RESULTS

RESULTS

From our study we had the following results:-

I. **Sex Incidence:-**

From table (4) and Figure (5), we see that, out of 371 patients 314 were males (84.6%), while the females were only 57 patients (15.4%).

Table (4)

Sex Incidence

Number of Patients	Sex	Percentage
314	male	84.6%
57	female	15.4%

II. **Age Incidence:-**Table (5), Figure (6).

Regarding age incidence, we can divide the patients into 5 groups.

Group (1) patients between 70-80 years. There were 44 patients. 11.9% of the total number.

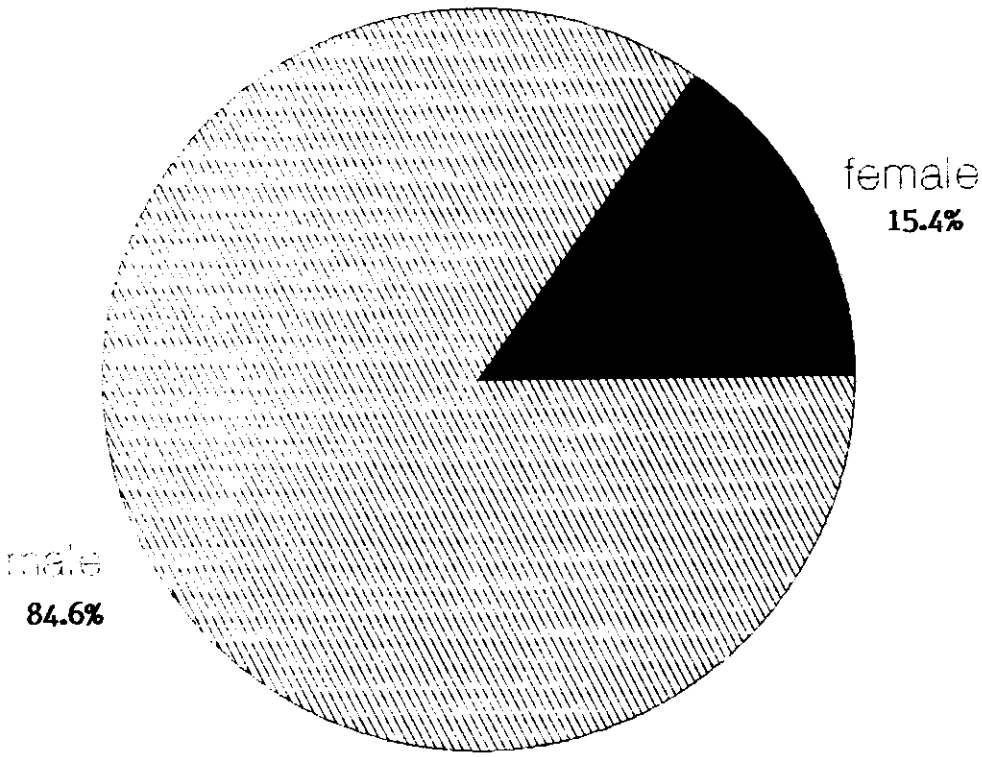


Fig. (5)

The relation between the number of patients and sex incidence

Group (2) patients between 60-70 years. There were 171 patients i.e. 45.9% of the total number.

Group (3) patients between 50-60 years. There were 114 patients i.e. 30.8% of the total number.

Group (4) patients between 40-50 years. There were 28 patients i.e. 7.6% of the total number.

Group (5) patients between 30-40. There were only 14 patients i.e. 3.8% of the total number.

There were no patients below this group of age.

The average age was 60,5 years. The maximum age incidence was between 60-70 years i.e. 45.9% of the total number, while minimum age incidence was between 30-40 years i.e. 3.8% of the total number.

Table (5)

Group of Age	No. of Patients	Percentage
70-80 years	44	11.9
60-70 years	171	45.9

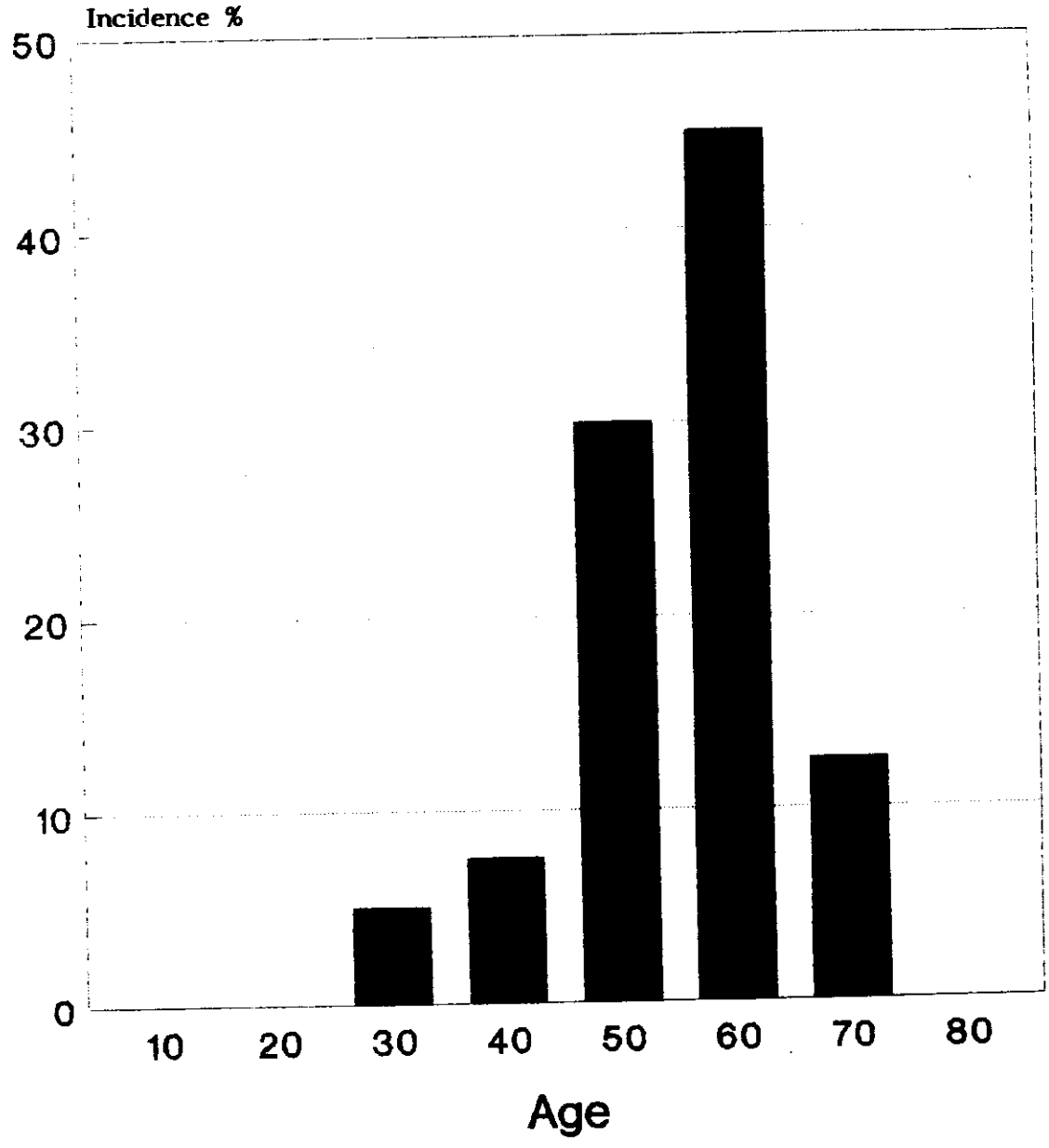


Fig. (6)
The relation between the age and incidence

50-60 years	114	30.8
40-50 years	28	7.6
30-40 years	14	3.8

III. **Special Habits:-.**

A. Smoking Cigarettes:- (Tables 6&7) (Figures 7&8):

As regard smoking, 286 patients were heavy smokers i.e. 77% of the total, while 85 patients i.e. 23% were non-smokers. Studying the sex of the smokers we found that, most of them were males (270 patients i.e. 94.4% of the total), while female smokers were only 15 accounting for 5.6% of the total.

Table (6)

The relation between the smoking and incidence

No. of Patients	Special Habit (Smoking)	Percentage
286	smokers	77
85	non-smokers	23

Table (7)

The relation between the smoking and sex incidence

No. of Patients	Special Habit (Smoking)	Percentage
270	male	94.4
16	female	5.6

B. Alcohol Intake:- (Table 8)(Fig. 9)

As for alcohol, 288 out of 371 patients were non drinker of alcohol beverage accounting for 77.6% of total, while the rest i.e. 83 patients were found lightly alcoholic i.e. 22.4% of the total.

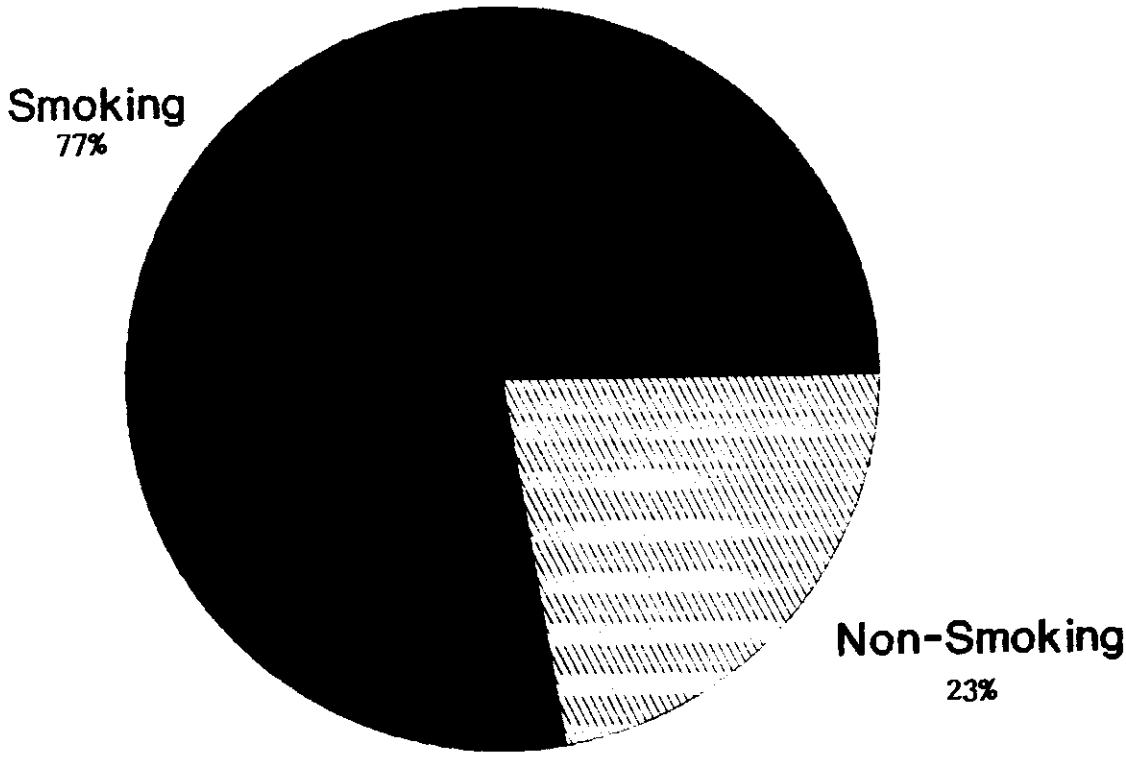


Fig. (7)

The relation between smoking and incidence

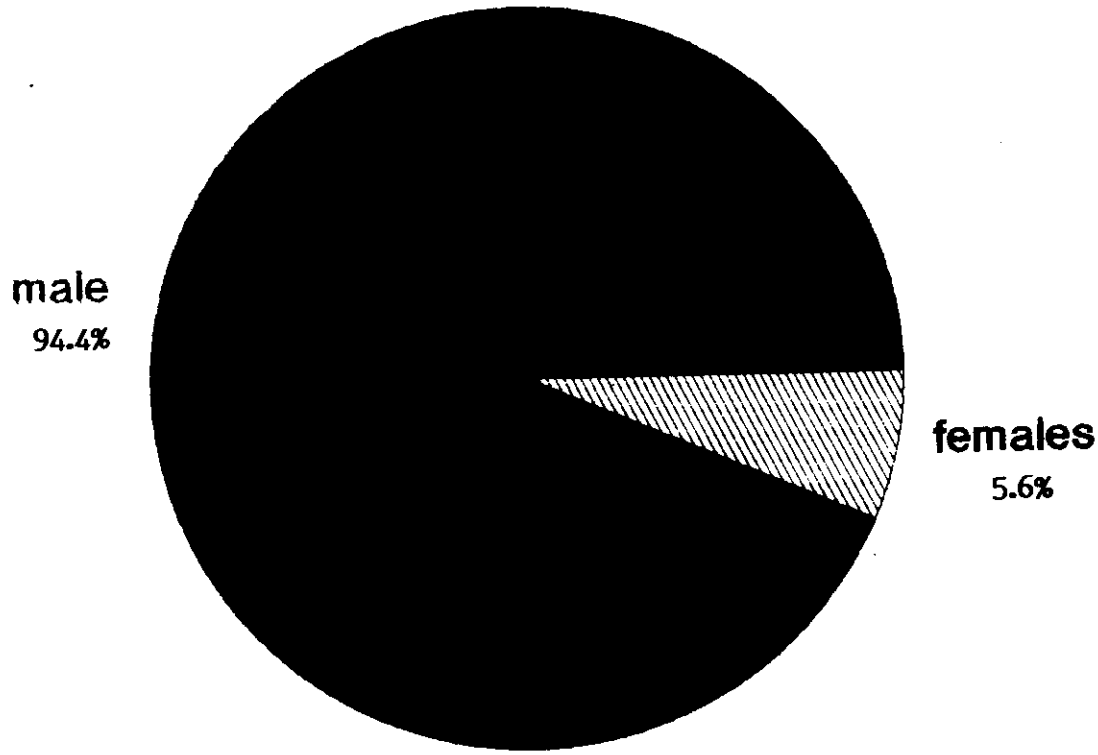


Fig. (8)

The relation of smoking and sex incidene

Table (8)

The relation between number of patients and alcohol intake

No. of Patients	Special Habits (Alcohol)	Percentage
288	non-alcoholic	77.6
83	alcoholic	22.4

IV. **Presenting Symptoms:-** (Table 9)(Fig. 10)

Rearding the symptoms, we found that most of the patients presented with lumps in the neck, (271 patients i.e. 73% of total). Other symptoms were rare, for example, referred ear ache (only 14 patient i.e. 3.8% of total), haemoptysis (only 14 patient i.e. 3.8% of total), dysphagia which appeared in 29 patients i.e. 7.8% of total, Foreign body sensation in 14 patients i.e. 3.8% of total and sore throat in 29 patients i.e. 7.8% of total.

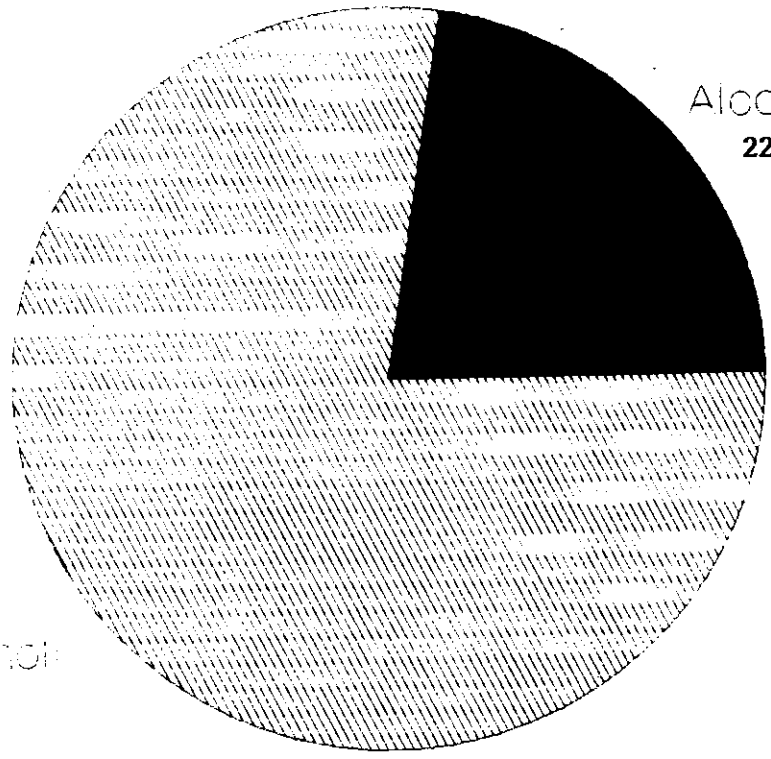


Fig. (9)

The relation between alcohol intake and incidence

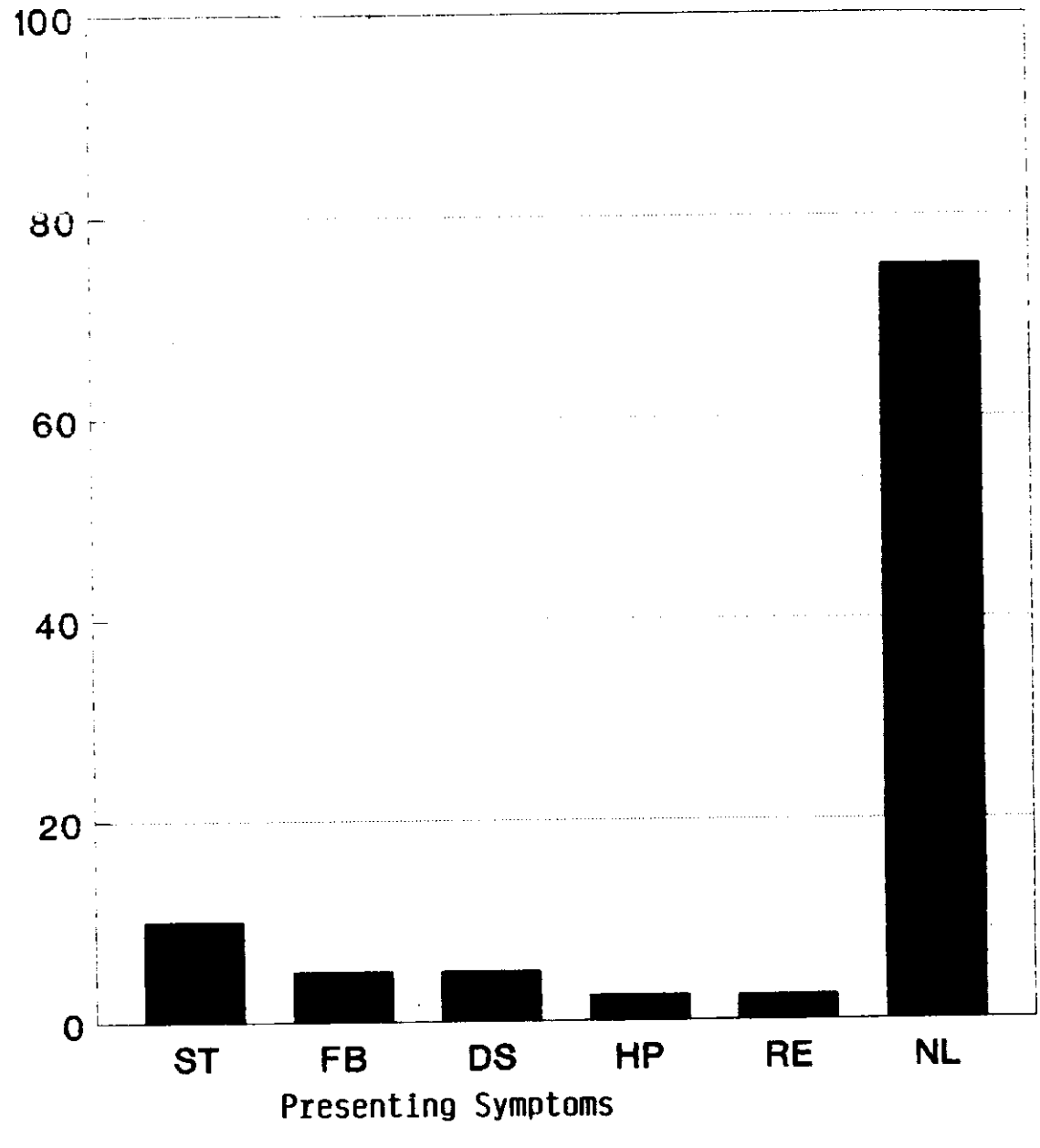


Fig. (10)

ST=Sore throat, FB=Foreign body,
DS= Dysphagia, HP= Haemoptysis.
RE= Referred ear ache,NL=Neck lump

Table (9)

The relation between the presenting symptoms and the number of patients

Presenting Symptoms	No. of Patients	Percentage
1. Lump in the neck	271	73
2. Sore throat	29	7.9
3. Dysphagia	29	7.9
4. Foreign body sensation	14	3.8
5. Referred ear ache	14	3.8
6. Haemoptysis	14	3.8

V. **Signs:-** (Figure 11) (Tables 10, 11, 12)

By examining the patients we found that most of the 299 patiens had a mass in the tonsil. Out of 371 patients the cancer was of the Exophtic type (80.6%), while 72 patients presented with ulcea-tive type (19.4%). The size of the mass varied, 16

patients Presented with tonsillar mass less than 2cm in size i.e. 4.3% of the exophytic total number (T_1). In 74 patients had tonsillar mass more than 2cm but less than 4cm i.e. 20.7% of total (T_2). While in 205 patients presented with tonsillar mass greater than 4cm in size. i.e. 55.3% of the exophytic total number (T_3). In 76 patients there were extensions to the nearby structures i.e. (T_4), the tongue, the soft palate, the mandible...ect.

Twinty eight patients presented with tonsillar mass crossing the midline i.e. 9.4% of the exophytic total number.

Twinty nine patients as regards the ulcerative type, had an ulcer about 1cm in diameter, i.e. 40% of the total number of ulcerative cases (T_1).

Forty three patients showed an ulcer more than 2cm but less than 4cm ulcer, 60% of the total number of ulcerative cases (T_2).

In all cases the ulcer had necrotic base and raised everted edges.

Table (10)

The incidence of presenting signs

No. of Patients	Presenting Signs	Percentage
299	exophytic type	80.6
72	ulcerative type	19.4

Table (11)

The incidence of different exophytic presenting signs

No. of Patients	Presenting Sings	Percentage
16	mass > 2cm T_1	4.3
74	mass between 2cm and 4cm T_2	20.1
205	mass < 4cm T_3	55.3
76	mass with extension to near by structures	20.3

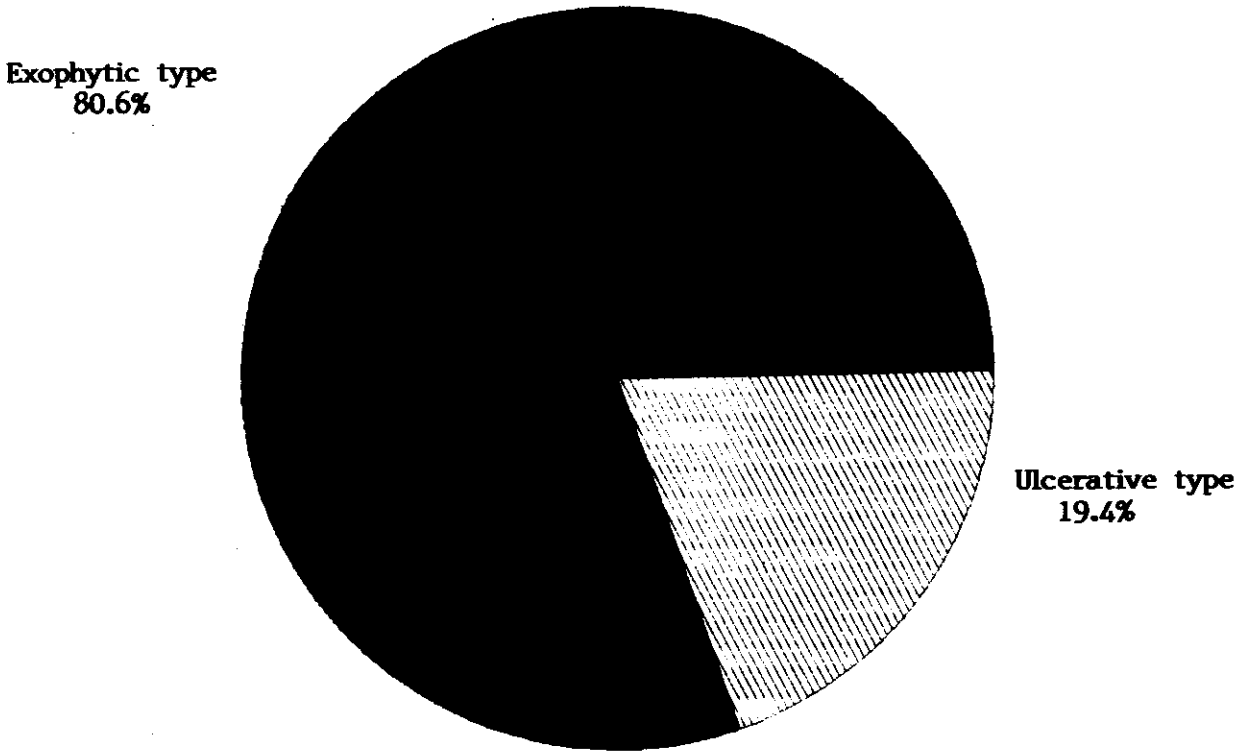


Fig. (11)

The relation between the presenting signs and incidence

Table (12)

The incidence of different ulcerative presenting signs

No. of Patients	Presenting Signs	Percentage
129	1cm ulcer	40
143	2cm ulcer or more	60

VI. **Lymph Node Involvement:**-(Table 12)(Fig. 12)

Cervical lymph nodes were clinically palpable in 296 patients i.e. 80% of the total, while no glands were not palpable in 75 patients i.e. 20% of total. The first group of lymph nodes involved was usually jugulodigastric group.

In six patients with clinically palpable (jugulodigastric) lymph nodes, the posterior group of the cervical lymph nodes were also palpable. Lymph node involved was classified according to AJC into three groups:

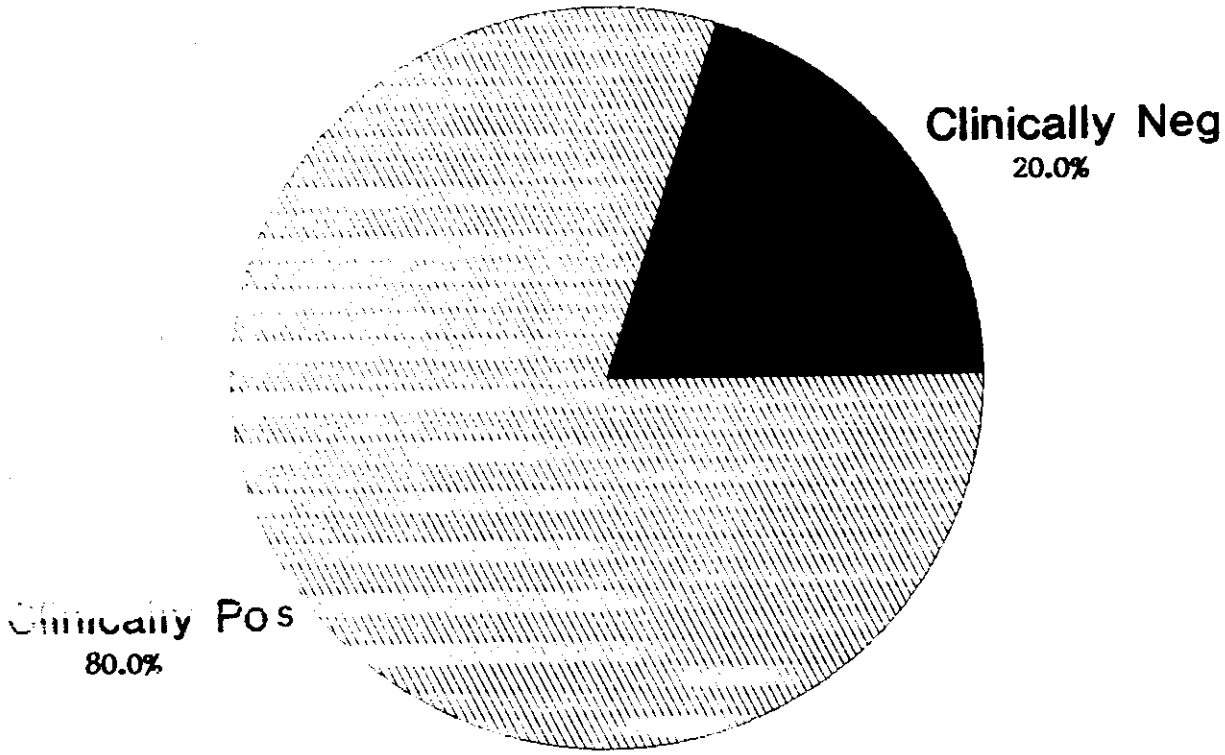


Fig. (12)

The incidence of lymph node involvement

(1) N_1 occurred in 83 patients i.e. 28% of total.

(2) N_2 occurred in 89 patients i.e. 30% of total.

(3) N_3 occurred in 124 patients i.e. 42% of total.

Table (13)

The incidence of lymph node involvement

No. of Patients	Lymph node	Percentage
296	palpable	80
75	not-palpable	20

VII. The Pathologic Diagnosis:- (Table 14).

Using the haematoxylin and Eosin stain, we found that out of 371 patients, 274 patients were diagnosed as squamous cell carcinoma i.e. 73.7% of total cases. As for non-Hodgkin's lymphoma we found 55 patients i.e. 15% of the total cases. Rare and unclassified malignant tumours were found in 37 patients i.e. 10% of the total cases. Two patients had malignant salivary neoplasm i.e. 0.5% of the

total cases, while 3 patients had metastatic tonsillar neoplasms i.e. 0.8% of the total cases.

Table (14)

Types of malignant tonsillar tumours in 371 cases

Type of neoplasm	No. of Patients	Percent
1. Squamous cell carcinoma	274	73.7
2. Lymphoma:		
Hodgkin's	-	-
Non-Hodgkin's	55	15
3. Rare and unclassified malignant tonsillar neoplasms	37	10
4. Malignant salivary neoplasms	2	0.5
5. Metastatic malignant tonsillar neoplasm	3	0.8
	371	**100**

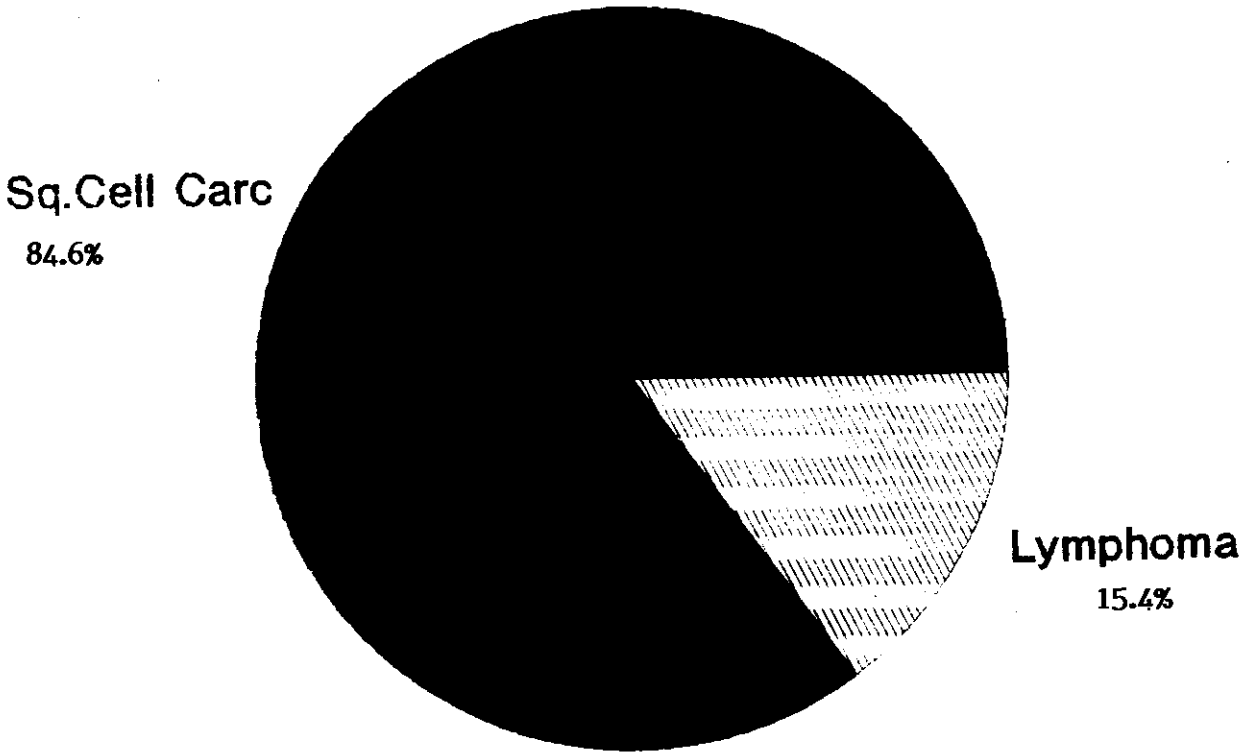
VIII. **Immunologic Diagnosis:-** (Table 15)(Fig. 13)

Twinty two patients were found positive to anticytokeratin and negative to antilymphoma. They can be considered undifferentiated squamous cell carcinoma i.e. 84.6% of the total. While 4 patients were found negative to anticytokeratin and positive to antilymphoma. They can be considered one of the malignant tonsillar lymphoma, accounting for 15.4% of the total cases.

Table (15)

The results of immunologic diagnosis

No. of Patients	Immunologic Diagnosis	Percentage
22	Sq. cell carc.	84.6
4	Lymphoma	15.4



Immunological diagnosis

Fig. (13)

The incidence of immunologic diagnosis

IX. Grading of Undifferentiated tonsillar squamous cellcarcinoma:- (Table 16)

As for the biologic reaction, 1 patient was found to be of grade I i.e. 4.6% of cases, while 21 patients were of grade II i.e. 95.4% of the total cases.

Table (16)

The relation between the grading of undifferentiated squamous cell carcinoma and the intensity of biologic reaction (22) cases

No. of cases	Grade	Intensity of Biologic reaction	pattern
-	grade I	+++ve to ++ve	diffuse
1	grade II	++ve	diffuse or focal
21	grade III	+ve	focal

Fig. (14a)
Undiferentiated tonsillar malignant tumour (squamous cell carcinoma) Haematoxyling and Eosin (H & E) Magnification (40)

Fig. (14b)
Undifferentiated tonsillar malignant tumour of the same patient in Fig.(14)
Slide from the same lesion stained by immunoperoxidase technique +ve for anticytokeratin
Magnification (63)

Fig. (14c)
Undifferentiated tonsillar malignant tumour of the same patient in Fig.(14). Slide from the same lesion stained by immunoperoxidase technique. -ve for T_{200}.
Magnification (63)

Fig. (15a)
Undifferentiated tonsillar malignant tumour (Squamous cell carcinoma). H & E.
Magnification (40)

Fig. (15b)

Undifferentiated tonsillar malignant umour of the same patient in Fig. 15. H&G Magnification (63)

Fig. (15c)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. 15. Immunoperoxidase technique. +ve for anticytokeratin. Magnification (40)

Fig. (15d)

Undifferentiated tonsillar malignant tumour of the same paitent in fig. (15)
Immunoperoxidase technique. -ve for T_{200} . Magnification (40)

Fig. (16a)

Undifferentiated tonsillar malignant tumour (squamous cell carcinoma).
Haematoxylin and Eosin (H&E). Magnification (40)

Fig. (16b)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **(16)**
Immunoperoxidase technique. +ve for anticytokeratin. Magnification (40)

Fig. (16c)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **(16)**
Immunoperoxidase technique. -ve for T . Magnification (40)

Fig. (17a)

Undifferentiated tonsillar malignant tumour (squamous cell carcinoma).
Haematoxylin and Eosin (H&E). Magnification (40).

Fig. (17b)

Undifferentiated tonsillar mmalignant tumour of the same patient in Fig. (17)
Immunoperoxidase technique. +ve for T_{200} . Magnification (40).

Fig. (17c)

Undifferentiated tonsillar malignant tumour of the same paitent in Fig. **(17)**
Immunoperoxidase technique. -ve for anticytokeratin. Magnification (40)

Fig. (18a)

Undifferentiated tonsillar malignant tumour (squamous cell carcinoma). H&E.
Magnification (63)

Fig. (18b)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **(18a)**
Immunoperoxidase technique. +ve for anticytokeratin. Magnification (63)

Fig. (18c)

Unifferentiated tonsillar malignant tumour of the same patient in Fig. **(18a)**
Immunoperoxidase technique. –ve for T_{200} . Magnification (63)

Fig. (19a)

Undifferentiated tonsillar malignant tumour (Malignant lymphoma). H&E. Mag. (40)

Fig. (19b)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **(19a)**
Immunoperoxidase technique. +ve for T_{200} . Magnification (40)

Fig. (19c)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **(19a)**
Immunoperoxidase technique. -ve for anticytokeratin. Magnification (40)

Fig. (20a)

Undifferentiated tonsillar malignant tumour (squamous cell carcinoma). H&E.
Magnification (63)

Fig. (20b)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **(20a)**
Immunoperoxidase technique. +ve for anticytokeratin. Magnification (63)

Fig. (20c)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **(20a)**
Immunoperoxidase technique. -ve for T_{200} . Magnification (40)

Fig. (21a)

Undifferentiated tonsillar malignant tumour (Malignant lymphoma). H&E. Magnification (25)

Fig. (21b)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **21a** Immunoperoxidase technique. +ve for T_{200} . Magnification (40)

Fig. (21c)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **(21a)**
Immunoperoxidase technique. -ve for anticytokeratin. Magnification (25)

Fig. (21d)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **(21a)**
Immunoperoxidase technique. +ve for T 200 .with -ve of the over lying epithelium . Magnification (25)

Fig. (22a)

Undifferentiated tonsillar malignant tumour (squamous cell carcinoma). H & E. Magnification (40)

Fig. (22b)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **(22a)** Immunoperoxidase technique. +ve for anticytokeratin. Magnification (63)

Fig. (22c)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **(22a)**
Immunoperoxidase technique. -ve for T_{200} . Magnification (40).

Fig. (23a)

Undifferentiated tonsillar malignant tumour (Squamous cell carcinoma). H & E.
Magnification (63).

Fig. (23b)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **(23a)**
Immunoperoxidase technique. +ve for anticytokeratin. Magnification (40).

Fig. (23c)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **(23a)**
Immunoperoxidase technique. +ve for anticytokeratin. Magnification (63).

Fig. (23d)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **(23a)**
Immunoperoxidase technique. -ve for T_{200} . Magnification (40)

Fig. (24a)

Undifferentiated tonsillar malignant tumour (Squamous cell carcinoma). H & E.
Magnification (25)

Fig. (24b)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **(24a)**
Immunoperoxidase technique. +ve for anticytokeratin. Magnification (25)

Fig. (24c)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **(24a)**
Immunoperoxidase technique. +ve for anticytokeratin. Magnification (63)

Fig. (24a)

Undifferentiated tonsillar malignant tumour of the same patient in Fig. **(24a)**

Immunoperoxidase technique.

-ve for T_{200} .

DISCUSSION

DISCUSSION

Malignant tonsillar tumours account for about 3% of malignant tumours of the whole body (Barrs, 1979). They are second in frequency to laryngeal malignant tumours among the upper respiratory system (Seda and Snow, 1969 and Whicker and Devine, 1974).

Over 70% of tonsillar malignancies are squamous cell carcinoma of varying degree of histo-differentiation (Batzakis, 1979).

Over 15% of tonsillar malignancy are malignant tonsillar lymphomas, and over 51% of primary lymphoma of the Waldeyer's Ring affects the palatine tonsil (Kapadia and Saul, 1985).

The so called lympho-epithelioma accounts for 5% of malignant tonsillar tumours (chen and Everts, 1975). This term was used to define a tumour with two components, epithelial and lymphoid elements present together. The origin of the tumour was obscured, whether it arosed from one or the other element was uncertain (Regaud and Reverchon, 1921; Schmincke, 1921).

Undifferentiated Tonsillar squamous cell carcinoma of and infilterated with lymphocytes is difficult or sometimes impossible to distinguish from malignant tonsillar lymphoma with the conventional methods even with skilled pathologist (Ewing, 1929; Loke, 1965; Bloom, 1969; Micheals and Hyams, 1977 and Batsakis, 1979).

The proper diagnosis of the nature of the so called lympho-epithelioma is important as, the treatment and prognosis of tonsillar squamous cell carnioma differs markedly from that of tonsillar lymphoma. In case of Squamous cell carcinoma the treatment depends on the stage of the tumour.

In case of T_1 and T_2 radiation and ipsilateral neck block dissection is commonly used. Occasionally in very small lesions are dealt with wide local excession or tonsillectomy. Larger lesions need a wide surgical attack hemi-mandibulectomy with tonsillar area including both pillars, a part of the soft palate, a part of the tongue and ipsilateral block dissection must be done. The defect made is closed by either myocutaneous

flap or deltopectral flap according to the size of the defect (Weichert et al., 1976; Million and Cassissi, 1984).

In moderately advanced T_2 and T_3 we may try radiation up to 5000 Rads, but local failure rate is approximately 20% and 30% respectively. In this case the use of combined therapy of pre-operative radiation then surgery is done.

In T_3 and T_4, if the lesion extends to mandible and not to the lymph node, surgery followed by radiation is the best line of treatment. But if there is lymp nodal involvement, radical radiation is the line of choice (Weichert et al., 1979).

Tonsillar lymphomas are divided into low grade and those of intermediate and high grade according to Working Formulation. The best treatment is a combined regime of chemotherapy (Nitrogen mustard, Vincristine, Procabazine and Prednisone) and radiation. The doses are calculated and repeated according to the stage and leucocytic count (Anderson et al., 1977; Portlock and Rosenberg, 1979).

The prognosis and survival rates were inversely proportionate to advanced age and nodal involvement. As for tonsillar squamous cell carcinoma the 5 years survival rates were, for T_1 81%, T_2 74%, T_3 59% and T_4 20% (Mizano and Diaz, 1986).

As regards the tonsillar lymphoma, the 5 years survival stage (I) 100%, Stage (II) 56%, Stage (III) 66% and Stage (IV) 38% (Fisher et al., 1981).

The precise diagnosis of the undifferentiated tumour whether it is squamous cell carcinoma or lymphoma helps in the choice of line of treatment which gives the patient the best chance for cure.

Only when using electron microscopy, the epithelial origin of the tumour is confirmed (Svoboda et al., 1967). This problem, urged us to find a new technique which is simpler, accurate and can be done in ordinary laboratories using light microscopy. We used tumour markers for both epithelial and lymphoid cells. Anticytokeratins for malignant epithelial cells and Antilymphoma (T_{200}) for malignant lymphoid cells were used.

The cytokeratins are epithelial cell products and are not found in cells of mesenchymal origin (Mardi and Barwick, 1982). They are most complex intermediate-sized filaments, constituting a family of water insoluble proteins. Until now at least 20 different human cytokeratin polypeptides have been recognized (Schlegal and Mcleod, 1980; Gown and Vogle, 1984).

Some of these polypeptides may be specific for a distinct type of epithelial cells, others may be specific for differentiation of stages of tumours (Moll et al., 1982; Van Mujen and Ruiter, 1984).

The body produces antikeratins of specific type but, Sun and Green, 1979 had found that AE, and AE_3 monoclonal antikeratin antibodies can recognize keratins of the Acidic (A) and Basic (B) subfamilies, respectively. The results also indicate that a combination of these two antibodies can recognize all known human epithelial keratins. Therefore, a pool of AE_1 that stains the epidermal basal layer and almost all epithelial IgG_1 and AE_3 that stains the entire epidermis and all epithelial IgG_1 provides an antikeratin an

tibody preparation that is broadly reactive with numerous keratins and less likely to produce false negative results in the identification of epithelial cell. AE_1 and AE_3 antibodies have shown to be highly specific for keratins with no known cross reactivities with any other cytoskeletal elements. The antibodies are also highly stable and have been used successfully for immunohistochemical staining of formaldehyde-fixed, paraffin embedded tissue sections.

Because of all the previous advantages the anticytokeratin was chosen in this work.

Also, the human lymphocytes (Tand B Cells) have surface antigens which are glycoproteins of high molecular weight. Chemical characterization shows that these glycoproteins are the human homologues of murine T_{200} glycoproteins. Recently, monoclonal antibody specific for the structural form of T_{200} glycoproteins has been obtained. This glycoprotein is found in various amounts on the majority of human haemopoietic cell lines. A similar broad distribution was found in

other lymphoid and haemopoietic tissues examined (e.g. the tonsil)(Omary and Battifora, 1980).

In common with most cell surface glycoproteins, the functions of T_{200} glycoprotein and its homologue are not know. The fact that the glycoprotein appears to be restricted to haemopoietic cells in several species strengthens the belief that the molecules serves some role related to the functions of this cell type (Omary and Battifora, 1980).

Monoclonal antibodies produced using this glycoprotein react with antigens present on human white cells. These reagents differ from other monoclonal antibodies of similar specificity in that antigens are resisten to conventional tissue fixation and embedding procedures. This reagents can be used in immunohistochemical staining of paraffin-embedded tissue sections.

When the unusual property of these antibodies were recognized, the possibility was raised that they might be applicable to the routine histopathological diagnosis of human lymphoma.

Warnke and Gatter, 1983 stressed that they are indeed of value in this way, since cells from all patients with lymphoma gave staining, whereas no cells from patients with malignancies of non-haemopoietic origin were labeled by the antibody.

Accordingly, this antilymphoma (T_{200} was the primary antibody of choice in this aspect.

Using the chosen tonsillar tumour markers we have to consider using one of the immunologic techniques. There are two common famous techniques accepted and successful that can be used, the immunoperoxidase and the immunoflourescence techniques.

The immunoflourescence techniques, have the following disadvantages:

The electron microscopy have to be used, cryostat used destroys the morphology of the tumour, no permenant slide is obtained, a fresh preparation is needed and a certain degree of non-specific tissue reaction in unavoidable (Taylor, 1978). While immunoperoxidase method has the following advantages:

The ordinary light microscopy is used, microtome used preserves the morphology of the tumour, a permenant slide is obtained, a fixed tussue in formaldehyde and paraffin embedded is only needed and non-specific tissue reaction does not occur. Hence the immunoperoxidase technique was chosen in our study.

There were major problems during the use of the immunoperoxidase with Avidin-Biotin method technique on formalin fixed paraffin embedded sections. The results of immunoperoxidase staining are only as good as the tissue specimen used. If the tissue is improperly fixed and processed, a variety of artifacts will results. Since the primary purpose of staining is to localize an antigen, then the main concern in fixation is to preserve that antigen. The antigen must be fixed, available and accessible to the primary antibody. If the antigen is not fixed, it will be washed out of the specimen and no staining will result. If the surrounding cells are not fixed, the resulting poor morphology, will interfere with proper interpretation. On the other

hand, overfixation can cause severe problems such as masking or denaturation of the antigen.

After fixation, tissue specimens should be embedded in pure paraffin because it can be completely and easily removed from the tissue at the time of staining. If the embedding media is heated to temperatures above 62°C, the plastic will start to form polymers, which are very difficult to remove from the tissue. In the oven, temperatures should not exceed 60°C to prevent antigen denaturation and damage to cellular morphology. If these precautions are observed, there should be no problem with non specific background.

Controls in our study were done by omitting the primary antibody and replacing it by phosphate buffer sabine. The buffers, were used for dilution, rinsing and wash out baths, in immunoperoxidase technique, phosphate buffer saline (PBS) was used in our study.

The anticytokeratin used in this work was supplied in a borate buffered saline solution (pH 8.0) which

contains 0.1% sodium azide, so it was very important that the tissue sections should be washed throughly to remove the azide which would otherwise interfere with the peroxidase activity.

Okon, 1985 said that it should be pointed out in determining whether or not the positively stained cells represent neoplastic cells. We depend upon morphologic distinction and comparison with Hx & E sections.

The incubation time, antibody dilutions, non-specific backgrown staining and specific staining intensity are all inter-related. The longer the incugation time, the higher the antibody dilutin. The more dilute the antibody the lower the non-specific stain, the greater the contrast with the specific stain, making interpretation easier. Often the poorly differentiated or metastatic tumours will not stain as intensely as with differentiated tumour tissue. To achieve greater staining intensity on these specimens without changing the antibody dilution, the incubation time may be increased. In our study, all these factors were kept in mind to make perfect staining,so that best optimum dilutions and incubation times are achieved. if

for any reason, staining procedure must be interrupted the slides were put in buffer bath. This goes with Bourne, 1983, who said that slides can be held in buffer for several hours or even several days with no decrease in staining intensity. The intensity of counterstain can be regulated by increasing or decreasing the staining time.

The ABC method used in this study solved the major problem of background staining and it allows the differentiation between extracellular and membranous staining as it gives no background staining.

Using the Haematoxylin and Eosin technique of staining we got the incidence of each type of malignant tonsillar neoplasm in table (16). It differs a little from the work of Crawford, 1979 except for, in our work we found no cases of malignant melanoma or granulocytic sarcoma and also no Hodgkin's lymphoma cases met with.

These differences can be explained as there is a great difference in the period of the study between 1945-1979 and the number of cases 1535 cases.

As regards, the incidence of undifferentiated tonsillar squamous cell carcinoma, in our work, it was 7%, while in Chen and Everts, 1975 study differs a little, they found it 5%.

The sex incidence of malignant tonsillar tumour; we found that there were a male predominance, with male to female ratio 5.5:1. This ratio is more or less similar to the results of Lipkin and Miller, 1985 who found male to female ratio 6.8:1. Stell, 1976 found that male to female ratio was 5:1. In the study of Mizona and Diaz, 1986 a male to female ratio was found to be 3:1. The increased percentage of male over female incidence can be attributed to the special habits in patients, namely heavy smoking and to much lesser extent alcohol intake, being far more common in males.

The age incidence, we found no patients below the age of 30, while minimum incidence (3.8%) was in the third decade, and the maximum incidence (46.1%) in the sixth decade of life. The average age of the patients was 60.5 years. This goes with the results of

Stell, 1976 who found that the neoplasm rarely occured below the age of 50 years. Also Krause and Lee, 1973 found that malignant tonsillar neoplasm occured below 40 years, with maximum age between 60-70 years. While Lipkin and Miller, 1985 issued a study on patients affected with malignant tonsillar tumours and found the tumours below the age of 40 years and the average age of his patients was 36.3 years.

We found that 77% of our patients were heavy smokers, while 23% were non-smokers. Most of the smokers were males (95%). This matches with results of Lipkin and Miller, 1985; Ogrady and Doyle, 1985; Wynder and Hoffman, 1976; Ballantyne and Johston, 1977 and Buttomely, 1979. The higher incidence of malignant tonsils in smokers can be explained by the fact that tobacco smoke has several substances which act as carcinogenic promotors or initiators. When these substances are dissolved in saliva, they become concentrated in the dependent parts of the oral mucous membrane causing destruction of the protective layer of

keratin making the oral mucous membrane vulnerable to carcinogenic agents found either in nicotine and tar or other chemical substances.

We found that 27% of the patients drink alcoholics while the rest are non-alcohol drinker.

This does not match with Lipkin and Miller, 1985 and Mushinki and Spivak, 1977. They got triple our results. This can be attributed to our religous belief which forbids alcohol drinking.

The main complaint of our patients was lump in the neck, (73% of the total numbers of our patients). Other symptoms were less frequent for example sore throat was complained of 7.8% of our patients, feeling of foreing body in 3.8%, dysphagia in 7.8%, Haemoptysis 3.8% and refered ear ache in 3.8%. These results do not match with the results of others (Stell, 1976; Barrs, 1979 and Givens, 1981) in which the presenting symptoms was a lump in the neck in only 10%. This can be explained by low socio-economic standard of our patients who dont seek medical advice except late in the disease.

The carcinoma in our cases was of the exophytic type in 80.6% of cases, while ulcerative tonsillar type accounted for only 19.4% of cases. Most of the exophytic masses arise from the upper pole, the size was from 2cm or less (T_1) (4.3% of cases) from 2cm to 4cm (T_2) in 20% of total, while masses more than 4cm accounted for 55.3% of cases. There were masses extended to the near- by structures (T_4) in 20.3%.

These results differs greatly from the results of Terz and Far, 1967 and Ballenger, 1985. As regards exophytic mass Terez found that 40% were T_1 i.e. masses 2cm or less, 20% of total presented in T_2 group, i.e. masses between 2-4cm, while T_3 i.e. masses more than 4cm accounted for 40% of total. Ballenger, 1985 found that masses of 2cm or less T_1 50%, while masses between 2-4cm T_2 in 30%, and masses over 4cm T_3 in 20% of total cases. While Mizono and Diaz, 1986 found that (T_1) accounted for 9.9%, (T_2) equal 33.3%, (T_3) accounted for 40.9% and (T_4) equal to 15.9% of the total.

As regards the ulcerative type, the ulcers were

2cm or less were in 40% of (T_1) while T_2 i.e. ulcers from 2cm to 4cm, occured in 60%. These ulcers usually were situtated near the upper pole, with raised everted edges and necrotic floor.

Terez and far, 1967 and Ballenger, 1985 reported that the ulcers below 2cm (T_1), were found in 60% of their patients while ulcers between 2-4cm (T_2) were found in 40% of the patients. These differences in results can be explained as well by the late presentation of the patients.

Again from our study we found that 80% of our patients have clinically palpable lymph nodes at the time of presentation. This is in contrast with the results of Stell, 1976 who found palpable glands in only 60% of his patients. Also Lindberg, 1976 found that 41% of his patients had palpable lymph nodes, Mendenhall, 1980 found that only 65% of his cases had palpable gland and that Orgarady et al., 1985 found 65% of his patients had palpable lymph nodes.

The difference between the results of our study

and results obtained by Lindberg, 1976; Stell, 1976; Mendenhall, 1980 and Orgrady et al., 1985 may be due again to the late presentation of the patients to the medical advice.

In our study we followed American Joint Committee, in nodal classification. 12.5% of cases presenting with galand, can be grouped in N_1 category, 43.75% of patients can be put in N_2 category while 43.75% can be grouped as N_3. These results of lymph node involvement differ greatly from results of Stell, 1976 who found that N_1 group accounted from 58.3%, N_2 group equal to 16.6% while N_3 was found only in 25.1% of the patients.

These differences can be explained as our patients seek medical advice late in the disease.

By using the immunologic technique immunoperoxidase with tumour markers anticytokeratin (AE_1+ AE_3) for detection of epithelial nature of the tumour cells and antilymphoma (T_{200}) for detection of malignant lymphoid nature of the tumour cells, we found that most of the undifferentiated tonsillar malignant tumour to be

squamous cell carcinoma i.e. in 84.6% of the patients, while only 15.4% were found to be malignant tonsillar lymphoma. These results matches with that of Batzakis, 1979 who found that over 70% of tonsillar malignancy was squamous cell carcinom of extreme degree of undifferentiation. ALso, Kapadia and Saul, 1985 found that over 15% of tonsillar malignancy were tonsillar lymphoma. Using anticytokeratins, in this technique the intesity of staining with anticytokeratin can be used in both determining the keratin (epithelial) origin of the tumour as the staining intensity correlates with the degree of tumour cell differentiation i.e. the more the degree of differentiation the more strongly is the positive reaction.

This idea was held by Shlegal and McLeod, 1980; Mardi and Barwick, 1982 ; Ziegels and Nadji, 1984, they showed that the increase in the intensity of the reaction goes with the increase in the differentiation of the tumour cell.

In our work, we found also that the distribution of positive staining with anticytokeratin in non keratinizing undifferentiated tonsillar squamous cell carcinoma was both membranous and cytoplasmic reactions. This can be explained by the fact that the intensity and the distribution of staining general correlate with the quantity and distribution of tonofilament bundles (Banks et al., 1975; Schegal and McLeod, 1980 and Battifora et al., 1980).

As the keratins are focally distributed in the cells and not diffused, the reaction was in the form of focal pattern. Thus it is possible that a small biopsy of such lesion might give a false negative result when analysed histochemically but when using anticytokeratin with immunoperoxidase thechnique we may detect any amount of small epithelial malignant cells.

The positive results with anticytokeratin antibody indicates an epithelial tumour, while negative results is much less specific and can indicate an origin from brain, nerve connective tissue, lymphoid tissue i.e. non-epithelial.

As regards the grading of the undifferentiated tonsillar squamous cell carcinoma, using biologic parameters, we found that 4.6% of the total can be classified in grade II, while 95.4% of the total were of grade III. In grade II the pattern of reaction was in focal manner and the intensity of the biologic reaction was ++ve, while in grade III the pattern of reaction was also in focal manner, but the intensity of biologic reaction was +ve only.

These results suggest that in all cases of undifferentiated tonsillar squamous cell carcinoma were of focal pattern of reaction due to distribution of keratin in a focal manner in the epithelial cell tumour. The intensity of the biologic reaction was inversely proportianated to the degree of undifferentiation of the tumour. The more undifferentiated the tumour was, the less the intensity of the biologic reaction and vice versa.

This grading is applicable only in cases of epithelial tumours only and not to lymphoid tumours.

In order to diagnose tumours of lymphoid origin we applied the second antibody to our cases T_{200} (common leucocytic antigen). There were two difficulties brought about by Pizzolo, 1980, the first is when tissues are fixed in formaline the use of some of monoclonal antibodies may not be possible. This difficulty is overcomed in our study by the use of T_{200} which is resistent to the stiffness of tissue processing and embedding. The second difficulty, is the difficulty to interpret the significance of negative reaction since these may equally well represent a tumour of non-lymphoid origin. Such problem can be avoided by using the antileucocyte common antibody in conjugation with anti-epithelial antibody, so that a tumour negative with one antibody is likely to be positive with another.

All positive reactions with T_{200} were located on the surface membrane of the lymphocytes, although some positive staining inside the cells may be detected. This positive staining, results from diffusion of the

antibody from the surface to the cytoplasm of the cell because T_{200} glycoprotein is surface and not cytoplasmic.

In his study, Lauder and Holland, 1984 stated also that antileucocyte to common antibody (T_{200}) was usually confined to the cell surface membrane.

There were no relation between the grade of lymphoma and the intensity of the positive staining. In general, the anticytokeratin antibodies are more useful than common leucocyte antibodies (T_{200}) as the first detect the nature of the tumour cell whether it is of epithelial or non epithelial also gives an idea abot the grading or degree of differentiation, the more positively the tumour cell are stained, the more differentiated is the epithelial tumour. While using T_{200}, positive results indicate the lymphoid origin of the tumour but no grading of lymphoma can be obtained. Thus, by using both types of antibodies, a diagnosis in all undifferentiated cases of malignant tonsillar neoplasms can be achieved.