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# Mycotoxin-Triggered Attacks of Nausea, Vomiting, and Abdominal Pain and Episodes of Pseudo-Obstruction

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## Abstract

*Determining the etiology of episodic abdominal pain, nausea and vomiting with and without pseudo-obstruction and implementing effective treatment can be challenging. Mycotoxins activate mast cells which rapidly degranulate releasing pro-inflammatory cytokines. Mast cells commonly reside in the gastrointestinal mucosa and adjacent to nerves. Aberrant mast cells with loss of control due to genetic abnormalities are present in mast cell activation syndrome, a common, yet often unrecognized multisystemic disorder. Mold exposure with consequent toxicity by its mycotoxins can present with complex multisystem disorders along with abdominal pain, nausea, and vomiting. A 63-year-old man presented with episodic attacks of abdominal pain, nausea and vomiting when he was exposed to dwellings with mold. Over a 4-year period he was admitted and there was radiographic evidence of dilation of the stomach and small intestine during three admissions and dilation of the colon in the other admission. When the patient was subsequently diagnosed and treated for underlying mast cell activation syndrome, the attacks ceased, and he has been healthy for the last three years. Recognition that mycotoxins can act as triggering factors is essential to effectively treat patients with and without mast cell activation syndrome who have these gastrointestinal attacks and episodic gastrointestinal pseudo-obstruction.*

**Keywords:** *Abdominal Pain, Gastroparesis, Ileus, Mast Cell, Mast Cell Activation Syndrome, Mold, Mycotoxins, Nausea, Pseudo-Obstruction, Vomiting*

## INTRODUCTION

Attacks of abdominal pain, nausea and vomiting may be difficult to diagnose and treat [1]. Quality of life is poor and economic impact is often significant for those afflicted with nausea [2]. Treating and determining the etiology of intestinal pseudo-obstruction is often difficult [3]. Indoor exposure to molds and mycotoxins is generally unrecognized as causes for nausea in both patients with and without mast cell activation syndrome (MCAS) [4-7]. The most common variant of mast cell activation disease (MCAD) is MCAS, and this presents with many inflammatory and allergic symptoms and systemic syndromes [8]. In Germany, the prevalence of MCAD was estimated to be 17% of the population [9]. Systemic mastocytosis (SM) is a well-known MCAD that is malignant and rare (incidence of 0.89 cases per 100,000 persons per year) [10]. The prevalence of nausea in a case series of 413 MCAS patients was 57% [5]. The prevalence of nausea in a case series of 83 SM patients was 23% [11].

Indoor mold exposure can be associated with nausea [12-14]. Lieberman et al reported that 16/48 (33%) consecutive mold-affected patients had nausea [12]. Hyvonen et al reported 24/47 (51%) students in a water damaged school had nausea as compared to 2/56 (4%) students in a control school ($p<0.001$) [15].

Mast cells are immune cells that are present in most tissues that surround blood vessels and nerves, and are found in the skin, lung mucosa, and digestive tract, as well as in the oral cavity, nose, and conjunctiva [16]. Mold contamination in buildings is quite common [17-19]. Molds produce numerous secondary metabolites known as mycotoxins [20]. Exposure to mycotoxins has been linked to activation of mast cells, and IgE antibodies to mycotoxins stimulate mast cells to release heparin, histamine, pro-inflammatory cytokines, and prostaglandin D2 [21,22]. Signs of this release include neurological symptoms such as brain fog, headaches, nausea, fatigue, and irritation of the respiratory tract [23-25]. Recent publications show that this stimulation by IgE antibodies to mycotoxins in serum can lead to Mast Cell Activation

A wide variety of infections and toxic exposures are triggers for activation of MCAS [18-24]. Mycotoxins can be absorbed via nasal mucosa, lungs, gastrointestinal track and skin and readily pass the blood-brain barrier [26-31]. Molds release

mycotoxins which activate normal mast cells (MCs) [25] as well as unregulated MCs in MCAS (authors personal observations). Indoor mold exposure has been linked to MC degranulation, secretion of cytokines and chemokines, neutrophil recruitment, and increased intestinal permeability [32-34].

## CASE REPORT

A 63-year-old Caucasian male property manager presented with a 4-year history of frequent attacks of nausea, vomiting and abdominal pain. These attacks occurred during work when he inspected houses that had water damage (Figure 1). He had four hospitalizations for the most severe of his numerous attacks and each showed an abnormal computerized tomography (CT) scan. In January 2016, there were dilated loops of small intestine and fluid in a distended stomach. In September 2016, the large intestine was dilated with gas and fluid. In August 2018, the jejunum and proximal to mid ileum were dilated (Figure 2) and he underwent exploratory surgery. The operative report described dilated loops of small intestine without evidence of a transition point or adhesions. Finally in January 2020, he had a small bowel ileus which required prolonged nasal gastric tube decompression. After 8 days, a diatrizoate meglumine and diatrizoate sodium solution study via a nasogastric tube demonstrated dilated small bowel loops and a 5-hour transit time to the cecum.

**Figure 1.** Moldy ceiling in one of the houses that the patient inspected.

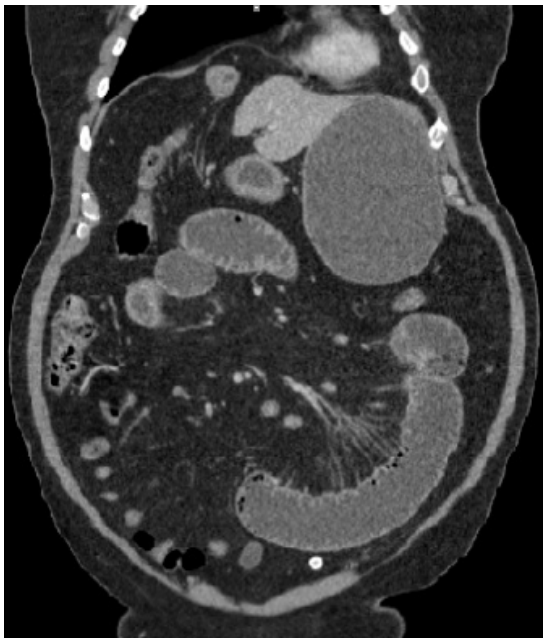

**Figure 2.** Computerized tomography (CT) image after 4 days of pain, vomiting and diarrhea with first emergency room evaluation. CT demonstrated a fluid-filled, enlarged stomach and mildly dilated and fluid-filled loops of the mid and proximal small intestine.

After discharge he sought a second opinion. A detailed review of medical records including the operative report, review of systems, childhood and teenage medical history, and family history was performed. The mast cell mediator release syndrome (MCMRS) questionnaire (Appendix A) symptom score was 15 in six of ten systems ("diagnostic criteria of a mast cell mediator release syndrome are clinically confirmed") [9].. His most severe symptoms included gastrointestinal symptoms, chronic fatigue, headaches, episodic chest pain, and dyspnea. Mild tinnitus was present for 10 years. He also had near fainting spells when working in moldy homes. His parents and siblings did not have undiagnosed medical syndromes or symptoms. His daughter has hypermobile Ehlers-Danlos syndrome and autonomic dysfunction with orthostatic intolerance. Physical examination showed that he was short of breath moving from the chair to the examining table. His lungs were clear to auscultation. The chest radiograph was normal. The abdomen exam was normal. A partial MCAS work up that measured four blood mediator tests revealed an elevated histamine level (3.9 ng/mL; normal ≤1.8). Urine mediator testing was not performed.

The patient has had significant improvements in all chronic symptoms with MC-directed therapy: histamine-1 and histamine-2 receptor antagonists, vitamins C and D, and low dose naltrexone [35,36]. He had no further GI attacks unless he went into a particularly severely moldy house without wearing a mask. He subsequently changed jobs and had no symptoms. In 2024 he had a brief admission for similar symptoms that occurred when he tried to remove moldy drywall from his own garage.

## DISCUSSION

Nausea and vomiting are common problems in MCAS patients and can have a significant impact on quality of life as demonstrated in this report. Activation of MCs by a variety of infections (bacteria, rickettsia, fungi, parasites, and viruses) occurs by direct and indirect mechanisms [33,37]. The role of mold and mycotoxin in MCAS is not well known in general medicine. Mold and mycotoxins activate normal MCs which lead to pro-inflammatory cytokines such as prostaglandin D2, histamine, and antibody production [32,33,38,39]. Stimulation of dysregulated, mutated MCs in MCAS patients activates normal MCs which further amplifies the effect.

Exposure to molds and mycotoxins can present with a multi-systemic illness in people without other diseases [40]. The commonly accepted presentation of mold toxicity is limited to asthma and sinusitis, but lately it has been expanded to include a wide array of multisystemic symptoms [12,40]. Nausea has been noted as a complaint in approximately 15% of 1,000 patients who were positive for mycotoxin antibodies (Andrew W. Campbell, M.D., personal observations). Autoimmune antibody response to molds and mycotoxins with subsequent demyelination and autoimmune nerve degeneration has been demonstrated [41,42]. One report linked two cases of new-onset chronic inflammatory demyelinating polyneuropathy (CIDP) to heavy indoor mold exposure [42]. Theoretically this could be caused by central or peripheral nervous system inflammation. To explain nausea, demyelination of the vagal nerve or enteric nervous system could lead to subsequent abnormal gastrointestinal motility. Alternatively, central nervous system inflammation in various areas of the brain could lead to nausea. Pro-inflammatory cytokines have been demonstrated in various brain regions in response to mycotoxins in a mouse model [43]. Theoretically, release of mediators by mast cells adjacent to gastrointestinal parasympathetic nerves could lead to spontaneous ileus. Spontaneous bloating is common in MCAS [6].

This case reports illustrates the severe impact of mold exposure and mycotoxins in a patient with previously undiagnosed MCAS. There was radiographic and surgical evidence of gastroparesis, small intestinal pseudo-obstruction, and colonic pseudo-obstruction. His acute attacks improved by having the patient wear a mask during his work and taking MC-directed medical therapy. Ultimately, he changed to a different job and remained healthy.

There are inherent problems in the diagnosis and treatment of mold-related diseases. There are no studies comparing immunoglobulin testing versus urine tests. There are no double-blind, placebo-controlled treatment studies. The benefit of using immunoglobulin testing is the specificity of the immune response [44]. A study by Vojdani et al reported that levels of antibodies to Penicillium notatum, Aspergillus niger, Stachybotrys chartarum, and Satratoxin-H were significantly higher in 500 patients exposed to high levels of mold as compared to 500 unexposed controls ($P<0.001$ for all 4 comparisons) [44].

Nausea can be a serious consequence of mold and mycotoxin exposure. MC activation by mycotoxins is a driver of inflammation [38]. Worsening of MCAS by mycotoxins has been observed by practitioners, but not reported in the literature to our knowledge. More studies are needed to determine the relationships between mold, mycotoxins, and their effects on MCs and MCAS.

This case is remarkable for the severe and lengthy clinical presentation with supporting radiographic evidence. The climate crisis is responsible for increasingly causing flooding of homes and business places which increases the risk of heavy mold exposure [45]. The impact of mycotoxins in the development of gastrointestinal and systemic disorders is not well appreciated among physicians and other medical professionals. Gastrointestinal symptoms may be the primary presentation of mycotoxin diseases.

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## APPENDIX 1.

MAST CELL MEDIATOR RELEASE SYNDROME QUESTIONNAIRE

Name: _____ Birth date: _____ Date: _____

Answer all of the following symptoms/questions, even if they are only slightly bothersome, rarely occurring (for instance, not necessarily present currently but in the past), or may seem not be related to your main problems.

Do you get colds regularly which then turn into bacterial nfections such as bronchitis or sinus infections? ..... Yes___ No___

Has the course of your illness been episodic (and/or with attacks)? ....Yes___ No___

Have the symptom-free periods become shorter and shorter? .....Yes___ No___

If the statement applies to you, check the box and then enter the intensity level on the line next to the box. The number should be graded when it was present the last time it occurred. If the statement does not apply, do not check the box or enter a number.

Use the range of 1 (very mild)to 10 (unbearable) to reflect the level of your discomfort. Example: ☑ 6

**The following symptoms occur repeatedly or may be constant:**

| **GENERAL HEALTH** | **Applies** | **Intensity** |
|---|---|---|
| Significant physicalweakness or fatigue doing everyday activities. | ☐ | _____ |
| Extreme fatigue attacks, it can be hard to keep eyes open ..... | ☐ | _____ |
| At times I lose weight despite maintaining my normal diet ..... | ☐ | _____ |
| **EYES, EARS, NOSE, MOUTH** | | |
| Ears have ringing or odd sounds ..... | ☐ | _____ |
| Eyesare dry, itchy,red,burning, or feel gritty ..... | ☐ | _____ |
| Runny or stuffy nose ..... | ☐ | _____ |
| Inflammation or ulcers of the mouth ..... | ☐ | _____ |

**Score 1 for any of the 4 symptoms ___**

| | | |
|---|---|---|
| **CHEST and HEART** | | |
| Burning and/or pressure pain in the chest..... | ☐ | _____ |
| (Normal electrocardiogram or stress test; or not severe enough to go to ER) | | |
| Rapid heart rate (rapid palpitations) ..... | ☐ | _____ |
| Redness or flushing of the skin, especially face or upper body ..... | ☐ | _____ |

**Score 2 if positive___**

| | | |
|---|---|---|
| Hot flashes (usually with dry skin lasting 2 to 5 minutes, rarely 10minutes and often occur with nausea or other symptoms. ..... | ☐ | _____ |
| (these are not menopausal hot flashes with wet sweats) **Score 2 if positive ___** | | |
| Sudden dizziness/lightheadedness withfainting or near faint..... | ☐ | _____ |

**Score 2 if positive ___**

| | | |
|---|---|---|
| **LUNGS** | | |
| Irritable dry cough or need to cough ..... | ☐ | _____ |
| Feeling of shortness of breath or difficulty taking a full breath ..... | ☐ | _____ |
| Asthma-like complaints (wheezing) ..... | ☐ | _____ |

**Score 1 if one or more is present ___**

**ABDOMEN**

Attacks of visible bloating or distension within minutes..... ☐ _____

Pain in the abdomen ..... ☐ _____

Pain is burning ..... ☐ _____

Pain is crampy or spastic ..... ☐ _____

Pain is associated with diarrhea (watery or loose stool) ..... ☐ _____

Nausea (with or without vomiting) ..... ☐ _____

Do antihistamines help reduce nausea (examples: Allegra, cetirizine,

Claritin, diphenhydramine, Xyzal, Zyrtec, etc.)? .....Yes___ No___

(this does not include nausea relief from Zofran or Ondansetron)

**URINE/PELVIS**

Bladderand/or pelvicpain (this applies to women and men) and is often associated withpainful, frequent and/or urgent urination and may be associated with pain during sex. ..... ☐ _____

(during these times bacterial cultures and urine analysis are normal)

**NEUROLOGIC and MUSCULOSKELETAL**

Migraine-like headaches (throbbing on one side only or have

been diagnosed as a migraine – (these are NOT tension headaches).. ☐ _____

Brain fog – word finding problems and/or concentration

difficulties with or without associated insomnia episodes ..... ☐ _____

Leg or arm pain and/or altered feelings including

numbness, tingling, burning, sharp pain, and pins and needles. ..... ☐ _____

(this does not respond to over-the-counter pain medicine)

**SKIN**(see the Photographic Examples at end)

Hives (red raised itchy spots) ..... ☐ _____

Hemangiomas (raised or flat bright red spots)..... ☐ _____

During attacks there are itchy skin lesions that look like acne in the

corners of thenasal-lip area, as well as, the chin and forehead ..... . ☐ _____

Knots or nodulesunder the skin ..... ☐ _____

Painless, non-itchy swelling (especially lips, cheeks, eyelids) ..... ☐ _____

Itching in area around the anus during attacks..... ☐ _____

**HEMATOLOGIC**

Bruising afterminor injuries ..... ☐ _____

Unusual nose bleeds..... ☐ _____

Women only: significant menstrual bleeding ..... ☐ _____

**Score 1 if one or more is present __**

**Are any** of the symptoms or complaints listed above worsened by:

High histamine foods (such as alcohol, cheese, chocolate,

tuna, cured fish/meat, left-over meat, raisins, tomatoes,...).. ☐ _____

Sleep deprivation (awake for more than 24 hours)..... ☐ _____

Hunger or fasting (no food all day)..... ☐ _____

**BONE**

Bone pain that usuallyoccursin more than one bone…….. ☐ _____

**Score 1 if present __**

Bone density test showed osteoporosis or osteopenia

without a known cause …….. ☐ _____

and/or

Whole-body nuclear scintigraphy showed areas of

increased bone metabolism without a known cause …….. ☐ _____

**Score 1 if one or more is present __**

_____

**Laboratory Data**

At least once during the disease phases there was:

| | **Applies** |
|---|---|
| Hyperbilirubinemia up to about 2.5 mg% with the exclusion of Meulengracht/Gilbert's syndrome or another hereditarydisorders | ☐ |
| Increase in transaminases: | |
| γGT and/or | ☐ |
| ALT and/or | ☐ |
| AST and/or | ☐ |
| Score 1 if one or more is present. | ☐ **1** |
| *AST increased >10 fold (subtract 1 point and look for other diseases)* | ☐ ***-1*** |
| Hypercholesterolemia (patient must be normal or underweight) | ☐ **1** |
| Low titer autoantibodies without a corresponding organ symptom | ☐ **1** |
| **Mast cell mediators:** | |
| Tryptase in serum was normal | ☐ **0** |
| Tryptasewas marginally increased | ☐ **3** |
| Tryptaseincreased >2 times the upper limit | ☐ **10** |
| Histamine in plasma was normal | ☐ **0** |
| Histaminewas marginally increased | ☐ **3** |
| Histamineincreased >2 times the upper limit | ☐ **10** |
| Prostaglandin D2 in plasma was normal | ☐ **0** |
| Prostaglandin D2 was marginally increased | ☐ **3** |
| Prostaglandin D2increased >2 times the upper limit | ☐ **10** |
| Heparin and/or factor VIII in plasma was/were normal | ☐ **0** |
| Heparin and/or factor VIII was/were elevated (and bleeding disorders were excluded). | ☐ **3** |
| Chromogranin-A in serum was normal | ☐ **0** |
| Chromogranin-Awas increased (and other causes were excluded) | ☐ **3** |
| Leukotriene E-4 in urine was normal | ☐ **0** |
| Leukotriene E-4was marginally increased | ☐ **1** |
| Leukotriene E-4was10 times the upper limit | ☐ **5** |
| Leukotriene E-4was >10 times the normal limit | ☐ **10** |
| N-methylhistamine in urine was normal | ☐ **0** |
| N-methylhistamine was marginally increased | ☐ **1** |
| N-methylhistamine was10 times the upper limit | ☐ **5** |
| N-methylhistamine was >10 times the normal limit | ☐ **10** |

| | |
|---|---|
| 2,3 dinor 11b PG F2 alpha in urine was normal | □ **0** |
| 2,3 dinor 11b PG F2 alpha was marginally increased | □ **1** |
| 2,3 dinor 11b PG F2 alpha was10 times the upper limit | □ **5** |
| 2,3 dinor 11b PG F2 alpha was >10 times the normal limit | □ **10** |
| Other conspicuous laboratory findings (please name with values) | □ **0** |

_____

**Procedures and Imaging**

| | |
|---|---|
| Esophagogastroduodenoscopy or associated biopsies had: | |
| no pathological findings | □ **0** |
| or | |
| mild inflammation | □ **1** |
| or | |
| Helicobacter pylori-negative and NSAID-negative erosions | |
| and/or ulcers | □ **3** |
| or | |
| diffuse and/or focalmast cell infiltrates ≥20/hpf with rounded shape | □**5** |
| or | |
| Mast cell nests and/or sheets of spindle-shaped mast cells | |
| and/or CD25-positive mast cells | □ **10** |
| | |
| Colonoscopy and associated biopsies had: | |
| no pathological findings | □ **0** |
| or | |
| mild inflammation | □ **1** |
| or | |
| focal and/or disseminated dense infiltrates of | |
| morphologically inconspicuous mast cells | □**5** |
| or | |
| Mast cell nests and/or sheets of spindle-shaped mast cells | |
| and/or CD25-positive mast cells | □ **10** |

| | |
|---|---|
| Enlargement of the spleen | □ |
| and/or enlargement of the liver | □ |
| | □ **1** |

Diseases and disorders below should be excluded in order help confirm the presence of a mast cell disorder. Symptoms in some organ/tissue systems can be similar in both. Evaluate both checklistsand the numerical values listed to the right of each box. Add together to get a sum. The data should be entered by the physician.

**Sum 9 to 13** = pathological activation of mast cells as cause of complaint is assumed.

**Sum ≥14** = diagnosis of mast cell mediator release syndromeisclinically confirmed.

***Sum of points: ____Diagnosis: mast cell mediator release syndrome***

***5 or more systems involved may also be important.***

**Differential diagnosis and testing for disorders that are multi-systemic diseases, present with some similar symptoms, or exacerbate mast cell activation**

**Endocrine disorders** – all are multi-sytemic

Adrenal insufficiency (cosyntrophin stimulation test)

Diabetes mellitus (history and labs – Type 1 or 2 with systemic complications)

Endometriosis (gynecologic exam, laparoscopy)

Fabry disease(neuropathic pain, fatigue, angiokeratomas, cloudy cornea, genetic test)

Porphyria (lab: spot PBG urine)

Thyroid disorders – multi-systemic and can exacerbate MCAS (labs: thyroid panel)

**Gastrointestinal disorders**

Amyloidosis – multi-systemic (fat biopsy, rectal biopsy)

Celiac disease – multi-systemic and can exacerbate MCAS (antibody studies, endoscopic biopsy)

Cholecystitis (ultrasound, DISIDA imaging)

Chronic bacterial infection or post-infectious autoimmune state (fecal PCR, lactulose breath test) – multi-systemic and may exacerbate MCAS (endoscopic biopsy, urea breath test, antibody study)

Hepatitis multi-systemic (hepatitis profile, other laboratory tests)

Inflammatory bowel disease – multi-systemic (colonoscopy/ileoscopy and biopsy; video capsule endoscopy)

Lactose, sucrose, or fructose intolerance as cause of bloating and altered bowel habits (history, specific breath tests)

Median arcuate ligament syndrome – multi-system and can exacerbate MCAS (auscultation, CT angiography with deep expiration views)

Microscopic colitis associated with celiac disease or cryptosporidiosis - multi-systemic (colonoscopy biopsy, cryptosporidiosis antigen)

Parasitic infection – multi-systemic (fecal antigen testing, ova and parasite exam, PCR testing)

Small bowel obstruction with and without small intestinal bacterial overgrowth – multi-systemic (adhesions, volvulus, hernia, tumors) (history, physical, imaging studies, lactulose breath testing)

Small intestinal bacterial overgrowth – multi-systemic and exacerbates MCAS (lactulose breath test, glucose breath test, duodenal or jejunal aspirate culture)

**Immunological, Inflammatory and Rheumatologic diseases**– all multi-systemic

Anti-phospholipid syndrome (history, antibody tests)

Asthma and atopic diseases including chronic urticaria (history, allergy testing – skin prick and RAST – MC mediators, ESR, CRP, TFT, ANA, H. pylori testing, AST, stool tests for parasites and fungus)

*Chronic fatigue syndrome* (history)

Chronic pelvic pain syndromes [interstitial cystitis, type III chronic prostatitis, vulvadyndia] (cystoscopy and biopsy, prostatic secretion testing)

Familial Mediterranean fever (history, family history, ESR)

Fever of unknown etiology. (history, extensive lab testing)

Fibromyalgia (history, physical, ESR)

Food allergy/sensitivity (history, skin prick tests, RAST, special investigations of biopsies, elimination diet)

Heredity alpha 1 tryptasemia (tryptase level, genetic testing)

Hypereosinophilic syndrome (CBC with differential)

Hypermobile Ehlers Danlos syndrome (history, physical exam)

Hereditary angioedema (family history, C4 level, C1-esterase inhibitor level)

Juvenile rheumatoid arthritis (history, RF, ESR, radiographs)

Lupus erythematosus (history, ANA, ESR)

Sjogren's disease (antibody studies including novel or early Sjogren's antibodies)

Vasculitis (clinical picture, ANA, ESR, and other laboratory studies)

**Vascular disorders**

Median arcuate ligament syndrome

nutcracker syndrome

pelvic vascular compression syndrome

**Infectious diseases** – all multi-systemic

*Bartonella - can also exacerbate MCAS (*PCR and FISH tests*)*

Mold infection - c*hronic inflammatory response syndrome - can also exacerbate MCAS (urine aflatoxin test and serum antibody tests)*

Syphilis - *can also exacerbate MCAS*

*Tick borne infections - can also exacerbate MCAS (antibody panel)*

*Tuberculosis (blood and skin tests, radiographs)*

**Neoplastic diseases**

Carcinoid tumor – multi-systemic (24 hr urine 5-HIAA, CT and octreotide imaging)

Intestinal lymphomas multi-systemic (CT studies, endoscopic procedures)

Pancreatic endocrine tumors [gastrinoma, insulinoma, glucagonoma, somatostatin, VIPoma] – multi-systemic (lab determination, imaging studies, endoscopic ultrasound)

Pheochromocytoma – multi-systemic (laboratory studies, CT imaging)

Systemic mastocytosis (tryptase level, bone marrow biopsy)

**Neurologic diseases**

Cyclic vomiting syndrome (history)

Migraine (history)

Munchausen's syndrome (history)

Postural orthostatic tachycardia syndrome – multi-systemic and exacerbates MCAS (orthostatic pulse exam, tilt table test, autonomic testing)

Serotonin syndrome (history, serotonin level)

Small fiber peripheral neuropathy – multi-systemic (skin biopsy)

**Citation:** Leonard B. Weinstock, Andrew W. Campbell, Luke Curtis, Jordan Gutovich, "Mycotoxin-Triggered Attacks of Nausea, Vomiting, and Abdominal Pain and Episodes of Pseudo-Obstruction", Universal Library of Medical and Health Sciences, 2024; 2(1): 59-69. DOI: https://doi.org/10.70315/uloap.ulmhs.2024.0201008.