphylaxis, persistent spontaneous urticaria, or irritable bowel syndrome.[1,7,8] To emphasize the importance of proper diagnosis, allergic reactions may not only be limited to red meat but also to other products containing alpha-gal, such as excipients in some drugs or vaccines, recombinant human proteins, monoclonal antibodies, bioprosthetic heart valves, sutures, or other animal-derived agents.[3,6]

We present the case of a 70-year-old hunter with a history of multiple tick bites who presented with recurrent urticaria and angioedema, earlier interpreted as drug allergy.

This case report followed CARE guidelines.[9]

2 | CASE PRESENTATION

Recurrent urticaria and suspected repaglinide hypersensitivity brought a 70-year-old man to our clinic. His previous medical conditions included arterial hypertension,

type 2 diabetes, and persistent rhinitis. Patient had a history of several tick bites due to hunting and dog-owning in continental Croatia (Zagreb). Metformin, insulin glargine, lercanidipine, and pantoprazole were his chronic medications.

Seven years before referral, urticaria occurred 6–8 h after ingesting simethicone, regarded as an allergic reaction. Two months later, 2 weeks after a tick bite, the second incident occurred without a clear cause. The patient also had generalized pruritus without rash after using tamsulosin. After eating sausages, he often had a slight urticarial rash. Notably, he regularly ingested red meat (beef, pork, and lamb) that had been cooked or roasted without developing symptoms.

Three short-lived urticarial eruptions preceded by fresh tick bites occurred in the month preceding the first referral to our center. The first two instances happened 6–8 hours after taking repaglinide and responded quickly to therapy. The last urticarial episode, accompanied by facial angioedema, began a day before referral. Urticaria worsened after using fexofenadine for symptom management. There were no respiratory, cardiovascular, or gastrointestinal symptoms.

The physical examination revealed generalized urticaria and left-sided facial angioedema. Chloropyramine and methylprednisolone quickly relieved all symptoms.

Laboratory testing showed elevated leukocyte count with neutrophilia, lymphopenia, and elevated C-reactive protein (CRP). In the following weeks, a comprehensive diagnostic workup revealed raised total IgE (tIgE), specific IgE (sIgE) to alpha-gal (47% of total IgE) and slightly higher blood tryptase, but without c-KIT D186V mutation (Table 1). Skin prick tests with gelatine tested positive, while those with house dust mites, cat and dog dander, molds, tree, grass, weed pollens, and milk tested negative. Serology tests revealed positive immunoglobulins M and G to Epstein–Barr virus (EBV; Table 2). Among tumor markers, elevated levels of chromogranin

TABLE 1 Overview of initial laboratory findings.

Laboratory findings		Reference interval
Complete blood count		
Erythrocytes	4.84×10^9/L	$4.34–5.72\times10^9$/L
Leukocytes	*10.6×10^9/L*	*$3.4–9.7\times10^9$/L*
Neutrophils	*9.62×10^9/L*	*$2.06–6.49\times10^9$/L*
Lymphocytes	*0.72×10^9/L*	*$1.19–3.35\times10^9$/L*
Eosinophils	0.09×10^9/L	$0–0.43\times10^9$/L
Basophils	0.01×10^9/L	$0–0.06\times10^9$/L
Monocytes	0.14×10^9/L	$0.12–0.84\times10^9$/L
Thrombocytes	241×10^9/L	$158–424\times10^9$/L
Inflammatory markers		
C-reactive protein	*21.8 mg/L*	*0–5 mg/L*
Erythrocyte sedimentation rate	8 mm/3.6 ks	3–23 mm/3.6 ks
Allergy tests		
Serum tryptase	*11.2 mcg/L*	*<11 mcg/L*
Total IgE[a]	*519 kIU/L*	*<130 kIU/L*
Alpha-gal antigen IgE[a]	*245.1 kIU/L*	*<0.35 kIU/L*
c-KIT D186V mutation	Negative	
Tumor markers		
Chromogranin A	*557 mcg/L*	*<102 mcl/L*
Carcinoembryonic antigen, cancer antigen 19–9, alpha-fetoprotein, prostate-specific antigen, neuron-specific enolase, cytokeratin 19 fragment 21–1, beta-2-microglobulin	Normal	
Electrophoresis of serum proteins with immunofixation of monoclonal protein	No abnormalities detected	

Note: The italic font emphasises values that are not in reference intervals.

[a]Immunoglobulin E.

TABLE 2 Overview of microbiological findings.

Microbiological findings	
Blood serology tests	
Toxoplasma gondii	IgG[a] positive
Epstein–Barr virus	*EBNA*[b] *IgG*[a] *positive*
	VCA[c] *IgG*[a] *positive*
	VCA[c] *IgM*[d] *positive*
Cytomegalovirus	IgG[a] positive
Hepatitis B	Negative
Hepatitis C	Negative
Human immunodeficiency virus	Negative
Borrelia burgdorferi	Negative
Parasitic infections	Negative
Stool analysis	
Parasitic infections	Negative
Helicobacter pylori	Negative
Perianal tape impression	Negative
Urethral swabs	
Chlamydia trachomatis	Negative
Ureaplasma urealyticum	Negative
Mycoplasma hominis	Negative

[a]Immunoglobulin G.
[b]Epstein–Barr virus nuclear antigen.
[c]Viral-capsid antigen.
[d]Immunoglobulin M.

A were identified, significantly decreasing after the pantoprazole was discontinued for 3 weeks (Table 1). Radiologic examination, which included chest X-ray, abdomen and peripheral lymph node ultrasound, revealed no pathology.

Based on medical history and diagnostic testing, the patient was diagnosed with alpha-gal syndrome. It was recommended he avoided all mammalian meat, gelatine- and animal-protein-containing drugs and new tick bites. He was instructed to take loratadine for urticaria and prednisone for angioedema, and in case of an anaphylactic reaction, an epinephrine auto-injector was prescribed.

The patient experienced no urticaria or angioedema during the three-month follow-up. Despite avoiding dry meat products, he consumed cooked red meat and reported no new tick bites. In follow-up laboratory tests, tIgE and sIgE decreased significantly (Figure 1). Tryptase, CRP, and complete blood count were within the reference interval.

At the four-month follow-up appointment, the patient reported no urticaria or other symptoms and no additional tick bites. He avoided red meat consumption entirely, and sIgE to alpha-gal decreased further (Figure 1).

3 | DISCUSSION

Alpha-gal syndrome was originally identified in 2008 in individuals with hypersensitivity responses to cetuximab, a cancer treatment monoclonal antibody.[3,10,11]

Despite its rising incidence, it is often undiagnosed, causing patient suffering, possibly life-threatening responses, increased healthcare use, and emergency department visits.[1] Flaherty et al. found that 21% of symptomatic alpha-gal syndrome individuals were detected within a year after the first presentation, whereas the remaining 79% took 7.1 years. Of over 100 medical contacts, fewer than 10% had an accurate diagnosis or successful workup.[11]

Our patient's path was similar. As a result of being misdiagnosed with various medication reactions, it took 7 years to diagnose him. Reactions depend on the meat's lipid content and alpha-gal concentration, most robust in innards like the kidney. Cooking meat decreases fat content without denaturing the alpha-gal antigen, which lessens the severity of responses.[2,6] Furthermore, the typical nocturnal onset of symptoms due to delayed reactions may obscure the identification of milder reactions.[6,12] Our patient consistently ate cooked red meat without allergic responses and exclusively responded to sausages with pork gut casings containing more alpha-gal. He had "consistently inconsistent" symptoms, which is practically a sign of alpha-gal syndrome.[6]

Cofactors such as exercise, nonsteroidal anti-inflammatory medications, and alcohol may also contribute to the variability of allergic reactions. In addition, it is well known that acute viral infections can facilitate allergic reactions.[13] Serology tests of our patient indicated a recently recovered EBV infection or reactivation (Table 2), which may have contributed to allergic responses. Pan et al. investigated the possible role of EBV infection in promoting IgE-mediated egg allergy. They showed that EBV-specific antibodies (EBNA-1 IgG) and two viral miRNAs were highly expressed in patients with egg allergy compared with controls and positively correlated with the level of egg-specific IgE.[14] EBV can induce the proliferation of B lymphocytes and the production of polyclonal antibodies. A possible mechanism behind the triggering of allergic reactions is the production of interleukin 4 and 5 by human B cells transformed by EBV, which stimulates the eosinophilic inflammatory response and production of IgE antibodies.[15] Our patient had no clinical symptoms of EBV infection; however, silent infection or reactivation is possible. EBV DNA testing was unfortunately not performed. Neutrophilia and lymphopenia at the first referral may be explained by glucocorticoid treatment and mildly elevated CRP levels by recent viral infection.

Chromogranin A elevation was suspected to be a consequence of long-term proton-pump inhibitor (PPI) therapy (pantoprazole). According to Pregun et al.,[16] the value of serum chromogranin A increases even after short-term proton-pump inhibitor therapy, but the most prominent increase was observed in patients treated with long-term regimen. Significant decrease in chromogranin A value is expected quickly after PPI cessation, which was also observed in our patient after 3 weeks of PPI cessation.[16]

Our patient is unique in that he developed urticaria that was more intense after taking certain medicines than after consuming dry meat. Furthermore, the primary cause for referral to an allergy expert was the investigation of a potential medication allergy.

Heparins, monoclonal antibodies (cetuximab and infliximab), abatacept, pancreatic enzymes, thyroid hormones, and antivenom contain alpha-gal.[6]

Additionally, responses to some medications are considered to be caused by excipients containing alpha-gal, such as gelatine, stearic acid, lactic acid, or glycerine. Our patient, sensitized to gelatine, had an allergic reaction to gelatine in tablets or capsules, even though significant allergic reactions to gelatine were usually induced by parenteral exposure (through plasma expanders or immunizations). Moreover, gelatine is a hidden heat-stable food allergen, and it can be found in shampoos, suppositories, collagen implants, sutures, and contact lenses.[3,12] Stearic acid is added to tablets as magnesium stearate to facilitate lubrication during production. It may be synthesized from animal-derived organic fatty acids (cows, pigs, and sheep) with alpha-gal contamination or plant-based cocoa butter and shea butter. Thus, people can tolerate plant-derived magnesium stearate but respond to animal-derived one. Lactic acid and glycerine can also be derived from either animal or plant sources and may contain alpha-gal if they are animal-derived. However, it is not sure if these products intrinsically contain alpha-gal or it could be present as contaminant.[3,17]

Since manufacturers do not provide the entire pedigree of each medication, alpha-gal patients cannot anticipate medicine allergies from ingredient lists.[3,6,18,19] Our patient developed urticaria to gelatine and magnesium stearate medications (Table 3). Magnesium stearate is also an excipient in fexofenadine (antihistamine), which may explain why the drug may have worsened urticaria. The patient frequently took and tolerated metformin with magnesium stearate, possibly due to its plant origin. In addition, patients with alpha-gal syndrome taking metformin in preparation for bariatric surgery could reintroduce dairy and red meat into their diets, possibly due to the modification of cytokine response and antigen reprogramming caused by the metformin.[6,20]

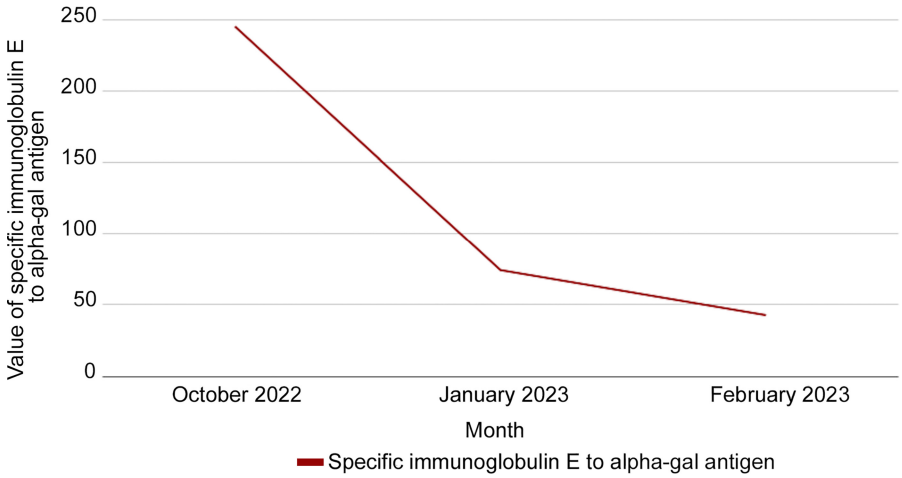

FIGURE 1 Value of specific IgE to alpha-gal antigen through time.

TABLE 3 Timeline and details of allergic reactions to drugs.

Year	Drug	Suspected culprit agent	Time of reaction onset after ingestion	Type of allergic reaction	Treatment
2015	Simethicone	Gelatine	6–8 h	Urticaria	Loratadine
2019	Tamsulosin	Gelatine	4–6 h	Pruritus	None
2022	Repaglinide	Magnesium stearate	4–6 h	Urticaria/Angioedema	Chloropyramine, methylprednisolone
2022	Fexofenadine	Magnesium stearate	4–6 h	Urticaria	Chloropyramine, methylprednisolone

The fact that only tick bites may cause sensitization or increase in sIgE levels may be explained by salivary adjuvants that stimulate an immune reaction, skin epithelial barrier damage and the process of wound healing that promotes Th2 immune response leading to B cell proliferation and antibody production.[4,21] The presence of sIgE to alpha-gal can be maintained or augmented by subsequent tick bites. Commins et al. demonstrated a 20-fold increase in sIgE following tick bite, accompanied by an increase in tIgE.[19] Avoiding tick bites decreases sIgE in most people, with variable reduction rate and still unknown sIgE levels that indicate tolerance to alpha-gal products. Platts-Mills et al. observed patients whose alpha-gal sIgE titers turned negative and who successfully reintroduced meat into their diet.[17] The fact that different tick species differ in their capacity to induce or enhance sIgE production explains why some patients noticed a decrease in alpha-gal sIgE levels despite receiving repeated tick bites.[22] The ratio of alpha-gal sIgE to tIgE may also be responsible for allergic reactions. Commins et al. described the case of an individual with elevated levels of alpha-gal sIgE and tIgE following a tick bite but with a sIgE/tIgE ratio less than 1%, who did not experience any allergic reactions to mammalian meat.[19]

In continental Croatia, where our patient lives, Ixodes ricinus is the most widespread tick species, which was reported to contain alpha-gal in the midgut.[4,23] After repeated tick bites in our patient, alpha-gal sIgE levels probably increased, causing more severe symptoms. Over the follow-up period without tick bites, alpha-gal sIgE levels decreased (Figure 1), despite the patient eating cooked red meat, which aligns with previous reports.[2,5,17,19,22]

Mammalian meat consumption has not been shown to increase alpha-gal sIgE production in previously sensitized people. No research has examined how mammalian meat exclusion diet affects sIgE levels in alpha-gal syndrome sufferers.[17]

Alpha-gal syndrome can mimic chronic spontaneous urticaria or irritable bowel syndrome. Hence, a thorough diagnostic workup is needed to exclude infections, neoplastic illnesses, and allergies.[1,7,8,24] Systemic mastocytosis increases anaphylaxis risk, and serum tryptase and c-kit mutation should be tested.[2]

Avoiding foods, medications, and other products with alpha-gal antigen might be difficult due to the absence of labeling of mammalian-derived sources.[3,6] Most alpha-gal syndrome individuals can tolerate dairy products. However, in case of inadequate symptom control, individuals are suggested to avoid dairy products. Eighty percent improve with abstinence from mammalian meat products only, whereas another 15% improve with the elimination of dairy products. Despite avoidance, some people still have allergy symptoms. Their diet should be additionally altered because certain food additives include alpha-gal.[6,25]

Despite tolerating cooked red meat, we advised our patient to avoid any red meat. We wanted to minimize the danger of severe allergic responses, which might vary based on the kind of meat, alpha-gal content, and cofactors. Since the patient recalled a few bouts of urticaria without a trigger, recall bias should be considered. Due to the considerable gap between eating and symptoms, the history may be inaccurate. The negative prick-to-prick test with fresh milk advised against dairy product avoidance.

Like our patient, some alpha-gal syndrome sufferers do not react to all red meat. Without cofactors, it is unclear whether these individuals should regularly consume tiny amounts of red meat to maintain tolerance.[2] Patients who accepted dairy products were more likely to remit. Hence, oral immunotherapy with cow's milk has been explored. Further research is needed to determine alpha-gal syndrome immunotherapy options.[2,25]

4 | CONCLUSION

Alpha-gal syndrome is a novel condition that largely remains unrecognized. Allergist referral is necessary considering that few centers have expertise diagnosing it. It may be a differential diagnosis for recurring or chronic urticaria, angioedema, gastrointestinal complaints, idiopathic anaphylaxis, or even drug hypersensitivity. Allergic responses vary and often occur 6–8 hours after eating mammalian meat or innards. Detailed history should include past tick bites, possible triggers including foods and drug excipients, consistency, and exposure-related allergic responses.

This case report aimed to educate healthcare practitioners about varied symptoms of alpha-gal syndrome, possible misdiagnosis of drug allergy, and required caution with the choice of medications in patients with confirmed diagnosis. Additionally, alpha-gal or mammalian content labeling in drugs, other medical products, and food is recommended.

AUTHOR CONTRIBUTIONS

Marina Božan: Conceptualization; data curation; investigation; resources; writing – original draft; writing – review and editing. **Vesna Vukičević Lazarević:** Conceptualization; data curation; investigation; resources; writing – review and editing. **Ivan Marković:** Conceptualization; resources; visualization; writing – review and editing. **Jadranka Morović-Vergles:** Validation; writing – review and editing. **Joško Mitrović:** Validation; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors have no conflict of interest to declare.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

CONSENT

Written informed consent was obtained from the patient to publish this case report in accordance with the journal's patient consent policy.

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