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Mast Cell Activation Disorders: Diagnosis & Management

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Abstract

Mast Cell Activation Disorder (MCAD) is a broad umbrella term which includes a heterogenous group of disorders characterized by the inappropriate and excessive release of mediators from mast cells. Mast Cell Activation Syndrome represents a severe and well-defined form within the broader spectrum of Mast Cell Activation Disorders. These disorders are generally divided into clonal and non-clonal categories. Clonal conditions include Systemic Mastocytosis, Cutaneous Mastocytosis, and Monoclonal Mast Cell Activation Syndrome, whereas non-clonal conditions comprise secondary MCAS, idiopathic mast cell syndromes, and Hereditary Alpha Tryptasemia. Considerable clinical overlap may occur between clonal and non-clonal presentations. Measurement of baseline serum tryptase (bST) plays an important role in diagnosing, categorizing, and monitoring patients with MCAS. Individuals with a bST concentration above 8 µg/L are commonly evaluated for HαT, which involves additional copies of the TPSAB1 gene, as well as the KIT D816V mutation. When the corrected bST value is higher than expected for the patient's genotype and is accompanied by compatible symptoms, the findings strongly indicate systemic mastocytosis. The initial management is similar in all subjects of MCAS. HαT positive individuals should undergo life-long immunotherapy if indicated. There are KIT targeting drugs like Avapritinib in systemic mastocytosis. This review highlights a practical framework for improving the understanding and clinical management of patients affected by Mast Cell Activation Syndrome.

Keywords: Mast Cell activation disorders (MCAD), mast cell activation syndrome (MCAS), hereditary alpha tryptasemia (HαT), serum tryptase, mastocytosis, cutaneous mastocytosis, monoclonal mast cell activation syndrome (MMAS), KIT D816V, TPSAB1 gene

INTRODUCTION

Mast Cell Activation Disorder (MCAD) is a broad umbrella term which includes a heterogenous group of disorders characterized by the inappropriate and excessive release of mediators from mast cells. Mast Cell Activation Syndrome (MCAS) is a specific, severe type of MCAD characterized by recurring, multi-system symptoms due to inappropriate mediator release (e.g., hives, diarrhoea, anaphylaxis). MCAS requires strict diagnostic criteria: symptoms in two or more systems, elevated mast cell mediators, and response to treatment. Mast cell disorders are classified into "clonal" versus "non-clonal" disorders. In "clonal disorders", there is well defined mutation as seen in [Systemic Mastocytosis (SM), cutaneous mastocytosis (CM), and Monoclonal Mast Cell Activation Syndrome (MMAS)] and In "non-clonal disorders", there is no such evidence of well-defined mutation, classified as Secondary Mast Cell Activation Syndrome (MCAS), Idiopathic MCS, and Hereditary alpha tryptasemia (HαT). Secondary MCAS is due to allergic or other hypersensitivity disorders in 2 or more organ systems such as skin, GIT, and airway. Symptoms worsen by predictable triggers (certain foods, temperature changes, stress, alcohol, medication, etc.). In addition, if there is no clonality and no other specific reason then, MCAS is accepted as idiopathic. There is a combined or mixed type of MCAS in which cases have both primary and secondary MCAS features as shown in (Figure 1).

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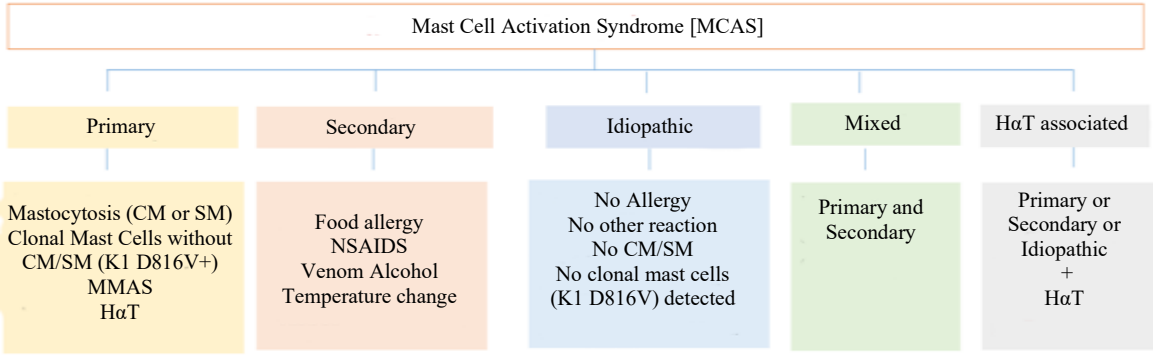

Figure 1. Classification of mast cell activation syndrome MCAS.

CLASSIFICATION OF MAST CELL ACTIVATION DISORDERS [MCAD] (TABLE 1)

- *Primary Mast Cell Activation Disorders:* They comprise a diverse range of conditions caused by abnormalities in mast cell precursor cells, leading either to excessive clonal proliferation or abnormal activation of mast cells. Clinical manifestations associated with primary Mast Cell Activation Disorders are mainly driven by mediator release and commonly include flushing, low blood pressure, abdominal cramps, vomiting, diarrhoea, and rapid heart rate [1, 2].
- *Secondary Mast Cell Activation Disorder:* In these patients, mast cell precursor cells are typically normal, and the overall mast cell count usually remains within the normal range. Nevertheless, mast cells become excessively sensitive to environmental or local stimuli, resulting in an exaggerated response. In such conditions, mast cell activation occurs through external mechanisms that are not directly driven by intrinsic mast cell abnormalities. Secondary forms of Mast Cell Activation Disorders include allergic conditions mediated by IgE or non-IgE pathways, physical urticarias, and chronic spontaneous urticaria. Clinical manifestations may occur intermittently or persist over time [3].
- *Idiopathic Mast Cell Activation Disorder:* This condition should be suspected in patients experiencing recurrent episodes of anaphylaxis, urticaria, or angioedema in whom no clonal disorder or identifiable mast cell-related cause can be established [3].

Systemic Mastocytosis (SM), Cutaneous Mastocytosis (CM), Hereditary Alpha Tryptasemia (HαT), and Monoclonal Mast Cell Activation Syndrome (MMAS).

Table 1. Classification of mast cell activation disorders (MCAD) [4, 5].

Primary mast cell disorders (increased numbers of identical mast cells [clones], or increased internal signalling of mast cells)	• Cutaneous mastocytosis. • More common in children, with more than 90% of cases resolving by adolescence. • Systemic mastocytosis. • Associated with gain-of-function mutations in proto-oncogene c-*KIT*, which has a role in proliferation and differentiation of mast cells. KIT D816V is detected in >90% of all cases, but other mutations may be implicated. • Mastocytoma. • Mast cell leukaemia. • Monoclonal mast cell activation syndromes (MMAS). • Present with symptoms of mast cell activation and lack cutaneous findings. Have either the KIT D816V mutation or CD25+ mast cells in their bone marrow. • Meet one or two minor diagnostic criteria for mastocytosis, but do not meet full criteria. • Hereditary alpha tryptasemia (HαT) – characterized by an increased number of copies of the *TPSAB1* gene, which leads to elevated baseline serum tryptase levels. Symptoms of Flushing, urticaria, pruritus, hypotension, tachycardia, syncope/presyncope, gastrointestinal symptoms.

Secondary mast cell disorders (normal mast cells and normal numbers, but "hyper-responsive" to external stimuli; have a condition that can induce mast cell activation)	• IgE-mediated hypersensitivity reactions. • For example, food, venom, drug-induced. • IgE-independent reactions (other receptors/pathways involved). • For example, vancomycin, opioids. • Mast cell hyperplasia. • associated with neoplasia, autoimmune conditions, chronic infections.
Idiopathic (no identifiable clonal or underlying mast cell pathology)	• Includes idiopathic anaphylaxis. • Includes mast cell activation syndrome (a syndrome defined by specific criteria).

CLINICAL SYMPTOMS IN MAST CELL ACTIVATION SYNDROME [MCAS]

Concerning clinical features may include flushing, unexplained episodes of low blood pressure, headaches, pruritus, skin eruptions, sensitivity to foods, medications or environmental factors, as well as various neuropsychiatric manifestations, as illustrated in Figure 2.

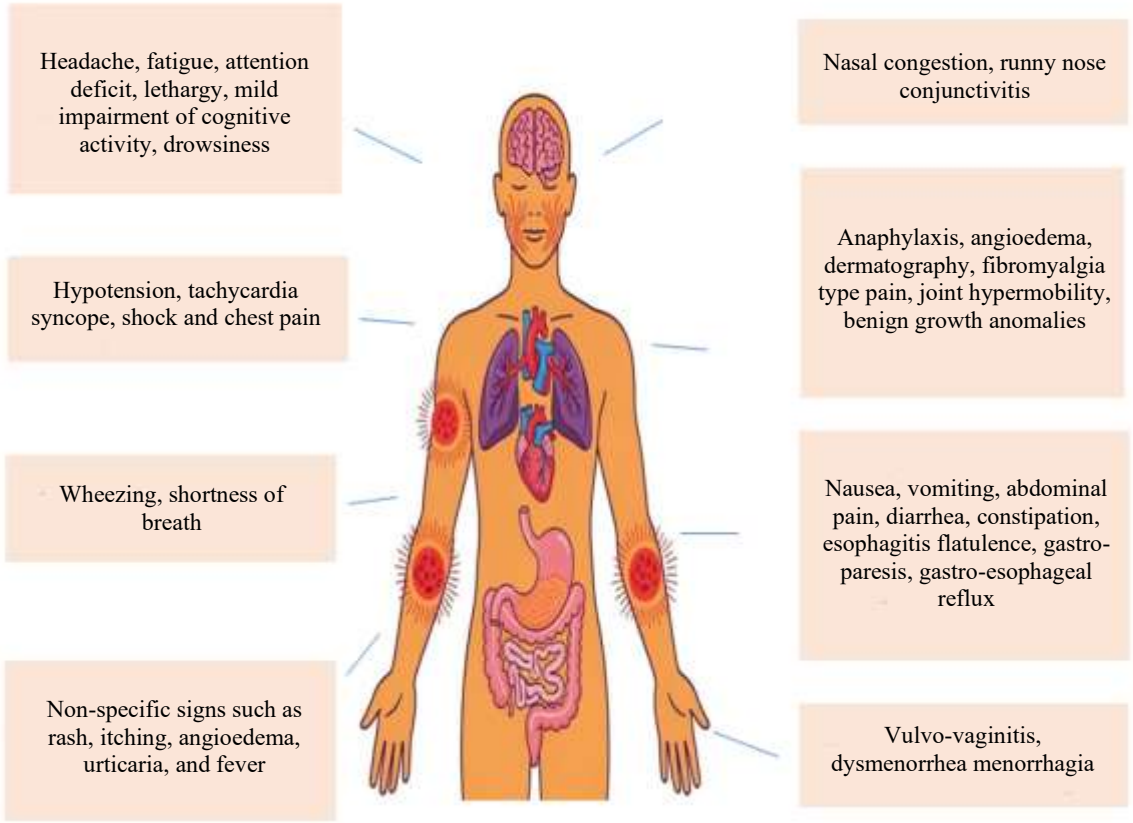

Figure 2. Clinical symptoms in MCAS.

DIAGNOSIS OF MCAS

To diagnose MCAS, following three criteria must be met (Figure 3) [6]:

- Characteristic findings include severe and recurrent episodes of systemic anaphylaxis, urticaria, and angioedema, with systemic involvement defined as manifestations affecting at least two organ systems.
- Mast cell involvement can be confirmed through serological evaluation by demonstrating an increase in serum tryptase levels to 120% of the patient's baseline value plus 2 ng/mL. Serum tryptase is considered the preferred biomarker for mast cell activation, and concentrations exceeding 11.4 µg/L are generally regarded as clinically significant. Additional mast cell-derived indicators, such as 24-hour or spot urinary histamine metabolites and prostaglandin D2 (PGD2) metabolites, may also support the diagnosis, although their practical clinical value remains

limited. Suggested diagnostic criteria for histamine or PGD2 metabolites include levels greater than 200% above the individual's baseline, provided the measured values also exceed the assay's normal reference range [7].

- Improvement of symptoms following treatment with mast cell-stabilizing medications, agents that inhibit mast cell mediator synthesis, or therapies that prevent mediator release or block the actions of mast cell-derived mediators support the diagnosis.

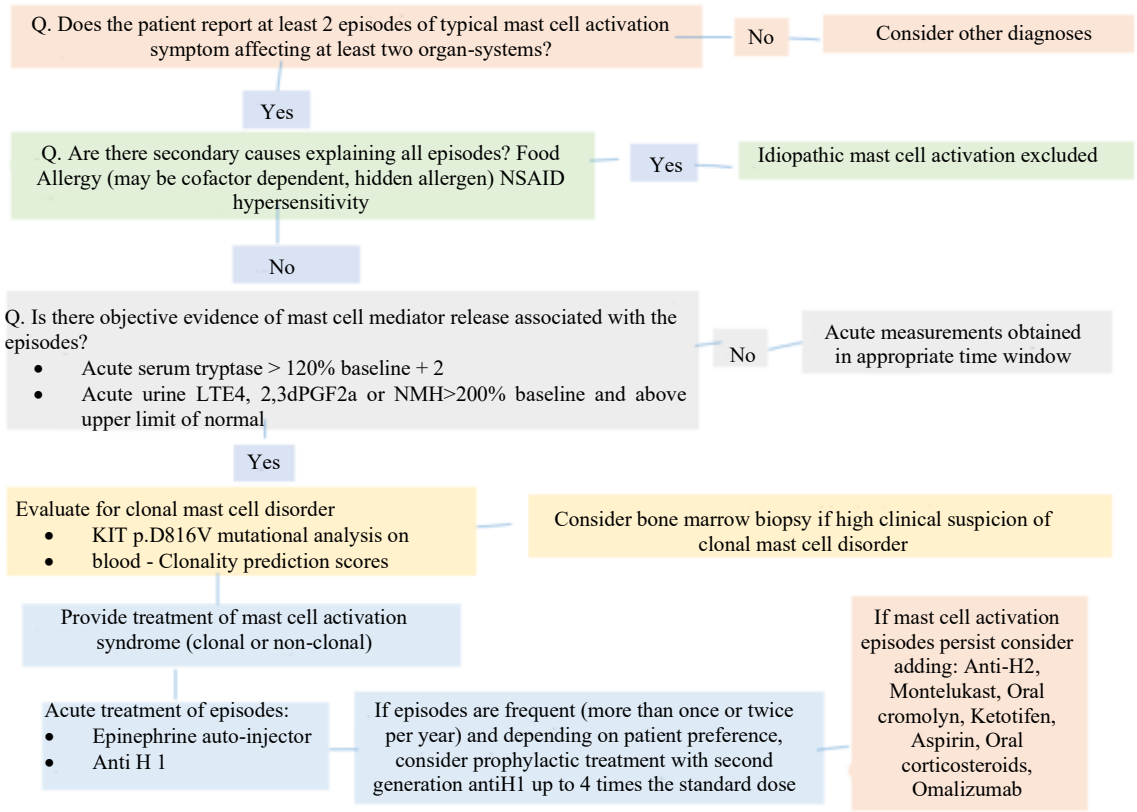

Figure 3. Flow-chart for diagnosis of MCAS.

Differential Diagnosis of MCAS

There is considerable overlap between the symptoms of MCAS and other clinical conditions as shown in Table 2.

Table 2. Differential diagnosis of MCAS.

Differential	Relevant signs and symptoms	Tests
Carcinoid syndrome	Flushing, diarrhoea, wheeze	Plasma 5-hydroxyindoleatic acid (HIAA). 24-hour urinary HIAA.
Phaeochromocytoma	Flushing, hypertension, tachycardia	Plasma metanephrines. 24 hr Urinary metanephrines and catecholamines.
Menopause	Flushing	FSH, LH, oestrogen.
Medullary carcinoma of thyroid	Flushing	Serum calcitonin.
Cardiac arrhythmias	Tachycardia, presyncope/syncope, hypotension	ECG.
Postural tachycardia syndrome	Tachycardia, presyncope/syncope, hypotension	Tilt table test.
Hereditary angioedema	Angioedema, throat tightness	C4+/C1 inhibitor levels and/or function.
Neuroendocrine tumors	May include flushing	Serum vasoactive intestinal peptide.

PRIMARY MCAS – MASTOCYTOSIS

Mastocytosis comprises a diverse group of neoplastic disorders characterized by abnormal proliferation and accumulation of clonal mast cells within different organ systems. The condition may occur as Cutaneous Mastocytosis or Systemic Mastocytosis, while a rare and highly aggressive localized variant is known as mast cell sarcoma (MCS). Cutaneous mastocytosis is more frequent in children with spontaneous regression in puberty, while Systemic mastocytosis is more frequent in adults and bone marrow biopsy is always needed for its diagnosis [8].

Mastocytosis – Symptoms

Occurrence of mediator-related symptoms is often acute and unpredictable (e.g., anaphylaxis). Mediator related symptoms can be present in all variants of mastocytosis regardless of severity and duration of the disease. Mast cell mediators are often dysregulated in patients with mastocytosis and are either dependent or independent from IgE mediated mechanism. They can appear at any time during the disease and their spectrum and intensity can modify over time. These symptoms are among the top concerns raised by patients and a major cause of poor quality of life in patients with mastocytosis (Figure 4).

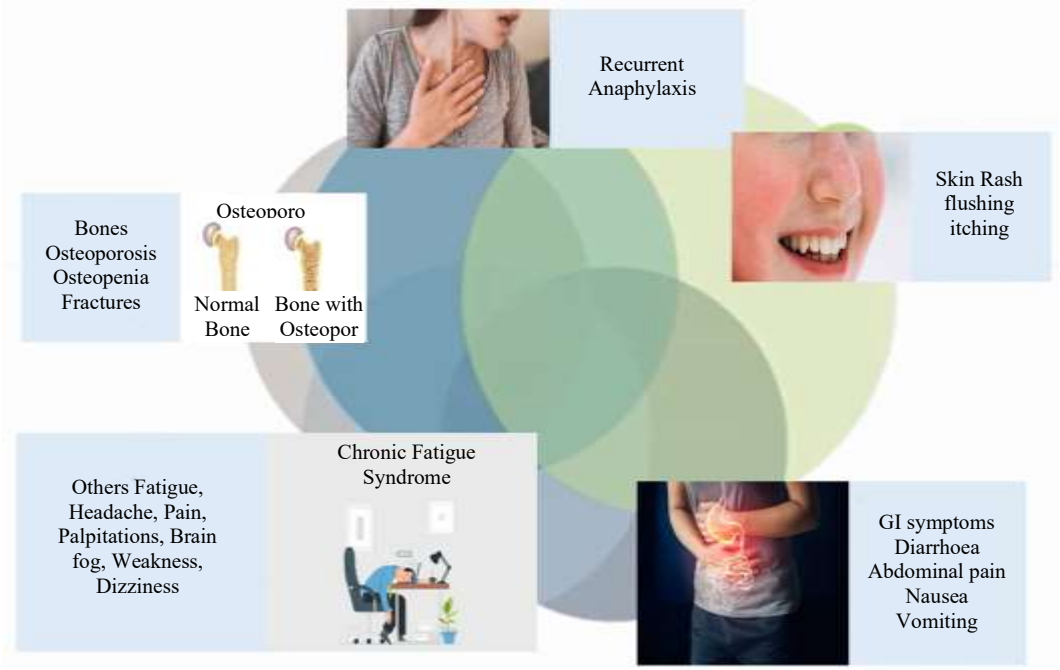

Figure 4. Symptoms in mastocytosis.

Diagnosis of Systemic Mastocytosis [SM]

Systemic mastocytosis can be divided into Non-advanced [Indolent SM (ISM), Bone marrow mastocytosis (BMM), Smoldering SM (SSM) and Advanced [SM with an associated haematological neoplasm (SM-AHN), Aggressive SM (ASM), and MC leukaemia (MCL)] (Table 3). In adults, most patients are diagnosed with SM regardless of the presence of absence of skin lesions. Anaphylaxis is a major issue in BMM, especially in cases diagnosed with a concomitant Hymenoptera venom allergy. The major and minor criteria is established in the diagnosis of SM [9].

Table 3. WHO criteria for classification and diagnosis of systemic mastocytosis [7].

Non-advanced	Advanced
Cutaneous Mastocytosis (CM)	SM with an Associated Hematological (Non-Mast Cell) Neoplasm (SM-AHN).
Indolent Systemic Mastocytosis (ISM)	Aggressive Systemic Mastocytosis (ASM).
Bone Marrow Mastocytosis	Mast Cell Leukemia (MCL).
Smoldering Systemic Mastocytosis (SSM)	Mast Cell Sarcoma (MCS).

Proposed Refined Major and Minor SM Criteria

Major Criterion

- Multifocal dense infiltrates of mast cells (>15 mast cells in aggregates) in bone marrow biopsies and in sections of another extracutaneous organ.

Minor Criteria

- >25% of all mast cells are atypical cells (type I or type II) on bone marrow smears or are spindle-shaped in mast cell infiltrates detected in sections of bone marrow or other extracutaneous organs.
- KIT-activating KIT point mutation at codon 816 or in other critical regions of KIT in bone marrow or another extracutaneous organ.
- Mast cells in bone marrow, blood, or another extracutaneous organ express one or more CD2 and CD25 and CD30.
- Baseline serum tryptase concentration >20 ng/mL (In the case of an unrelated myeloid neoplasm, an elevated tryptase dose does not count as an SM criterion. In known HaT, tryptase level should be corrected).

DIAGNOSIS

If at least 1 major and 1 minor or 3 criteria are fulfilled, the diagnosis is SM.

Mastocytosis and Bone Marrow Biopsy

Symptomatic patients presenting with bST >8 ug/L should be tested for HαT and PB KIT D816V and bone marrow biopsy should be done in those with positive PB KITD816V. The following are the established indications for bone marrow biopsy in mastocytosis:

• Patients with hypersensitivity reaction to Hymenoptera sting and High Level of Tryptase (with or without skin involvement).
• Patients with hypersensitivity reaction to Hymenoptera sting and Negative skin and serological tests.
• Patients with hypersensitivity reaction to Hymenoptera sting characterized by severe hypotension without urticaria/ angioedema also with normal basal levels of tryptase.
• Patients with hypersensitivity reaction to Hymenoptera sting after termination of venom immunotherapy.

Hereditary Alpha Tryptasemia (HαT)

Hereditary Alpha Tryptasemia is a relatively common autosomal dominant genetic condition associated with elevated baseline serum tryptase levels (generally >8 ng/mL) caused by additional copies of the TPSAB1 gene, which encodes alpha-tryptase. The condition affects approximately 5.2% of the general population. Patients with HαT who also mastocytosis have often experience mast cell activation symptoms, including hypotension and anaphylaxis, more frequently and with greater severity than individuals without HαT.

HαT is most frequently identified in middle-aged women, and several manifestations may overlap with menopausal hormonal changes, including hot flashes, muscle pain, joint discomfort, mood alterations, and sleep-related complaints. Clinical features of HαT are broadly categorized into five groups: cutaneous manifestations such as flushing, urticaria, angioedema, and pruritus; connective tissue abnormalities including joint hypermobility, retained primary teeth, and congenital skeletal defects; atopic conditions such as hymenoptera venom-induced anaphylaxis, eczema, asthma, and multiple allergies; gastrointestinal symptoms including irritable bowel syndrome, recurrent diarrhoea, abdominal pain, and gastroesophageal reflux disease; and neuropsychiatric manifestations such as dysautonomia, postural orthostatic tachycardia syndrome, chronic pain syndromes, fibromyalgia, vulvodynia, anxiety, depression, and sleep disturbances [9, 10].

Serum tryptase concentrations below 6 μg/L have not been reported in HαT. Most affected individuals exhibit baseline serum tryptase values above 8 μg/L, with nearly 90% showing levels greater

than 11.4 µg/L and approximately 20% demonstrating concentrations exceeding 20 µg/L. The suggested classification of serum tryptase levels in HαT is summarized in Table 4.

Table 4. Proposed classification of tryptase ranges [7].

Proposed term of tryptase range	BST range
Normal range where HαT is rather unlikely	1.0–8.0 ng/mL
Normal range when excluding most individuals with HαT	1.0–11.4 ng/mL
Clinical meaningful normal range that includes (asymptomatic)individuals with HαT	1.0–15.0 ng/mL
Hypertryptasemia	15.1–200.0 ng/mL
Excessive Hypertryptasemia	>200.0 ng/mL

MANAGEMENT OF MCAS

The wide variability in clinical presentations and associated coexisting conditions should be considered while managing patients with Mast Cell Activation Syndrome. At present, no treatment specifically targets the underlying genetic basis of the disease. Consequently, the management of Mastocytosis and Hereditary Alpha Tryptasemia remains primarily symptom-directed and supportive. Patient and caregiver education is essential, particularly regarding the avoidance of factors that may precipitate anaphylaxis, angioedema, or urticaria, as well as limiting exposure to histamine-rich foods, as illustrated in Table 5.

Table 5. Foods rich in histamine [11, 12].

Food types	High histamine foods
General	• Consuming fresh, minimally processed foods over ultra-processed foods.
	• Freezing leftover protein rich foods to retard histamine production.
Fruit	Avocado, citrus fruits, strawberries, kiwifruit, papayas, pineapples, dried fruits.
Vegetables	Tomatoes, spinach, eggplant.
Animal Protein	Fish: Mackerel, tuna, sardines, anchovies, herring, eggs.
	Aged beef, cured meats (salami, ham), leftover meats or fish.
Dairy	Aged cheeses: Cheddar, Gouda, Roquefort, Parmesan.
Alcohol	All, (Wine red > white).
Other	Nuts, chocolate, vinegar, fermented foods such as kimchi, sauerkraut.

Treatment Options for MCAS

- *In Addition to 2nd Gen. H1-AH (up to 4-Fold):* Sodium-Cromoglycate, Leukotriene antagonists
- *Instead of H2-AH:* Proton-pump-inhibitors
- *Anaphylaxis:* Specific Immunotherapy, Omalizumab
- *Bisphosphonate-Resistant Osteoporosis:* Denosumab (anti-RANKL), Romosozumab (anti-sclerostin).

Novel Treatment Options

Treatment-resistant, uncontrolled symptoms (moderate/severe): Avapritinib, Elenestinib, Bezuclastinib

- *Anaphylaxis Prevention:* In patients at risk of severe anaphylactic episodes, an emergency treatment kit containing an H1 antihistamine, corticosteroids, and a self-administered adrenaline injector should be prescribed. Individuals are advised to carry an epinephrine auto-injector at all times. For patients experiencing Hymenoptera Venom Allergy-related anaphylaxis, venom immunotherapy is recommended. However, the optimal duration of venom immunotherapy in patients with or without underlying clonal mast cell disorders remains uncertain [13].

Second Generation H1-Anti-Histamine

Anti-histamines effectively alleviate symptoms such as urticaria, angioedema and pruritus. Second generation H1-anti-histamine (sg-AH) should be initiated as a standard dose and updated if needed to 4-times the standard dose (Figure 5). Rupatadine 20 mg daily has shown significant improvement in quality of life in adult mastocytosis [14].

H2-Antihistamine (Anti-H2)

It is used as an add-on therapy for patients experiencing GI symptoms associated with MCAS episodes. In addition, there is evidence that combination of anti-H1 and anti-H2 is more effective than either alone in reducing symptoms of pruritus, urticaria and angioedema.

Leukotriene Receptor Antagonists (LTRA)

Moneteluakst is the seocnd most commonly used drug for symptom control after sg-AH. In patients who have marked incraese in cystein leukotriene during episodes of systemic MCAS [14, 15].

Sodium Cromoglicate (Cromolyn)

Oral cromolyn should be considered in individuals with persistent GI symptoms (abdominal cramping and diarrhoea) associated with MCAS episodes despite the use of anti-H1 and anti-H2 [15, 16]. Oral cromolyn is minimally absorbed in GIT and is generally well tolerated. It should be started with a low dose and gradually increase to the recommended dose of 200 mg 4-times daily. It should be taken on empty stomach before meal and bedtime. The recommended mode of administration involves opening the capsule(s), emptying the contents into a cup, dissolving the powder in one teaspoon of very hot water, and then adding four teaspoons of cold water for dilution. Cromolyn Sodium therapy should generally be continued for a minimum of one month before its effectiveness is evaluated [6].

Ketotifen

It is a first generation anti-H1 known for its mast cell-stabilizing property. Many experts suggest using ketotifen as it is more effective in patients who fail to be controlled with a high dose sg-AH [14–17]. It should be administered at low dose and initially taken at bedtime due to its sedative effect. The recommended dose is 2 mg twice a day.

Omalizumab (Anti-IgE)

Omalizumab, although used off-label, appears to be an effective therapeutic option for reducing symptoms related to mast cell mediator release and is generally associated with a favorable safety profile [14–17]. Omalizumab is a recombinant DNA-derived humanized (IgG1k) monoclonal antibody that binds to free human immunoglobulin E (IgE) in the blood & interstitial fluid and indirectly leads to reduction of membrane-bound form of IgE (mIgE) present on the surface of effector cells.

GENETIC COUNSELLING AND SPECIALIZED CARE (CLONAL DISEASE)

Testing for first degree relatives of individuals with clonal disease is recommended for genetic counselling due to inherited nature of the condition. In mixed MCAS (primary & secondary), specific treatment modality may be necessary in advanced SM and severe IgE dependent allergy may require a drug directed c-KIT mutation or combined treatment with Avapritinib and Omalizumab to control MCAS events. Flow charts for management of associated cutaneous symptoms, GI symptoms and osteoporosis is mentioned in Figure 5–7.

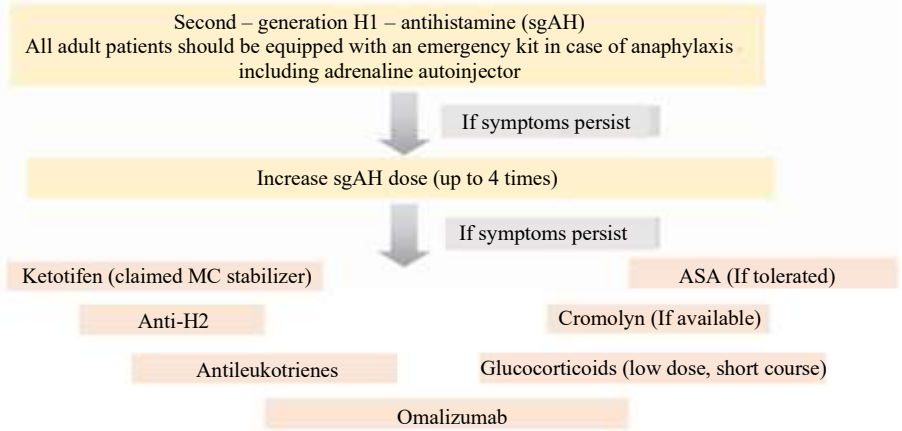

Figure 5. Proposed stepwise treatment of cutaneous mediator-related symptoms (flushing, pruritus, urticaria, angioedema.

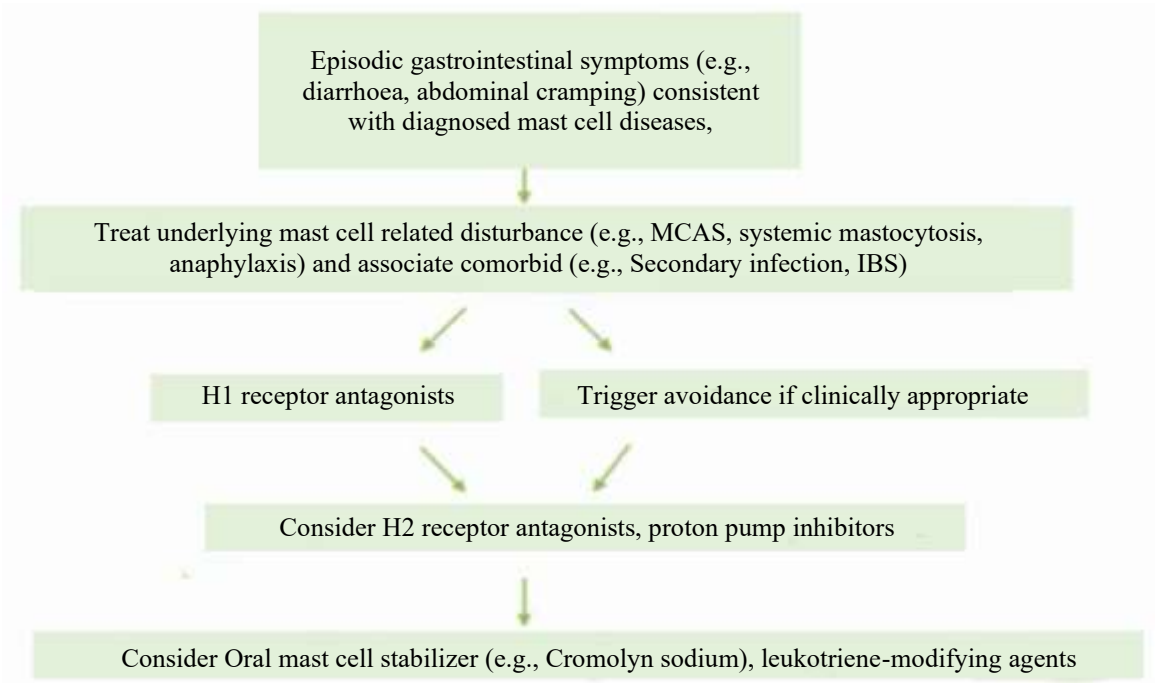

Figure 6. Stepwise management of GI symptoms in MCAD.

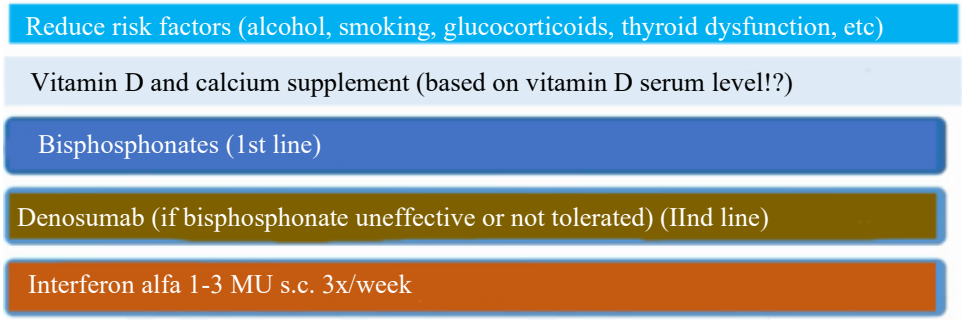

Figure 7. Treatment strategies for osteoporosis.

CONCLUSION

MCAD is a heterogenous group of multi-organs, multi-system disorders having complex pathology with variable course. Bone marrow biopsy is done to confirm or rule-out clonal disorders. The most important trigger of disease evolution and the target in advanced systemic mastocytosis is KIT mutant D816V. Further research into molecular mechanism is crucial for developing better diagnostic strategy and more effective management of MCAD.

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