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Evaluation and Differential Diagnosis of Eosinophilia

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ABSTRACT: Eosinophilia is defined as an increase number of eosinophils in peripheral blood (PB) or tissues above what is considered to be the normal range. Eosinophils in PB of normal individuals range from 0.0 to 6.0% of leukocytes and have an absolute eosinophil count of 0.02–0.5 × 10^9/L. The normal range for eosinophils in bone marrow (BM) is 1–6%. A definition of BM eosinophilia has been proposed that requires ≥20% of marrow cells to be eosinophils, with or without PB eosinophilia [1,2]. Tissue eosinophilia is defined as increased eosinophils or signs of eosinophil degranulation in extramedullary sites such as the gastrointestinal tract, lung, thymus, spleen or lymph nodes[3].

Hypereosinophilia (HE) is defined as a persistent elevated eosinophil count of ≥1.5 × 10^9/L.[5].HE can be one of the dominant manifestations of a hematopoietic myeloid neoplasm or secondary/reactive to an underlying medical condition. If a cause of HE and its associated tissue/organ damage is not determined, the condition is considered to be idiopathic hypereosinophilic syndrome (HES).[6]

MATERIALS AND METHODS : This is a prospective study done in the Department of Pathology, Mahatma Gandhi Medical College, Indore. We included 30 cases with eosinophilia on bone marrow aspirates ,peripheral blood and tissue sections who presented to our department from October 2019 to February 2021. A record of patient details and history was made. Evaluation of peripheral smear,bone marrow aspirate smears and H & E stained tissue sections was done for eosinophilia.

Inclusion Criteria –All smears showing eosinophilia .

Exclusion Criteria-Cases having no eosinophilia.

Results – In our study,30 cases with eosinophilia were studied,Out of which 10 cases were of Chronic myeloid leukemia,04 myelodysplastic syndrome,02 AML M4 E0,02 hodginks lymphoma,02 hypereosinophilic syndrome,02 parasitic infections, 04 allergic conditions,01 dermatitis herpetiformis,01 pneumonia and 02 eosinophillic gastroenteritis.

Most cases of monocytosis will prove to be reactive in nature. Proper evaluation is required to exclude the minority of cases which are neoplastic. This requires the initial assessment of clinical history, laboratory data, and morphology. Important clues to suggest a neoplastic process include an unexplained and persistent monocytosis, the
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Conclusion–Most cases of eosinophilia is secondary due to reactive expansion of eosinophils caused by primary disease process, such as parasitic infestation, drugs, allergies, autoimmune diseases, or malignant tumors. Proper evaluation is required to exclude the minority of cases which are neoplastic . This requires the initial assessment of clinical history, laboratory data and morphology.

Keywords:Hypereosinophilia,Hypereosinophilic syndrome , Chronic myeloidleukemia,MDS,dermatitisherpatitisformis

I. INTRODUCTION–

Elevations in the levels of peripheral blood and tissue eosinophils can occur in a wide variety of disease processes that include infectious, allergic, neoplastic, primary hematologic disorders, and other, often less well-defined entities[7,8,9]. Eosinophils are normally controlled by cytokines interleukin (IL)-5, GM-CSF, and IL-3 produced by T-lymphocytes, mast cells, and stromal cells [10]. Upon activation, eosinophils release their granules, such as eosinophil peroxidase, [10]Physiologically, eosinophil levels in the peripheral blood have a diurnal variation with a peak in the morning, a time at which endogenous steroids are the lowest.[11]

If Hypereosinophilia is persistent (≥6 months) and there is associated tissue damage, the disorder would be classified as hypereosinophilic syndrome (HES)[12,13]. A history of 6 months may not be necessarily enforced if the diagnostic work-up is adequate and treatment is needed to minimize organ damage caused by the eosinophilic infiltrate. Therefore, it is generally accepted that in the presence of tissue injury, if blood absolute eosinophil count > 1.5 × 109/L on 2 occasions in an interval ≥1 month, HE could be considered as "persistent"[12] . On the other hand, if a patient is asymptomatic and an underlying cause is not identified, a minimal duration of 6 months would be needed to consider a case to be idiopathic HE[13]

Secondary HE is a reactive expansion of eosinophils driven by a primary disease process, such as parasitic infestation, drugs, allergies, autoimmune diseases, or malignant tumors.[14]
Secondary eosinophilia can be associated with a malignancy. Examples are lymphoblastic leukemia/lymphoma [15], peripheral T-cell lymphoma [16, 17], and classical Hodgkin lymphoma [18]

AIM: To evaluate and study the differential diagnosis of eosinophilia.

II. MATERIALS AND METHODS

This is a prospective study done in the Department of Pathology, Mahatma Gandhi Medical College, Indore. We included cases with eosinophilia on bone marrow aspirates ,peripheral blood and tissue sections who presented to our department from October 2019 to february 2021. A record of patient details and history was made. Evaluation of peripheral smear,bone marrow aspirate smears and H & E stained tissue sections was done for eosinophilia.

Inclusion Criteria –All smears showing eosinophilia.
Exclusion Criteria-1)Cases having no eosinophilia.

III. RESULTS-

In our study,30 cases with eosinophilia were studied,Out of which 10 cases were of Chronic myeloid leukemia,04 myelodysplastic syndrome,02 AML M4 E0,02 hodginks lymphoma,02 hypereosinophilic syndrome,02 parasitic infections, 04 allergic conditions,01 dermatitis herpetiformis,01 pneumonia and 02 eosinophillic gastroenteritis.
Out of 30 cases ,21 cases of eosinophilia were seen in males and 09 cases were seen in females.

Table 1:No . of cases with eosinophilia

	Diseases	No.of cases

S.no		
1	CML	10
2	MDS	04
3	AML M4E0	02
4	Hodginks Lymphoma	02
5	Parasitic infections	02
6	Allergic condition	04
7	Dermatitis herpatiformis	01
8	Hypereosinophillic syndrome	02
9	Pneumonia	01
10	Gastroentritis	02
	Total	**30**

Table 2:-CASES WITH PERCENTAGE OF EOSINOPHILIA IN PERIPHERAL BLOOD.

S.NO	CASES	EOSINOPHILIAS (%)
1	CML	08
2	CML	10
3	CML	14
4	CML	09
5	CML	16
6	CML	09
7	CML	07
8	CML	08
9	CML	07
10	CML	10
11	MDS	08
12	MDS	07
13	MDS	08
14	MDS	09
15	AML M4E0	14
16	AML M4E0	11
17	HODGINKS LYMPHOMA	07
18	HODGINKS LYMPHOMA	06
19	HYPEREOSINOPHILIC SYNDROME	20
20	HYPEREOSINOPHILIC SYNDROME	18
21	PARASITIC INFECTIONS	08
22	PARASITIC INFECTIONS	10
23	ALLERGIC CONDITIONS	10
24	ALLERGIC CONDITIONS	09

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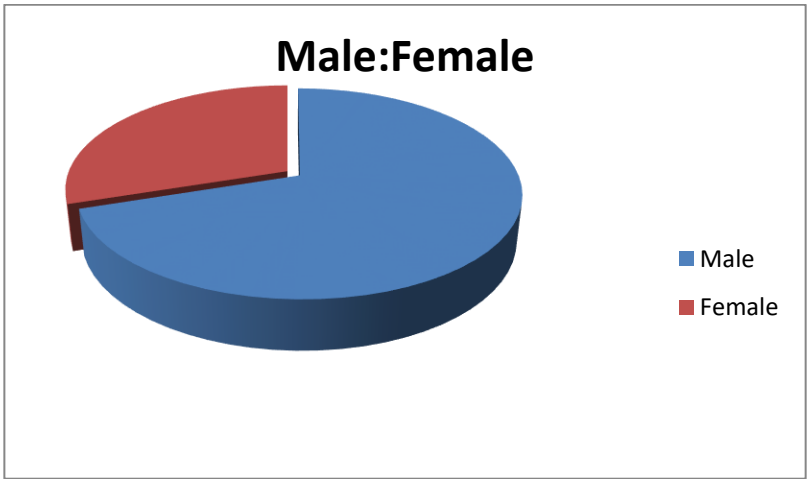
25	ALLERGIC CONDITIONS	08
26	ALLERGIC CONDITIONS	09
27	DERMATITIS HERPETIFORMIS	06
28	PNEUMONIA	05
29	GASTROENTERITIS.	07
30	GASTROENTERITIS	06



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IV. DISCUSSION:

Eosinophils are bone marrow-derived leukocytes whose development and terminaldifferentiation are under the control of several cytokines (IL-3, GM-CSF, and IL-5), with IL-5being the cytokine that is primarily responsible for eosinophilopoiesis[19].

Eosinophils, particularly in disease states associated with hypereosinophilia, can have a varietyof phenotypic and functional changes felt to reflect cellular activation. In these situations, theeosinophil on a peripheral smear can appear vacuolated with alterations in granule size, and,on flow cytometric analysis, the eosinophil has characteristic changes in surface moleculeexpression [20] By electron microscopy, eosinophils demonstrate piecemeal degranulation 42when activated.

HYPEREOSINOPHILIC CONDITIONS:

A. Infectious Diseases

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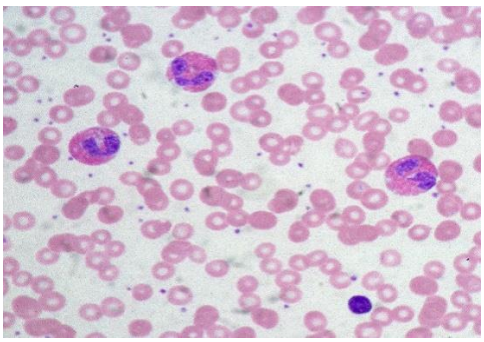
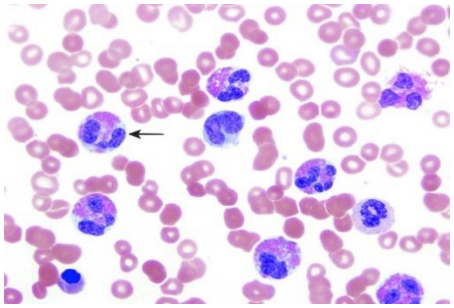
A wide variety of infectious agents, almost exclusively helminth (worm) parasites, elicit eosinophilia.[21,22] The pattern and degree of eosinophilia in parasitic infections is determined by the development, migration, and distribution of the parasite within the host as well as by the host's immune response[21,23]. In general, it is useful to remember that parasites tend to elicit marked eosinophilia when they or their products come into contact with immuneeffector cells in tissues, particularly during migration. Eosinophilia is highest among parasites with a phase ofdevelopment that involves migration through tissue.[24]In our study out of 30 cases,02 cases were of parasitic infections caused by ascaries which showed peripheral eosinophilia.



Peripheral blood smear showing eosinophilia

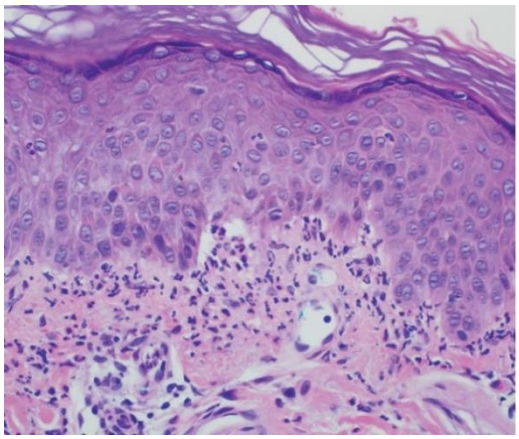
B. Atopic/Allergic Diseases-

Blood eosinophilia is seen in allergic conditions like allergic rhinitis, nonallergic rhinitis witheosinophilia syndrome (NARES) or even in asthma (both allergic and non-allergic) Because so many medications (as well as nutritional supplements and alternative therapies)have been associated with eosinophilia, a detailed history of current and past medicationsshould be obtained from all patients with eosinophilia[25,26]In our study out of 30 cases,04 cases were of allergic condition, which showed peripheral blood eosinophilia.



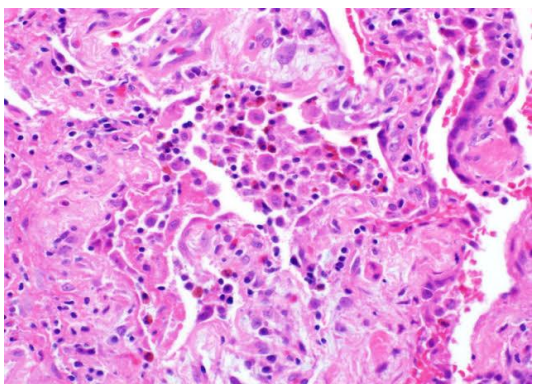
C.Skin and Subcutaneous Tissues:

Eosinophils are seen in the inflammatory infiltratein numerous dermatologic conditions. Blood and tissue eosinophilia are common in atopicdermatitis.[27] Tissue eosinophils are seen in blistering diseases, such as bullous pemphigoid,pemphigus vulgaris and dermatitis herpetiformis[28]. In our study out of 30 cases,we got one case of dermatitis herpetiformis which showed infiltration of skin with eosinophilia.



H& E stained image showing dermatitis herpetiformmis with inflammatory cell infiltrate comprising of neutrophils and eosinophils.

D.Pulmonary- Eosinophilic lung diseases are group of disorders characterised by the presence oflarge numbers of eosinophils in the airways or parenchyma of the lungs with a clinical presentation of pneumonia accompanied by abnormal chest radiograph/CT and peripheral bloodeosinophilia and tissue eosinophilia.It includes acute and chronic eosinophilic pneumonia.[29]We in our study got 01 case of pneumonia with eosinophils infiltrating in the lung parenchyma.



E.Gastrointestinal-

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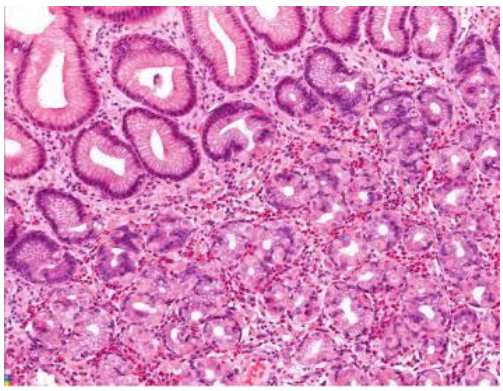
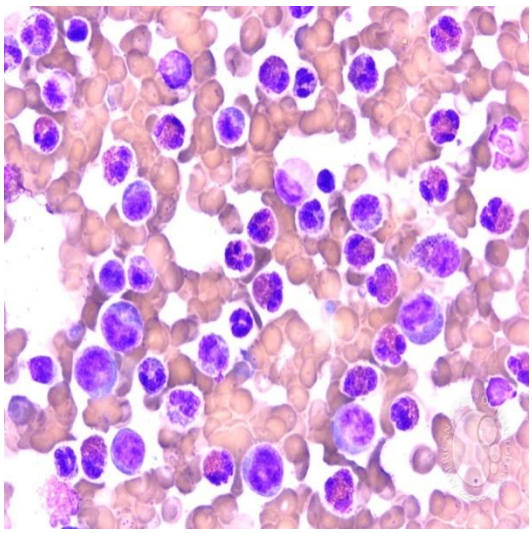
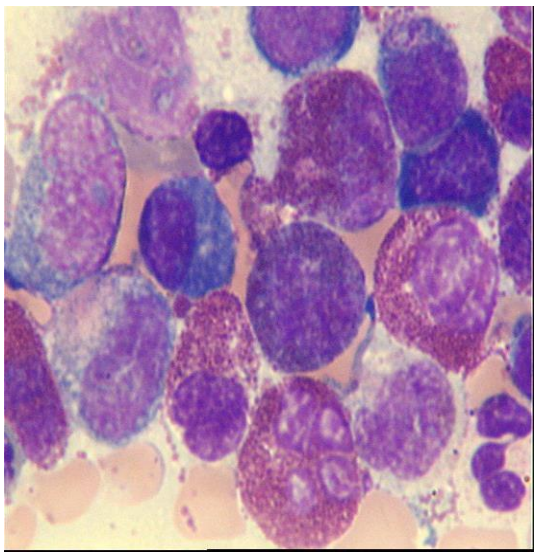
Blood eosinophilia can develop with a number gastrointestinal diseases like eosinophilic esophagitis, eosinophilic gastroenteritis[30]. We included 02 cases of gastroenteritis with eosinophilia in the lamina propria.



F.Hematologic/Neoplastic-
Eosinophils in clonal myeloid disorders are often derivedfrom hematopoietic progenitors bearing the same
molecular genetic aberrations. These clonal myeloid neoplasmscan be further categorized into 3 large groups:
(1)myeloid/lymphoid neoplasms with eosinophilia and rearrangementsof PDGFRA, PDGFRB, FGFR1 or provisionally
PCM1-JAK2 [31,32,33]; (2) HE associated with anotherwell-defined myeloid neoplasm, such as chronic myeloidleukemia (CML); and (3) chronic eosinophilic leukemia(CEL) not otherwise specified (NOS).
The BM is usually hypercellular with increased eosinophils,including eosinophilic precursors.
We included 10 cases of chronic myeloid leukemia,02 cases of AML M4 E0 and 04 cases of myelodysplastic syndrome with eosinophiliaon peripheral smear and bone marrow aspirate.



Peripheral smear showing chronic myeloid leukaemia with eosinophilia and eosinophilic precursor.



Aml m4Eo with eosinophilia

G.Lymphoid malignancies-Eosinophilia is often associated with Hodgkin'sDisease.Reed Sternberg cells generate IL5 which leads to eosinophilia.[34,35]We also got in our study two cases of hodginkslymhoma with eoniphilia.

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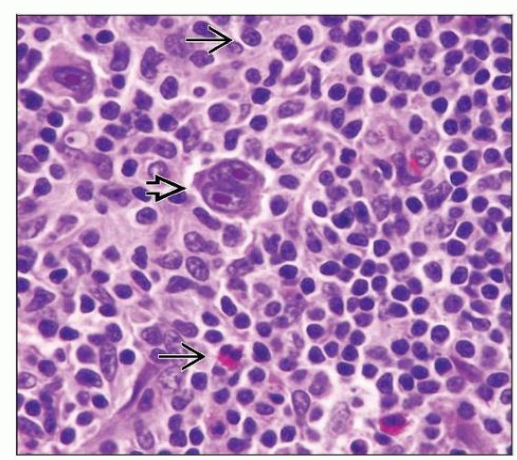
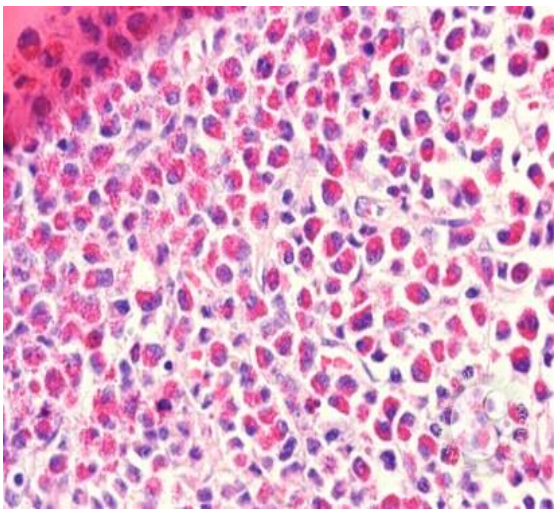


Hypereosinophilic syndrome-When hypereosinophilia is persistent(≥6 months), and there is associated tissue
damage, the disorder would be classified as hypereosinophilicsyndrome (HES)[36].We included 02 cases of HES in our study.



Peripheral smear of hypereosinophilic syndrome

APPROACH TO THE EVALUATION OF A PATIENT WITH HIGH GRADE EOSINOPHILIA

A careful and detailed history should be taken about the symptoms present,travel history, occupational and dietary history.
Previous eosinophils count should be noted.
A complete medication history should be taken that includes medications like supplements, herbal preparations, and vitamins.
Any medication known to induce eosinophilia should be discontinued.
Detailed family history like diseases commonly found in their family.
Any previous allergies to medications or to environmental allergens must also be addressed**.**
Careful physical examination should be done.
Special attention should be given to skin, soft tissues, lungs, liver, and spleen.
Additional examination based on the patient's specific symptoms or chief complaint is very important.

Initial Laboratory Evaluation

Routine hematologic test like CBC and estimation of the absolute eosinophil count should be done.
Studies to assess organ function (liver function tests, renal function tests, urinalysis, chest radiograph), inflammation (CRP/ESR), immune status (immunoglobulins, IgE).
Further Diagnostic Evaluations (based on initial laboratory findings or localizing symptoms)
• Tissue examination (biopsies) if necessary
• Specimen collection (CSF, sputum, bronchoalveolar lavage, stool, urine) that can identify the
• CT and MRI to define better focal lesions.
• Bone marrow aspirates and biopsies to assess fully the nature of the process underlying the eosinophilia.
• Additional disease-defining tests to exclude particular diagnoses (e.g. serum tryptase/cKIT mutations for systemic mastocytosis, antineutrophilcytoplasmic antibodies (ANCA) for CSS and other vasculitis, serologies for helminths)

V. CONCLUSION

Most cases of eosinophilia is secondary due to reactive expansion of eosinophils caused by primary disease process, such as parasitic infestation, drugs, allergies, autoimmune diseases, or malignant tumors. Proper evaluation is required to exclude the minority of cases which are neoplastic . This requires the initial assessment of clinical history, laboratory data and morphology.

REFERENCES

[1]. Valent P, Klion AD, Horny HP, Roufosse F, Gotlib J, Weller PF, Hellmann A, Metzgeroth G, Leiferman KM, Arock M, Butterfield JH, Sperr WR, Sotlar K, Vandenberghe P, Haferlach T, Simon HU, Reiter A, Gleich GJ: Contemporary consensus proposal on criteria and classification of eosinophilic disorders and related syndromes. J Allergy Clin Immunol 2012; 130: 607–612.e9.
[2]. Reiter A, Gotlib J: Myeloid neoplasms with eosinophilia. Blood 2017; 129: 704–714. 3 Gleich GJ: Mechanisms of eosinophil-

associated inflammation. J Allergy Clin Immunol 2000; 105: 651–663.
[3]. 3 Gleich GJ: Mechanisms of eosinophil-associated inflammation. J Allergy Clin Immunol 2000; 105: 651–663.
[4]. Gotlib J: World Health Organization-defined eosinophilic disorders: 2015 update on diagnosis, risk stratification, and management. Am J Hematol 2015; 90: 1077– 1089.
[5]. Gotlib J: World Health Organization-defined eosinophilic disorders: 2017 update on diagnosis, risk stratification, and management. Am J Hematol 2017; 92: 1243–1259.
[6]. Bain BJ, Horny HP, Hasserjian RP, Orazi A: Chronic Eosinophilic Leukaemia, NOS. Lyon France, IARC Press, 2017, pp 54–56.
[7]. Rothenberg ME. Eosinophilia. N Engl J Med 1998;338:1592. [PubMed: 9603798]
[8]. Simon D, Simon HU. Eosinophilic disorders. J Allergy Clin Immunol 2007;119:1291. [PubMed: 17399779
[9]. Wilson, ME.; Weller, PF., editors. Tropical Infectious Diseases. Principles, pathogens and practice. 2. Churchill Livingstone; Philadelphia: 1999. Eosinophilia, ed First; p. 1400
[10]. Gleich GJ: Mechanisms of eosinophil-associated inflammation. J Allergy Clin Immunol 2000; 105: 651–663.
[11]. Uhrbrand H. The number of circulating eosinophils; normal figures and spontaneous variations. Acta Med Scand 1958;160:99. [PubMed: 13532489
[12]. Gotlib J: World Health Organization-defined eosinophilic disorders: 2017 update on diagnosis, risk stratification, and management. Am J Hematol 2017; 92: 1243–1259.
[13]. Bain BJ, Horny HP, Hasserjian RP, Orazi A: Chronic Eosinophilic Leukaemia, NOS. Lyon France, IARC Press, 2017, pp 54–56. or FGFR1; in Swerdlow SH, Campo E, Harris NL, Jaffe ES, Pileri SA, Stein H, Thiele J, Vardiman JW (ed): WHO Classification of Tumours of Haematopoietic and Lymphoid Tissues. Lyon, International Agency for Research on Cancer (IARC) 2008, pp 68– 73.
[14]. Tefferi A, Patnaik MM, Pardanani A: Eosinophilia: secondary, clonal and idiopathic. Br J Haematol 2006; 133: 468–492.
[15]. Catovsky D, Bernasconi C, Verdonck PJ, Postma A, Hows J, van der Does-van den Berg A, Rees JK, Castelli G, Morra E, Galton DA: The association of eosinophilia with lymphoblastic leukaemia or lymphoma: a study of seven patients. Br J Haematol 1980; 45: 523–534.
[16]. Kawasaki A, Mizushima Y, Matsui S, Hoshino K, Yano S, Kitagawa M: A case of T-cell lymphoma accompanying marked eosinophilia, chronic eosinophilic pneumonia and eosinophils,pleural effusion. A case report. Tumori 1991; 77: 527–530.
[17]. Jin JJ, Butterfield JH, Weiler CR: Hematologic malignancies identified in patients with hypereosinophilia and hypereosinophilic syndromes. J Allergy Clin Immunol Pract 2015; 3: 920–925.
[18]. Endo M, Usuki K, Kitazume K, Iwabe K, Okuyama Y, Urabe A: Hypereosinophilic syndrome in Hodgkin's disease with increased granulocyte-macrophage colony-stimulating factor. Ann Hematol 1995; 71: 313–314.philic 16 Bain GA, Flower CD: Pulmonary eosinophilia. Eur J Radiol 1996; 23: 3–8.
[19]. Weller PF. The immunobiology of eosinophils. N Engl J Med 1991;324:1110. [PubMed: 2008184]
[20]. Mawhorter SD, Stephany DA, Ottesen EA, et al. Identification of surface molecules associated with physiologic activation of eosinophils: application of whole blood flow cytometry to eosinophils. J Immunol 1996;156:4851. [PubMed: 864813
[21]. Moore TA, Nutman TB. Eosinophilia in the returning traveler. Infect Dis Clin North Am 1998;12:503. [PubMed: 9658256]
[22]. Weller PF. Eosinophilia in travelers. Med Clin North Am 1992;76:1413. [PubMed: 1405826]
[23]. Brandborg LL, Goldberg SB, Briedenbach WC. Human coccidiosis-a possible cause of malabsorption: the life cycle in small-bowel biopsies as a diagnostic feature. N Engl J Med 1970;283:1306. [PubMed: 5478452
[24]. Uhrbrand H. The number of circulating eosinophils; normal figures and spontaneous variations. Acta Med Scand 1958;160:99. [PubMed: 13532489
[25]. Donhuijsen K, Haedicke C, Hattenberger S, et al. Granulocyte-macrophage colony-stimulating factorrelated eosinophilia and Loeffler's endocarditis. Blood 1992;79:2798. [PubMed: 1586727
[26]. Ishimitsu T, Torisu M. The role of eosinophils in interleukin-2/lymphokine-

activated killer cell therapy. Surgery 1993;113:192. [PubMed: 8381564.
[27]. Butterfield JH, Leiferman KM, Gleich GJ. Nodules, eosinophilia, rheumatism, dermatitis and swelling (NERDS): a novel eosinophilic disorder. Clin Exp Allergy 1993;23:571. [PubMed: 8221258
[28]. Leiferman KM. Eosinophils in atopic dermatitis. J Allergy Clin Immunol 1994;94:1310. [PubMed: 7798571]
[29]. Wechsler ME. Churg Strauss Syndrome and pulmonary eosinophilia. Allergy Clin Immunol Clinics NA. 2007in press.
[30]. Rothenberg ME. Eosinophilic gastrointesinal diseases. Allergy Clin Immunol Clinics NA. 2007in press.
[31]. Bain BJ, Horny HP, Hasserjian RP, Orazi A: Chronic Eosinophilic Leukaemia, NOS. Lyon France, IARC Press, 2017, pp 54–56.
[32]. Vega F, Medeiros LJ, Bueso-Ramos CE, Arboleda P, Miranda RN: Hematolymphoid neoplasms associated with rearrangements of PDGFRA, PDGFRB, and FGFR1. Am J Clin Pathol 2015; 144: 377–392.
[33]. Jackson CC, Medeiros LJ, Miranda RN: 8p11 myeloproliferative syndrome: a review. Hum Pathol 2010; 41: 461–476.;
[34]. Gruss HJ, Brach MA, Drexler HG, et al. Expression of cytokine genes, cytokine receptor genes, and transcription factors in cultured Hodgkin and Reed-Sternberg cells. Cancer Res 1992;52:3353. [PubMed: 1596893]
[35]. Samoszuk M, Nansen L. Detection of interleukin-5 messenger RNA in Reed-Sternberg cells of Hodgkin's disease with eosinophilia. Blood 1990;75:13. [PubMed: 2403816
[36]. Gotlib J: World Health Organization-defined eosinophilic disorders: 2017 update on diagnosis, risk stratification, and management. Am J Hematol 2017; 92: 1243–1259.