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Scientific Letter

Transplantation in Advanced Systemic Mastocytosis: A Single-Center Case Series Highlighting Gastrointestinal Toxicity and GvHD Risk

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Introduction. Systemic mastocytosis (SM) is a rare neoplastic disease characterized by clonal proliferation of altered mast cells (MCs). The organs primarily involved are the skin and bone marrow, but the gastrointestinal (GI) tract, liver, and spleen are often infiltrated.[1] SM diagnosis relies on the identification of atypical MCs by morphological, histological, cytofluorimetric, and molecular analyses, the detection of the D816V KIT mutation, and the presence of B or C findings that define organ involvement and dysfunction.[2-4] On this basis, five main clinical variants can be identified with different prognoses: indolent SM (ISM), smoldering SM (SSM), aggressive SM (ASM), SM with associated hematological neoplasm (SM-AHN), and mast cell leukemia (MCL). Treatment of ISM and SSM relies on controlling symptoms driven by mediator release, whereas TKIs are required for patients with AdvSM.[5] When conventional treatments fail to control the disease, allogeneic transplantation could be a therapeutic option to discuss with younger patients, although long-term efficacy is questionable.[6] Recently, guidelines have been published to help guide decisions on the most favorable timing of transplant, adequate conditioning regimens, and stem cell source.[7] In this report, we aimed to discuss the indication for transplant, therapeutic options, and outcomes in three patients with AdvSM to provide further information for the management of these patients. Clinical characteristics of disease, conditioning regimen, GvHD prophylaxis, and engraftment are reported in **Table 1**. IWG-MRT-ECNM response criteria were considered for response assessment.[8]

Case 1: Young patient with progressive MCL. A 32-year-old female patient was diagnosed with MCL in February 2013. She presented to the emergency room (ER) with fever, vomiting, and epigastric pain. Physical examination revealed diffuse brown papular skin lesions and hepatosplenomegaly. Laboratory tests showed grade II anemia and thrombocytopenia, with circulating MCs noted on peripheral blood smear and serum tryptase at 200 mcg/L. A bone marrow evaluation revealed 80% infiltration by a multifocal cluster of CD2/CD25+ MCs, with the presence of the KIT D816V mutation, but no fibrosis. Upper endoscopy revealed only chronic inflammation, apparently without detectable MCs infiltration. She started subcutaneous pegylated (PEG) interferon 180 mcg weekly, and concomitant off-label dasatinib was initiated. Moreover, HLA typing and familiar donor screening were performed. In April 2013, she presented to the ER for abdominal pain, worsening of fever, and progressive leucocytosis: cytarabine 3000 mg intravenously for 4 days was started, and an application for compassionate use of midostaurin was completed. In June 2013, she started midostaurin 100 mg twice a day, with limited benefit over clinical symptoms and bone marrow infiltration. In September 2013, bone marrow evaluation demonstrated 90% infiltration by MCs and KIT D816V mutation: she underwent allogeneic transplant from an HLA-matched unrelated donor. The pre-engraftment period was complicated by urinary tract infection and flushing syndrome, which were resolved after appropriate antibiotic therapy and steroids, respectively. In October 2013, bone marrow evaluation did not show MCs infiltration and KIT D816V mutation, while full donor chimerism was reported; serum tryptase was 20 mcg/l. She developed cutaneous and intestinal GvHD, for which extracorporeal photopheresis, steroids, and two lines of immunosuppressive therapy were employed (infliximab, rituximab). She died in November 2013 from progressive intestinal GvHD and multiple infections.

Case 2: Life-threatening GvHD. A 59-year-old male patient was diagnosed with ASM in January 2018. He had a previous diagnosis of tubular adenoma of the colon with low-grade dysplasia and Helicobacter pylori

Table 1. Characteristics of patients.

	Patient 1	Patient 2	Patient 3
Diagnosis subtype	MCL	ASM	ASM
Age	32y	59y	48y
KIT mutation	positive	positive	positive
BM burden	80%	25%	45%
Fibrosis	none	grade I	grade III
GI involvement	chronic inflammation	colon and gastroduodenal infiltration	none
Prior therapies	PEG IFN, dasatinib, cytarabine, midostaurin	midostaurin, 2CDA	imatinib, midostaurin, 2CDA, avapritinib
Disease status at transplant	SD	PD	PR
Donor type	MUD	haplo	sibling
Conditioning regimen	BuCy	TBF	TBF
GvHD profylaxis	Thymo, CSA, MTX	Cy, CSA, MMF	CY, CSA, MMF
Engraftment	30 days	30 days except platelet count	18 months
Post transplant ASM status	BM CR	BM PR, persistent fibrosis	CR
GvHD	skin, intestinal	gastrointestinal	none
Outcome	death	death	alive
Follow-up	60 days	120 days	64 months

Legenda: MCL mast cell leukemia, ASM aggressive systemic mastocytosis, BM bone marrow, GI gastro intestinal, PEG IFN peghylated interferon, 2CDA cladribine, SD stabel disease, PD progressive disease, PR partial response, CR complete response, GvHD graft versus host disease.

(Hp) related chronic gastritis. Since 2014, he has reported grade II neutropenia for which he has performed periodic blood count analysis. In December 2017, he presented to the ER with persistent fever: physical examination revealed splenomegaly confirmed by US evaluation (spleen 18 cm), and laboratory tests showed grade II anemia and grade IV neutropenia. Bone marrow evaluation revealed 25% infiltration by a a multifocal cluster of atypical CD2-/CD25+ MCs and grade I fibrosis, together with the presence of the KIT D816V mutation; serum tryptase was 100 mcg/L. In September 2018, he started midostaurin 100 mg twice daily, with limited benefit; in November 2020, bone marrow evaluation showed 40% MC infiltration and grade II fibrosis; concomitant serum tryptase was 132 mcg/L. Endoscopy revealed colon and gastro-duodenal infiltration of MCs (**Figure 1, A to D**), together with Hp infection. Cladribine 0.14 mg/kg daily infusion for 5 consecutive days was initiated; HLA typing and family donor screening were also performed. In March 2021, he presented to the ER with fever, nausea, vomiting, and scrotal abscess: he received multiple courses of antibiotic therapy and red blood cell transfusions. In July 2021, he underwent an allogeneic transplant from a haploidentical donor: bone marrow evaluation showed 75% MCs infiltration and grade III fibrosis. The pre-engraftment period was complicated by suspected veno-occlusive disease, diarrhea, and fever, which resolved after defibrotide and empiric antibiotic therapy. In August 2021, bone marrow evaluation detected a 25% MCs infiltration with persistent KIT D816V and grade II fibrosis. In September 2021, he came to the ER because of worsening diarrhea and vomiting, suspicious for intestinal GvHD. After a negative microbiological workup, extracorporeal photopheresis, steroids, and ruxolitinib were employed. Bone marrow evaluation revealed persistent 30% MCs infiltration and grade II fibrosis, and gastro-intestinal biopsies detected lamina propria inflammation suggestive of GvHD but no MCs infiltration or Hp infection (**Figure 1, E-F**). He experienced progressive worsening of clinical conditions after the onset of pleural effusion, hypotension requiring amine support, and abdominal pain with bloody vomiting. He was transferred to the intensive care unit (ICU) and died a few days later in November 2021 from progressive deterioration.

Case 3: late recovery of bone marrow function. A 48-year-old male patient was diagnosed with ASM in July 2016. He complained of progressive fatigue and bone pain, associated with weight loss >10% in the previous 6 months. Physical examination revealed splenomegaly confirmed by US evaluation (spleen 17 cm), and laboratory tests showed grade III anemia. A bone marrow evaluation revealed infiltration by multifocal clusters of atypical CD2/CD25+ MCs and grade III fibrosis, together with the presence of KIT D816V mutation; serum tryptase was normal. Imatinib 100 mg daily was withdrawn after a few months due to an absent clinical response. In October 2018, the patient started midostaurin 100 mg twice a day with fluctuating clinical benefit. In December 2019, worsening of blood count

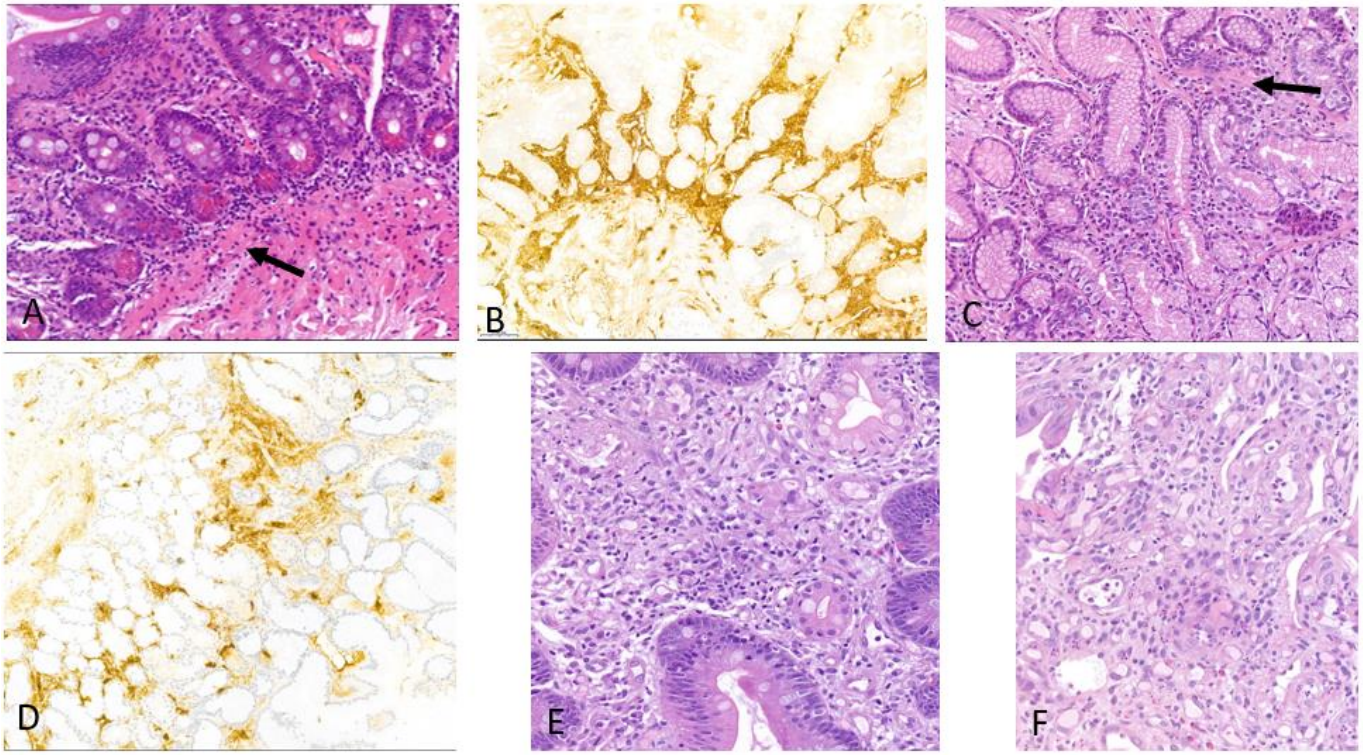

Figure 1. A. Hematoxylin and eosin (H&E) staining of colon (30x): MCs infiltration indicated by arrow. **B**. Tryptase antibody staining of colon (30x): yellow indicates triptase expression. **C**. H&E staining of stomach (20x): MCs infiltration indicated by arrow. **D**. Tryptase antibody staining of stomach (20x): yellow indicates triptase expression. **E and F**. H&E staining of colon and stomach (20x): inflammatory infiltrates suggestive of GvHD, no MCs infiltration has identified.

and persistent bone pain and fatigue revealed BM infiltration by 60% MCs with persistent grade III fibrosis and KIT D816V mutation. Cladribine 0.14 mg/kg daily infusion for 5 consecutive days was initiated; HLA typing and family donor screening were also performed. In June 2020, the patient presented the ER with a fever and received antibiotics as an inpatient for severe pulmonary infection. Bone marrow examination reported persistent 65% BM infiltration, grade III fibrosis, and KIT D816V mutation. In December 2020, avapritinib 200 mg daily was initiated in a compassionate program, with sustained improvement in platelet and absolute neutrophil counts and partial resolution of bone pain and fatigue. In February 2021, bone marrow evaluation showed sparse microaggregates of CD25+ MCs and an undetectable KIT D816V mutation, with persisting grade III fibrosis; he underwent allogeneic transplant from a sibling donor. The pre-engraftment period was complicated by a bloodstream infection caused by E. coli, which resolved after antibiotic therapy. The patient experienced prolonged incomplete recovery of platelet count and hemoglobin, for which weekly red blood cell transfusions were scheduled for 14 months. Periodic bone marrow evaluation revealed poor myelopoiesis with only sparse normally shaped MCs, full donor chimerism, and undetectable KIT mutation. In August 2022, 18 months after transplant, complete recovery of bone marrow function was obtained: at last follow-up in March 2026, full donor chimerism and undetectable KIT mutation were confirmed while bone pain and moderate asthenia persisted as the only symptoms.

Conclusions and Future Perspectives. Allogeneic transplant in AdvSM remains controversial, not only because of the rarity of the disease. The discovery of cKIT mutations and the subsequent availability of targeted therapy have changed the prognosis of AdvSM: disease control has improved, with median estimated 24-month OS rate of 76% overall.[9] Nevertheless, AdvSM may present as a rapidly progressive disease, or relapse may occur early after initial response: subsequent available treatments are limited, waiting for experimental drugs to be evaluated.

Comparing the three most considerable studies about transplant in AdvSM, OS, and PFS after transplant were largely overlapping: at 3 years, about 40-50% of patients progressed, and almost 50% died.[6,10,11] Some considerations could be discussed. First, the study's population includes mostly patients with SM-AHN, among whom the associated hematological neoplasm is often responsible for the indication to transplant. Different authors have reported a reduced efficacy of graft-versus-mast cell effect compared to other hematopoietic compartments. This is particularly prominent in extra-medullary disease, irrespective of the intensity of conditioning regimens,[6,10] as reported for most myeloid neoplasms.[12] Furthermore, single case reports have shown a delayed clearance of bone marrow MCs infiltration.[13,14] Second, the time of observation is

wide and not directly comparable to today, when TKI are widely available for AdvSM treatment. Nevertheless, recent changes in transplant management have improved non-relapsed mortality and mitigated the severity of GvHD.

According to clinical conditions and age, the transplant option should be discussed early with patients to plan HLA typing and familiar/unrelated donor screening. Myeloablative conditioning (MAC) regimens are known to be highly effective in disease control, although a higher rate of drug toxicity and increased GvHD severity have been observed. In the last few years, the age limit for the procedure has progressively increased, following the introduction of reduced-intensity conditioning (RIC) regimens. A worse survival was reported following RIC among 57 patients transplanted between 1990 and 2013,[6] although RIC was used in patients with lower performance status. More recently, a retrospective analysis of 71 patients transplanted between 1999 and 2021 could not conclusively establish the superiority of one conditioning regimen over another, likely due to the wide range of regimens used.[10] In our patients, the use of MAC regimens obtained disease control in all cases and timely engraftment in 2/3 cases, while severe GvHD was fatal for two patients.

Transplant should be performed when the best disease control has been achieved: lack of response was reported to predict an adverse prognosis among 71 patients transplanted between 1999 and 2021.[10] In our third patient, the use of avapritinib before transplant reduced the burden of disease, while grade III fibrosis persisted at the time of transplant: the presence of fibrosis since diagnosis may have impaired bone marrow recovery irrespective of conditioning regimens, as for other myeloid neoplasms.[15]

In our case series, we reported good tolerability of MAC, with effective disease eradication and variable timing of engraftment. A potential difficulty in GvHD control has emerged, especially if the target organ has been a site of disease involvement: prior GI mast-cell involvement may warrant careful pre-transplant assessment, as severe post-transplant GI GvHD/toxicity may be difficult to control. The limitations of this report are related to the small number of cases and the relatively long observation period, during which both therapeutic options and transplant procedures have advanced. More data are warranted to better understand the potential correlation between MC infiltration and GvHD severity.

In this case series, two patients reported symptoms predictive of MCs' infiltration of the GI tract at the onset of AdvSM. In the post-engraftment phase, both developed acute severe GI GvHD, irrespective of conditioning regimen and GvHD prophylaxis. Acute GvHD has been reported in 42% of patients in two studies,[6,10] while it was about 20% after wider use of RIC in a more recent study.[11] No apparent correlation with prior disease infiltration has been reported so far. Recently, different studies have identified elevated MCs infiltration in the GI tract in patients with unexplained chronic diarrhea, disorders of gut-brain interaction, and irritable bowel syndrome.[16,17,18] MC activation causes hypersecretion, alterations in intestinal motility, visceral hypersensitivity, and alterations in the gut epithelial barrier through the release of inflammatory mediators such as histamine, prostaglandins, serotonin, and tryptase.[19,20] Taken together, these observations may suggest a contributory role for mediators released from MCs after the graft-versus-mast cell effect, increasing local toxicity and ultimately causing damage. In patients with AdvSM, pre-transplant GI involvement may be a clinical warning sign requiring careful assessment and monitoring.

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Footnotes. The study was conducted in accordance with the Declaration of Helsinki. Ethical review and approval were waived for this study due to report of a case series with no formal analysis aiming to evaluate the efficacy of a therapeutic approach.

Informed consent to publication was obtained verbally from living patients and recorded in the medical record.

Author contributions. CM Conceptualization; Data curation; Writing – original draft. FL, CP, and SS Data curation; Writing – review & editing. PL Conceptualization; Data curation; Writing – review & editing.

Availability of data and materials. All data reported in the manuscript are available on request.

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References:

1. Valent P, Akin C, Metcalfe DD. Mastocytosis: 2016 updated WHO classification and novel emerging treatment concepts. Blood 2017:129, 1420-1427
https://doi.org/10.1182/blood-2016-09-731893
PMid:28031180 PMCid:PMC5356454
2. Arber DA, Orazi A, Hasserjian R, Thiele J, Borowitz MJ, Le Beau MM, Bloomfield CD, Cazzola M, Vardiman JW. The 2016 revision to the World Health Organization classification of myeloid neoplasms and acute leukemia. Blood 2016; 127:2391-2405.
https://doi.org/10.1182/blood-2016-03-643544
PMid:27069254
3. Khoury JD, Solary E, Abla O, Akkari Y, Alaggio R, Apperley JF, Bejar R, Berti E, Busque L, Chan JKC, Chen W, Chen X, Chng WJ, Choi JK, Colmenero I, Coupland SE, Cross NCP, De Jong D, Elghetany MT, Takahashi E, Emile JF, Ferry J, Fogelstrand L, Fontenay M, Germing U, Gujral S, Haferlach T, Harrison C, Hodge JC, Hu S, Jansen JH, Kanagal-Shamanna R, Kantarjian HM, Kratz CP, Li XQ, Lim MS, Loeb K, Loghavi S, Marcogliese A, Meshinchi S, Michaels P, Naresh KN, Natkunam Y, Nejati R, Ott G, Padron E, Patel KP, Patkar N, Picarsic J, Platzbecker U, Roberts I, Schuh A, Sewell W, Siebert R, Tembhare P, Tyner J, Verstovsek S, Wang W, Wood B, Xiao W, Yeung C, Hochhaus A.The 5th edition of the World Health Organization Classification of Haematolymphoid Tumours: Myeloid and Histiocytic/Dendritic Neoplasms. Leukemia. 2022;36(7):1703-1719.
https://doi.org/10.1038/s41375-022-01613-1
PMid:35732831 PMCid:PMC9252913
4. Arber DA, Orazi A, Hasserjian RP, Borowitz MJ, Calvo KR, Kvasnicka HM, Wang SA, Bagg A, Barbui T, Branford S, Bueso-Ramos CE, Cortes JE, Dal Cin P, DiNardo CD, Dombret H, Duncavage EJ, Ebert BL, Estey EH, Facchetti F, Foucar K, Gangat N, Gianelli U, Godley LA, Gökbuget N, Gotlib J, Hellström-Lindberg E, Hobbs GS, Hoffman R, Jabbour EJ, Kiladjian JJ, Larson RA, Le Beau MM, Loh ML, Löwenberg B, Macintyre E, Malcovati L, Mullighan CG, Niemeyer C, Odenike OM, Ogawa S, Orfao A, Papaemmanuil E, Passamonti F, Porkka K, Pui CH, Radich JP, Reiter A, Rozman M, Rudelius M, Savona MR, Schiffer CA, Schmitt-Graeff A, Shimamura A, Sierra J, Stock WA, Stone RM, Tallman MS, Thiele J, Tien HF, Tzankov A, Vannucchi AM, Vyas P, Wei AH, Weinberg OK, Wierzbowska A, Cazzola M, Döhner H, Tefferi A. International Consensus Classification of Myeloid Neoplasms and Acute Leukemias: integrating morphologic, clinical, and genomic data. Blood. 2022;140(11):1200-1228
https://doi.org/10.1182/blood.2022015850
PMid:35767897 PMCid:PMC9479031
5. Pardanani A, Reichard K, Tefferi A. Advanced systemic mastocytosis-Revised classification, new drugs and how we treat. Br J Haematol. 2024;204(2):402-414
https://doi.org/10.1111/bjh.19245
PMid:38054381
6. Ustun C, Reiter A, Scott BL, Nakamura R, Damaj G, Kreil S, Shanley R, Hogan WJ, Perales MA, Shore T, Baurmann H, Stuart R, Gruhn B, Doubek M, Hsu JW, Tholouli E, Gromke T, Godley LA, Pagano L, Gilman A, Wagner EM, Shwayder T, Bornhäuser M, Papadopoulos EB, Böhm A, Vercellotti G, Van Lint MT, Schmid C, Rabitsch W, Pullarkat V, Legrand F, Yakoub-Agha I, Saber W, Barrett J, Hermine O, Hagglund H, Sperr WR, Popat U, Alyea EP, Devine S, Deeg HJ, Weisdorf D, Akin C, Valent P. Hematopoietic stem-cell transplantation for advanced systemic mastocytosis. J Clin Oncol. 2014;32(29):3264-74
https://doi.org/10.1200/JCO.2014.55.2018
PMid:25154823 PMCid:PMC4876356
7. McLornan DP, Czerw T, Damaj G, Ethell M, Gurnari C, Hernández-Boluda JC, Polverelli N, Schwaab J, Sockel K, Raffaella G, Onida F, Sánchez-Ortega I, Battipaglia G, Elena C, Gotlib J, Reiter A, Rossignol J, Ustun C, Valent P, Yakoub-Agha I, Radia DH.Allogeneic haematopoietic cell transplantation for advanced systemic mastocytosis: Best practice recommendations on behalf of the EBMT Practice Harmonisation and Guidelines Committee. Leukemia. 2024;38(4):699-711.
https://doi.org/10.1038/s41375-024-02182-1
PMid:38472477
8. Gotlib J., Pardanani A., Akin C., Reiter A., George T., Hermine O., Kluin-Nelemans H., Hartmann K., Sperr W.R., Brockow K., et al. International Working Group-Myeloproliferative Neoplasms Research and Treatment (IWG-MRT) & European Competence Network on Mastocytosis (ECNM) consensus response criteria in advanced systemic mastocytosis. Blood. 2013;121:2393-2401
https://doi.org/10.1182/blood-2012-09-458521
PMid:23325841 PMCid:PMC3612852
9. DeAngelo DJ, Radia DH, George TI, Robinson WA, Quiery AT, Drummond MW, Bose P, Hexner EO, Winton EF, Horny HP, Tugnait M, Schmidt-Kittler O, Evans EK, Lin HM, Mar BG, Verstovsek S, Deininger MW, Gotlib J.Safety and efficacy of avapritinib in advanced systemic mastocytosis: the phase 1 EXPLORER trial. Nat Med. 2021;27(12):2183-2191
https://doi.org/10.1038/s41591-021-01538-9
PMid:34873347 PMCid:PMC8674134
10. Lübke J, Christen D, Schwaab J, Kaiser A, Naumann N, Shoumariyeh K, Jentzsch M, Sockel K, Schaffrath J, Ayuk FA, Stelljes M, Hilgendorf I, Sala E, Kaivers J, Schönland S, Wittke C, Hertenstein B, Radsak M, Kaiser U, Brückl V, Kröger N, Brümmendorf TH, Hofmann WK, Klein S, Jost E, Reiter A, Panse J. Allogeneic Hematopoietic Cell Transplantation in Advanced Systemic Mastocytosis: A retrospective analysis of the DRST and GREM registries. Leukemia 2024 38:810-821
https://doi.org/10.1038/s41375-024-02186-x
PMid:38448757 PMCid:PMC10997505
11. Ustun C, Zhang MJ, Peterson A, Baek A, Agha M, Alkhateeb H, Chhabra S, Coltoff A, de Lima M, Gandhi A, Ho V, Kassim A, Lin A, Gowda L, Borthakur G, DeAngelo DJ, McGuirk J, Mensah F, Nadiminti KV, Nishihori T, Pantin J, Trunk A, Uberti J, Marcucci G, Gotlib J, Akin C, Hamadani M, Pullarkat V, Valent P, Grunwald M, Juckett M, Oran B, Saber W, Burns LJ. Allogeneic Hematopoietic Cell Transplantation in Advanced Systemic Mastocytosis in New Era: A CIBMTR study. Br J Haematol. 2025; 207(6): 2486-2495
https://doi.org/10.1111/bjh.70154
PMid:40983528 PMCid:PMC12519450
12. Chong G, Byrnes G, Szer J, Grigg A. Extramedullary relapse after allogeneic bone marrow transplantation for haematological malignancy. Bone Marrow Transplant 2000; 26: 1011-1015
https://doi.org/10.1038/sj.bmt.1702659
PMid:11100282
13. Nakamura R, Chakrabarti S, Akin C, Robyn J, Bahceci E, Greene A, Childs R, Dunbar C E, Metcalfe D D, Barrett A J. A pilot study of nonmyeloablative allogeneic hematopoietic stem cell transplant for advanced systemic mastocytosis. Bone Marrow Transplant. 2006;37(4):353-8
https://doi.org/10.1038/sj.bmt.1705245
PMid:16400343
14. Gromke T, Elmaagacli A H, Ditschkowski M, Hegerfeldt Y, Koldehoff M, Hlinka M, Ottinger H, Trenschel R, Beelen D W. Delayed graft-versus-mast-cell effect on systemic mastocytosis with associated clonal haematological non-mast cell lineage disease after allogeneic transplantation. Bone Marrow Transplant 2013;48, 732-310 733
https://doi.org/10.1038/bmt.2012.198
PMid:23085825
15. Salit RB, Hexner EO, Gagelmann N, Kröger N, McLornan DP, Jain T, Gupta V, Hobbs GS, Tamari R, Robin M, Scott B, Saber W. Defining remission following hematopoietic cell transplant for myelofibrosis: an international expert panel consensus.Defining remission following hematopoietic cell transplant for myelofibrosis: an international expert panel consensus. Leukemia 2025;39(12):2862-2865
https://doi.org/10.1038/s41375-025-02754-9
PMid:41083679 PMCid:PMC12713569
16. Wouters MM, Vicario M, Santos J. The role of mast cells in functional GI disorders. Gut. 2016;65:155-168.
https://doi.org/10.1136/gutjnl-2015-309151
PMid:26194403
17. Bashashati M, Moossavi S, Cremon C, Barbaro MR, Moraveji S, Talmon G, Rezaei N, Hughes PA, Bian ZX, Choi CH, Lee OY, Coëffier M, Chang

L, Ohman L, Schmulson MJ, McCallum RW, Simren M, Sharkey KA, Barbara G.Colonic immune cells in irritable bowel syndrome: a systematic review and meta-analysis. Neurogastroenterol Motility. 2018;30:e13192
https://doi.org/10.1111/nmo.13192
PMid:28851005

18. Robles A, Perez Ingles D, Myneedu K, Deoker A, Sarosiek I, Zuckerman MJ, Schmulson MJ, Bashashati M. Mast cells are increased in the small intestinal mucosa of patients with irritable bowel syndrome: A systematic review and meta-analysis. Mast cells are increased in the small intestinal mucosa of patients with irritable bowel syndrome: a systematic review and meta-analysis. Neurogastroenterol Motility. 2019;31:e13718
https://doi.org/10.1111/nmo.13718
PMid:31498961
19. Wouters MM, Balemans D, Van Wanrooy S, Dooley J, Cibert-Goton V, Alpizar YA, Valdez-Morales EE, Nasser Y, Van Veldhoven PP, Vanbrabant W, Van der Merwe S, Mols R, Ghesquière B, Cirillo C, Kortekaas I, Carmeliet P, Peetermans WE, Vermeire S, Rutgeerts P, Augustijns P, Hellings PW, Belmans A, Vanner S, Bulmer DC, Talavera K, Vanden Berghe P, Liston A, Boeckxstaens GE. Histamine receptor H1-Mediated Sensitization of TRPV1 mediates visceral hypersensitivity and symptoms in patients with irritable bowel syndrome. Gastroenterology. 2016;150(4):875-87.e9.
https://doi.org/10.1053/j.gastro.2015.12.034
PMid:26752109
20. Barbara G, Stanghellini V, De Giorgio R, Cremon C, Cottrell GS, Santini D, Pasquinelli G, Morselli-Labate AM, Grady EF, Bunnett NW, Collins SM, Corinaldesi R. Activated mast cells in proximity to colonic nerves correlate with abdominal pain in irritable bowel syndrome. Gastroenterology. 2004;126(3):693-702
https://doi.org/10.1053/j.gastro.2003.11.055
PMid:14988823 PMCid:PMC9848657