ISSN: 0976-3031

Available Online at http://www.recentscientific.com

International Journal of Recent Scientific Research
Vol. 7, Issue, 5, pp. 11321-11324, May, 2016

International Journal of Recent Scientific Research

Research Article

"A HISTOPATHOLOGY STUDY OF GRANULOMATOUS INFLAMMATIONS"-A RETROSPECTIVE AND PROSPECTIVE STUDY OF 4 YEARS

Harshpreet Kaur[1]., Shilpi Sahu[2]., Reeta Dhar[3] and Borkar D.B[4]

[1,2,3,4]MGM Medical College Kamothe, Navi Mumbai

ARTICLE INFO

Article History:

Received 17th February, 2016
Received in revised form 21st March, 2016
Accepted 06th April, 2016
Published online 28th May, 2016

Keywords:

Granuloma, histopathology, FNAC, special stains, etiology

ABSTRACT

This study was conducted to find out the etiology of all granulomatous lesions on Histo-pathology and Cytological studies. Our study was done at MGM Medical college, Kamothe, Navi Mumbai 4 years (2012-2015) . All the cases which were clinically diagnosed and suspected of granulomas, on haematoxylin and eosin stained sections were studied. Special stains like ZiehlNeelsen stain, PAS stain and FiteFaraco stain were done whenever required. The morphological features and special staining helped us to find the specific etiology of Granulomas in 86 cases whereas it could not be determined in 14cases even after special stains. The most common etiology of granuloma in our study was tuberculosis (49cases) followed by leprosy in 15 cases and no specific etiology in 16 cases. This may be due to reason India is a tropical country and tuberculosis is a leadinginfection In our study 25 cases FNAC was done and correlation was found in 18 cases. Hence FNAC is adjuvant to diagnose granulomatous lesion.

INTRODUCTION

The granulomatous inflammatory response is ubiquitous in pathology, being a manifestation of many infections, toxic, allergic, autoimmune and neoplastic diseases, vasculitis, immunological aberration, leukocyte oxidase deficiency, hypersensitivity, chemicals, and also conditions of unknown etiology. Schistosomiasis, tuberculosis and leprosy, all infective granulomatous diseases, together affect more than 200 million people worldwide, and granulomatous reactions to other irritants are a regular occurrence in everyday clinical histopathology. Granulomatous inflammation is best defined as a special variety of chronic inflammation in which cells of the mononuclear phagocyte system are predominant and take the form of macrophages, epithelioid cells and multinucleated giant cells. In most instances, these cells are aggregated into well demarcated focal lesions called granulomas, although a loose, more diffuse arrangement may be found. In addition there is usually an admixture of other cells, especially lymphocytes, plasma cells and fibroblasts.[1]According to Dorland, the term "granulomatous" was expressed initially by Virchow to describe a tumor-like mass or nodule of granulation tissue.[2]

The term Granuloma is a hallmark of human disease of great significance, the most important of which is Tuberculosis,[2] followed by those with leprosy, rhinoscleroma, actinomycosis, fungal infections, foreign body granuloma and granuloma of unknownaetiology. Granuloma is "a focal chronic inflammatory response to a tissue injury, which is evoked by a poorly soluble substance which is characterized by the accumulation and proliferation of leukocytes, prinicipally of the mononuclear type"[3]

The provocative agents of granulomatous inflammation appear to be non-degradable by both neutrophils and non-active macrophages. The actions of polymorphonuclearleucocytes ,non activated macrophages and chemical mediators which are associated with the tissue injury are insufficient to completely digest and eradicate the offending agents. For such a degradation, the action of transformed macrophages which are formed with the help of CD4+T cells is required. The CD4+T cells secrete various mediators such as IL2, IFγ, TNF and lymphotoxin for the transformation of the macrophages into epithelioid cells and giant cells, which are component of Granulomas[4]. All the cases which were clinically suspected and diagnosed of granulomas on haematoxylin and eosin stained sections were studied. Special Stains like ZiehlNeelsen stain, PAS stain, Fite Faraco stain and FNAC correlation were done whereever required. The correlation of histopathology with PCR, serological tests and culture correlation would have helped further.

Corresponding author: **Harshpreet Kaur*
MGM Medical College Kamothe, Navi Mumbai

MATERIALS AND METHODS

A Retrospective & Prospective study of 100 cases of Histopathology of granulomatous lesions in 4 years (2012-2015) period in department pathology will be conducted. The clinical details will be obtained from the medical records department and from histopathological requisition forms accompanying the specimens. The Specimens are received in 10% buffered formalin and then 3-5 microns thick sections are cut and stained with haematoxylin and eosin.

Study Design

Prospective & Retrospective study -4 Years

RESULTS

A Study of 100 histopathologically proven granulomas were done to find their etiology. The most common etiology of granuloma in our study was tuberculosis 49 cases followed by leprosy in 15 cases and no specific etiology in 16 cases. In our study 25 cases FNAC was done and correlation was found in 18 cases. In our study Tuberculosis was most common because of overcrowding and malnourishment. The cervical lymph node was involved commonly because TB is a chronic air borne infectious disease.

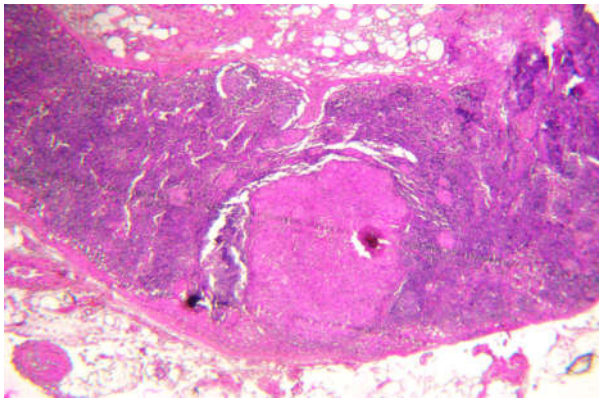

Figure 1(a) H&E Stained Section from cervical lymph node showing large areas of caseous necrosis surrounded by collar of lymphocytes – **TuberculousLymphadenitis** (40x)

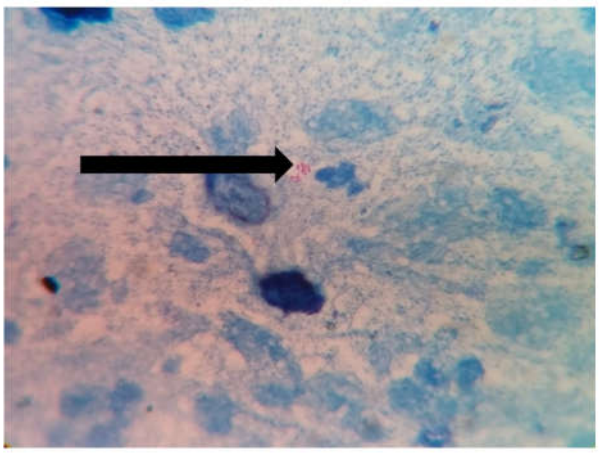

Figure 1(b) ZN Stain shows **AFB positive (10x)**

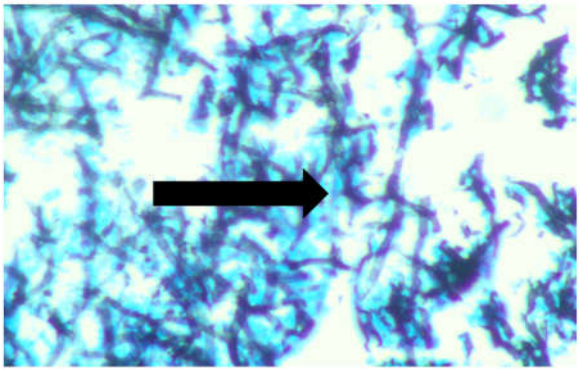

Figure 2 Mucormycosis GMS Stain- Positive

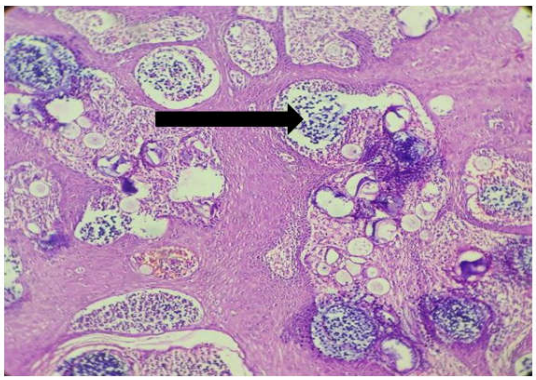

Figure 3 Rhinosporodiosis- H&E Stained section show Cupshaped sporangia with spores

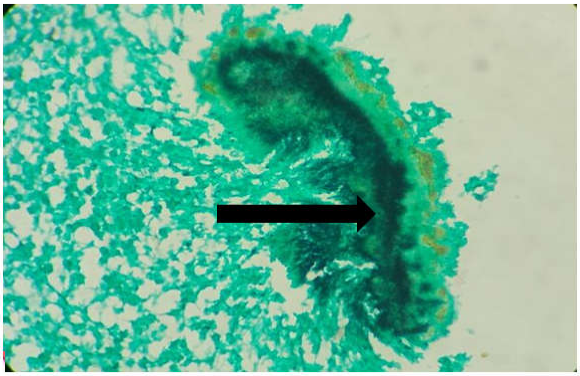

Figure 4 Actinomycosis GMS Stain shows black colonies

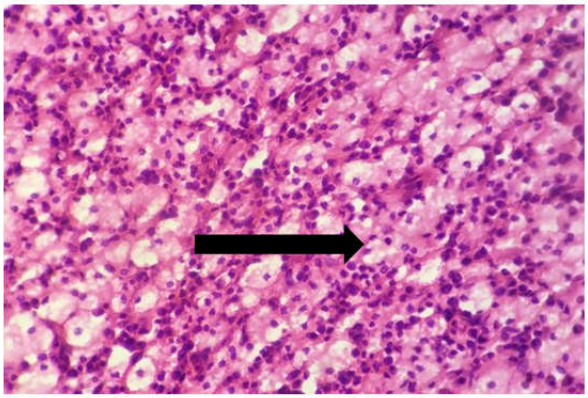

Figure 5 H&E Stained section from **Rhinoscleroma** shows foamy cells and histiocytes

Table no D1 Comparison of Age wise Distribution of granulomatous disease of our study with other studies

Age	Our Study	Jayshreepawal *et al* Baglakot&Hubli, Karnataka	Harish S Premi *et al*, Manglore, Karnataka	Babaria KR *et al*, Rajkot, Gujarat, India	Adhikari RC *et al*
0-9Yrs	4%	5.88%	4%	7.33%	6%
10-19yrs	10%	15.29%	11.40%	12.00%	16.5%
20-29yrs	28%	27.06%	23.60%	29.33%	27.8%
30-39yrs	27%	20.59%	18.16%	20.00%	17.2%
40-49yrs	13%	17.65%	14.90%	12.67%	10.8%
50-59yr	6%	8.82%	18.52%	11.33%	12.2%
60-69yrs	9%	4.71%	8.70%	03.67%	6.5%
70-79yrs	2%	0.00%	00.36%	3.34%	3.1%
80-89yrs	1%	0.00%	00.36%	0.33%	0.00%
Total	100%	100%	100%	100%	100%

Table no-D2 Comparison of Incidence of various types of Granulomatous diseases with other studies

Type of Granulomatous Diseases	Present Study	Jayashreepawal *et al*	Harish S Premietal	Adhikarietal	Patel *et al*	BabariaKR *et al*
Tuberculosis	49%	49.41%	47.26%	61.7%	81%	56.33%
Leprosy	15%	17.65%	12.72%	5%	7%	17.67%
Fungal granuloma	4%	5.88%	8.73%	3.1%	4%	1.33%
Foreign body granuloma	8%	7.65%	8.36%	1.7%	2%	12.67%
Parasitic	2%	0%	1.45%	7%	1%	0.33%
Actinomycosis	2%	1.18%	1.45%	0%	0%	0.33%
Rhinoscleroma	2%	11.76%	5.10%	0%	0%	-
Sarcodiosis	2%		00%	5%	0%	-
Non specific etiology	16%	6.47%	8%	28.9%	5%	11.34%

Table no D3 Comparison of Site wise distribution of granulomas of our study with other study

Site wise distribution of granuloma	Our study percentage	Jayshreepawa letal Study	Harish S Premietal Study	Adhikari Etal Study	Patel et al study	Babaria KR *et al* study
Lymph node	21%	18%	21.46%	41.1%	55%	11%
Skin	44%	31%	24.72%	22%	8%	35%
Bones and joints	8%	8%	18.18%	11.5%	-	8.67%
Respiratory system	1%	17%	9.46%	7.7%	5%	10.67%
GIT Tract	2%	7%	8%	5.5%	5%	21.33%
Male genital system	4%	3%	3.64%	1.4%	2%	
Urinary system	3%	1%		2.4%	-	
Breast	6%	6%	5.82%	0.5%	0.7%	
Thyroid	1%	-	1.46%	0%	-	

Table no 1 Age wise distribution of granuloma in our study

Table1: Distribution according to Age (in Years)		
Age	**Frequency**	**Percentage**
0-9	4	4.0%
10-19	10	10.0%
20-29	28	28.0%
30-39	27	27.0%
40-49	13	13.0%
50-59	6	6.0%
60-69	9	9.0%
70-79	2	2.0%
80-89	1	1.0%
Total	**100**	**100.0%**

Table no 2 Causes of granuloma according to etiology in our study

Causes of Granulomas According to Etiology	Frequency	Percentage
Tuberculosis	49	49.0%
Leprosy	15	15.0%
Actinomycosis	2	2.0%
Sarcodiosis	2	2.0%
Foreign body granuloma	8	8.0%
Rhinoscleroma	2	2.0%
Fungal	4	4.0%
Langerhan Cell Histocytosis	1	1.0%
Non Specific Etiology	16	16.0%
Total	**100**	**100.0%**

Table no 3 Site wise Distribution of Granuloma in our study

Site Wise distribution of Granulomas	Frequency	Percentage
Lymph node	21	21.0%
Skin and Subcutaneous tissue	44	44.0%
Bone and joints	5	5.0%
Respiratory System	2	2.0%
GIT Tract	2	2.0%
Female Genital System	0	0.0%
Urinary System	3	3.0%
Pericardium	0	0.0%
Male Genital System	4	4.0%
Eye and ocular adenxae	1	1.0%
Omentum	0	0.0%
Breast	6	6.0%
Brain	0	0.0%
Synovium	4	4.0%
Thyroid	1	1.0%
Foot Biopsy	1	1.0%
Spine	3	3.0%
Peritoneal Tissue	2	2.0%
Ear	1	1.0%
Total	**100**	**100.0%**

DISCUSSION

A Study of 100 histopathologically proven granulomas were done to find their etiology. Special stains like Fitefaraco stain,

GMS Stain, PAS Stain were done wherever required and FNAC corelation also was done.

CONCLUSION

In our study, study of 100 histopathologically proven granulomas were done to find their etiology. In our study etiology was determined in 86% of cases out of 100 cases. The most common etiology in our study was tuberculosis followed by leprosy and no specific etiology. Tb was most common because of overcrowding and malnourishment. In our study 25 cases FNAC was done and correlation was found in 18 cases. Cooperation between the clinician and the pathologist is more important to derive the greatest benefit from the biopsy. Additional tests like culture, serological investigations and PCR may be necessary to confirm the results.

References

1. Williams G T and Williams W J, Granulomatous inflammation—a review *J ClinPathol*1983; 36: 723-733.
2. Thurlbeck W, Churg A. *Pathology of the Lung* 2nd edition, 1995. Thieme Medical Publishers. Chapter 13, p234.
3. Algood HM, Chan J, Flynn JL. Chemokines and tuberculosis. *Cytokine Growth Factor Rev*. 2003; 14 (6): 467-77.
4. Hirsh BC, Johnson W C. Concepts of granulomatous inflammation. *International Journal of dermatology* 1984 March; 24(2): 90-100.
5. Adams DO. The granulomatous inflammatory response. *American Journal of Pathology* 1976 July; 84(1): 164-187.
6. Chensue SW, Ward P A. Inflammation. In: Damjanov I, Linder J editors. Anderson's Pathology.10th ed. Vol 1.London: Mosby; 1996.
7. Blessing K. Mini- Symposium: Inflammatory skin Pathology: cutaneous granulomatous inflammation. Current Diagnostic Pathology 2005 August; 11 (4): 219-235.

****

How to cite this article:

Harshpreet Kaur.2016, "A Histopathology Study of Granulomatous Inflammations"-A Retrospective And Prospective Study of 4 Years. *Int J Recent Sci Res.* 7(5), pp. 11321-11324.