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Comparison of Oral Squamous Cell Carcinoma in Younger and Older Patients in India

M. Kuriakose, M. Sankaranarayanan, M.K. Nair, T. Cherian, A.W. Sugar, C. Scully and S.S. Prime

This study examines the demographic, aetiological and clinico-pathological features of 37 patients with oral squamous cell carcinoma (SCC) who were less than 35 years old and a comparable number of patients who were greater than 60 years old. The study was undertaken at the Regional Cancer Centre, Trivandrum, India, between 1988 and 1990. In patients younger than 35 years old, oral SCC occurred more commonly in females, was apparent in all social classes and was associated with fewer aetiological factors. The tumours manifested predominantly as invasive lesions affecting the tongue and there was early spread to lymph nodes. By contrast, in patients older than 60 years of age, oral SCC was more common in males, occurred more frequently in social classes III and IV and was always seen in association with smoking, alcohol or pan chewing. These latter tumours presented as exophytic lesions of the buccal mucosa or gingivae and spread late to lymph nodes. The results indicate that the biological behaviour of oral SCC in young patients may be distinct from that occurring in older patients.
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INTRODUCTION

ORAL SQUAMOUS cell carcinoma (SCC) is seen predominantly after the fifth decade of life [1, 2], although recent data indicates that the average age of cases is declining [3]. Whilst a number of studies [4–6] have suggested that the occurrence of mouth cancer is falling, it is disturbing to note that oral cancer in the young is increasing, for example in the United States [7], Scandinavia [8] and Scotland [3]. Indeed, oral cancer rates are increasing in most of the areas where records are available [9, 10].

Oral cancer in young adults may be dissimilar from that in an older population. Although the site prevalence is common to all patients with oral cancer [11–17], it has been reported from the Regional Cancer Centre, Trivandrum that, unlike cancer in the older population, no significant habits such as tobacco smoking, alcohol or Betel quid (Pan) chewing are seen in younger patients [17]. Furthermore, oral cancer in younger adults tends to be more frequently anaplastic [12] and metastatic [18], findings which are likely to account, in part, for the poor patient prognosis (2 year survival of 57%) in younger patients [19].

Apart from the above isolated findings, no attempt has yet been made to analyse all of these parameters in a single large series. The objective of this study, therefore, was to elucidate clinical and pathological features of a younger Indian population with oral SCC.

Correspondence to S.S. Prime.
S.S. Prime and C. Scully are at the Department of Oral Medicine, Surgery and Pathology, Bristol Dental Hospital and School, Lower Maudlin Street, Bristol, BS1 2LY; M. Kuriakose and A.W. Sugar are at the Department of Oral and Maxillofacial Surgery, St Lawrence Hospital, Chepstow, U.K.; and M. Sankaranarayanan, M.K. Nair and T. Cherian are at the Regional Cancer Centre, Trivandrum, India.
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PATIENTS AND METHODS

Patients aged 35 or younger with oral SCC [ICD-9: lip (140), tongue (141), gum (143), floor of mouth (144) and unspecified parts of the mouth (145)] who presented at the Regional Cancer Centre, Trivandrum, India between 1988 and 1990 were analysed for the clinical, pathological features and treatment response. Out of a total of 2046 patients, 37 patients were younger than 35 years and this formed the study group (Table 1). A similar number of patients with oral SCC who were over 60 years were selected randomly and used as the control group (Table 2). Only patients with histopathologically confirmed SCC were included in the study.

The following parameters were examined: (1) age and sex distribution; (2) social class; (3) history of any known aetiological factors; (4) site of tumour; (5) clinical staging [20]; (6) morphological type of tumour and histopathological grading classification [20]; (7) treatment measures and response.

Statistical analysis of the data was carried out using the χ^2 test with $P<0.05$ being taken as significant.

RESULTS

Age and sex distribution

The age distribution in the <35 age group ranged from 21 to 34 with a mean age of 27.7 years. The >60 age group had an age range of 60–86, with mean age of 64.1 years.

20 of the total 37 in the <35 age group were females; the sex ratio was 1:1.2 in favour of females. The sex ratio in the >60 age group, however, was reversed at 2.7:1 in favour of males; 27 patients in this group were male and 10 female (Fig. 1). Thus, there were significantly ($P<0.03$) more females in the <35 age group than the >60 age group.

Table 1. Data of patients <35 years old

No.	Age/Sex	Social class*	Aetiology†			Pathology‡			Clinical stage§				Treatment	Outcome‖	
			Alcohol	Smoking	Pan	Site	Histology	Morphology	T	N	M	Gradc		Response	Status
1	34/M	IV	—	—	4×12	T	G2	EX	3	2c	0	IV	R+C	PR	AD
2	31/F	IV	2×8	10×13	12×13	G	G2	EX	4	1	0	IV	S	CR	NED
3	32/M	III	—	48×10	—	T	G1	IN	3	1	0	III	R	PR	AD
4	24/M	III	—	—	—	T	G1	IN	2	1	1	IV	R	PD	DD
5	25/M	II	—	—	—	T	G1	EX	4	2c	0	IV	R+C	PD	DD
6	34/M	IV	—	15×14	3×9	B	G1	EX	1	0	0	I	S	CR	NED
7	27/M	IV	2×13	—	3×13	B	G1	IN	3	1	0	III	R+C	CR	NED
8	32/M	IV	2×8	20×17	6×7	B	G3	IN	3	1	0	III	S+R	PR	AD
9	23/F	IV	—	—	—	T	G2	IN	1	1	0	III	S	CR	NED
10	27/F	IV	—	—	—	P	G1	EX	1	0	0	I	R	CR	NED
11	27/F	I	—	—	—	T	G1	IN	2	1	0	III	R+C	CR	NED
12	25/F	I	—	—	—	T	G1	EX	3	3	0	IV	R	PR	AD
13	33/F	I	—	—	—	T	G1	EX	2	0	0	II	R+C	PR	AD
14	24/F	II	—	—	—	T	G1	IN	1	1	0	III	R	CR	NED
15	20/F	II	—	—	—	T	G3	IN	4	2c	0	IV	R+C	PD	AD
16	34/F	III	—	—	—	T	G2	IN	3	1	0	III	S+R	PR	AD
17	30/F	II	—	—	—	T	G1	IN	3	2b	0	IV	S+R	PD	DD
18	20/M	IV	—	—	—	T	G1	IN	3	2c	0	IV	S+R+CPR		AD
19	33/M	IV	—	—	—	T	G2	IN	2	0	0	II	S	CR	NED
20	23/F	III	—	—	—	T	G2	IN	3	1	0	III	S+R+CPR		AD
21	30/F	III	—	—	—	T	G1	IN	3	0	0	III	R	CR	NED
22	31/F	III	—	—	—	T	G1	IN	3	0	0	III	R+C	CR	NED
23	26/M	IV	—	—	—	T	G1	IN	1	1	0	III	S	CR	NED

24	30/M	II	—	12 × S	8 × 10	T	G3	IN	2	1	0	III	R	CR	NED
25	34/M	IV	—	20 × 14	2 × 5	G	G2	EX	2	1	0	III	R	CR	NED
26	27/M	III	1.5 × 13	—	3 × 13	B	G1	IN	2	1	0	III	R+C	CR	NED
27	24/F	II	—	—	—	T	G1	IN	2	3	0	IV	S+R	PR	AD
28	26/F	III	—	—	—	B	G1	IN	3	1	0	III	R+C	PD	AD
29	25/F	II	—	—	—	T	G2	IN	1	1	0	III	S	CR	NED
30	21/F	III	—	—	—	G	G3	IN	4	3	0	IV	S	PD	AD
31	24/M	IV	—	—	—	T	G1	IN	3	1	0	III	S+R	PR	AD
32	28/M	IV	—	—	4 × 13	B	G1	EX	3	1	0	III	R+C	CR	NED
33	25/F	IV	—	—	—	G	G2	IN	3	3	0	IV	R	PD	AD
24	27/M	IV	—	—	—	T	G2	IN	2	1	0	III	S+R	PR	AD
35	28/F	IV	—	—	—	T	G2	IN	2	1	0	III	R	PD	DD
36	28/M	IV	—	—	—	B	G1	EX	3	1	0	III	R	CR	NED
37	34/F	II	—	—	—	T	G1	IN	1	0	0	I	S+R	CR	NED

*Social-economic class. I, professional; II, managerial and lower professional; III, non-manual or manual skilled and partly skilled; IV, unskilled [33].
†Aetiology. Number of units of alcohol per day; number of cigarettes per day; number of Pans per day. Data is expressed as a multiple of years of exposure (i.e. 2 × 8 reflects 2 units of alcohol per day for 8 years).
‡Pathology. Site—tongue (T); gingivae (G); buccal mucosa (B); palate (P); lip (L). Histology—well-differentiated (G1), moderately differentiated (G2), poorly differentiated (G3) and undifferentiated (G4) oral SCC; grade cannot be assessed (GX). Morphology—exophytic (EX); infiltrative (IN).
§Clinical stage. Tumour size (T)—<2 cm (1); 2–4 cm (2); >4 cm (3); tumour fixed to adjacent structures (4). Nodal metastases (N)—no regional lymph node metastases (NO); metastases in a single ipsilateral node of <3 cm diameter (N1); metastases in a single ipsilateral node of 3–6 cm diameter (N2a); metastases in multiple ipsilateral nodes and <6 cm diameter (N2b); metastases in bilateral or contralateral nodes and <6 cm diameter (N2C); metastases in any node >6 cm diameter (N3).
Distant metastases (M). Grade—T1, NO, MO (I); T2, NO, MO (II); T3, NO, MO or T1–2, N1, MO (III); T4, NO, MO or T1–3, N2/N3, MO or T1–2, NO-3. M1 (IV).
‖Outcome. Treatment—surgery (S); radiotherapy (R); chemotherapy (C). Response—complete response (CR); partial response (PR); progressive disease (PD); stable disease (SD); failed follow-up (FF). Status—no evidence of disease (NED); alive with disease (AD); died with disease (DD); failed follow-up (FF).

Table 2. Data of patients >60 years old

No.	Age/Sex	Social class*	Aetiology†			Pathology‡			Clinical stage§				Treatment	Outcome‖	
			Alcohol	Smoking	Pan	Site	Histology	Morphology	T	N	M	Grade		Response	Status
1	62/M	III	—	20×44	10×22	T	G1	IN	1	0	0	I	R	CR	NED
2	78/F	IV	—	—	5×60	P	GX	EX	2	1	0	III	R	PR	AD
3	62/M	IV	—	8×42	10×50	T	G1	EX	2	1	0	III	S	CR	NED
4	67/F	IV	—	—	2×10	B	G1	EX	3	1	0	III	S+R	CR	NED
5	65/F	III	—	—	3×45	B	G1	EX	2	0	0	II	R	CR	NED
6	64/M	IV	2×2	50×30	1×20	T	G2	IN	2	0	0	II	R+C	CR	NED
7	65/M	IV	—	30×50	—	G	G2	EX	3	0	0	III	S+R	CR	NED
8	68/F	II	—	—	—	T	G1	IN	2	1	X	III	S	CR	NED
9	60/M	IV	—	—	5×48	B	G1	EX	2	1	0	III	R	CR	NED
10	66/M	IV	—	—	5×40	B	G2	EX	4	2c	0	IV	R	PR	AD
11	65/F	IV	—	—	4×35	P	G1	EX	3	1	0	III	R	PR	AD
12	75/M	IV	—	7×40	7×40	T	G1	EX	3	2b	0	IV	R	PR	AD
13	67/F	III	—	—	12×45	L	G1	EX	1	0	0	I	R	CR	NED
14	67/M	IV	—	15×47	7×47	B	G1	EX	3	0	0	III	R+C	PR	AD
15	64/M	IV	—	—	20×49	B	G1	EX	3	2c	0	IV	R	CR	NED
16	65/F	IV	—	—	6×40	T	G1	IN	2	1	0	III	S+R	CR	NED
17	65/M	IV	—	10×30	5×30	G	G2	IN	4	1	0	IV	S+R	PD	DD
18	60/M	IV	—	10×40	15×45	T	G1	EX	2	0	0	II	S+R	CR	NED
19	62/M	II	—	10×4	2×42	B	G1	EX	2	0	0	II	R+C	SD	AD
20	85/M	IV	—	—	10×60	B	G1	EX	3	0	0	III	R+C	PR	AD
21	86/M	IV	—	15×65	10×30	B	G1	EX	2	0	0	II	R	PR	AD
22	85/M	III	—	5×40	5×40	B	G3	EX	2	0	0	II	R	CR	NED
23	85/F	IV	—	—	6×59	B	G1	EX	3	1	0	III	R	PR	AD
24	65/F	IV	—	—	3×50	T	G3	EX	4	2c	0	IV	R	PR	NED
25	65/M	III	—	30×40	—	T	G3	IN	3	2c	0	IV	R+C	PR	AD
26	61/M	IV	1×40	2×40	15×45	T	G1	EX	2	1	0	III	None	—	FF
27	65/M	III	—	—	15×40	L	G1	EX	2	0	0	II	S+R	CR	NED
28	62/M	IV	1×42	20×42	7×42	B	G2	EX	4	1	0	IV	S+R	CR	NED
29	65/M	IV	—	—	15×40	G	G1	EX	2	1	0	III	R	CR	NED
30	60/M	II	—	25×35	7×29	B	G1	EX	3	1	0	III	R	CR	NED
31	69/M	IV	—	10×50	—	T	G1	EX	1	0	0	I	R+C	CR	NED
32	63/M	IV	2×10	6×35	8×43	T	G1	EX	2	0	0	II	R+C	CR	NED
33	70/M	III	—	20×30	5×40	G	G1	EX	4	2c	0	IV	R+S	CR	NED
34	75/M	IV	—	10×20	6×25	G	G2	EX	3	0	0	III	None	—	FF
35	66/F	III	—	—	10×32	L	G1	EX	3	0	0	III	S	CR	NED
36	73/M	IV	—	15×53	4×53	G	G2	EX	3	0	0	III	R+C	PR	AD
37	75/M	IV	1×45	25×20	15×15	B	G1	EX	4	0	0	IV	R+S	CR	NED

For explanation of symbols see legend to Table 1.

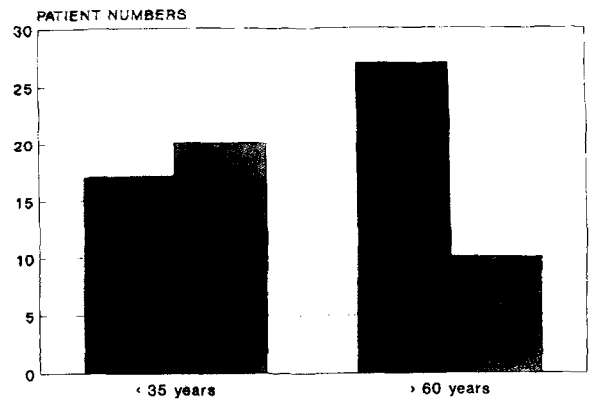

Fig. 1. The sex distribution in patients <35 years and >60 years with oral SCC. Males (■); females (▨).

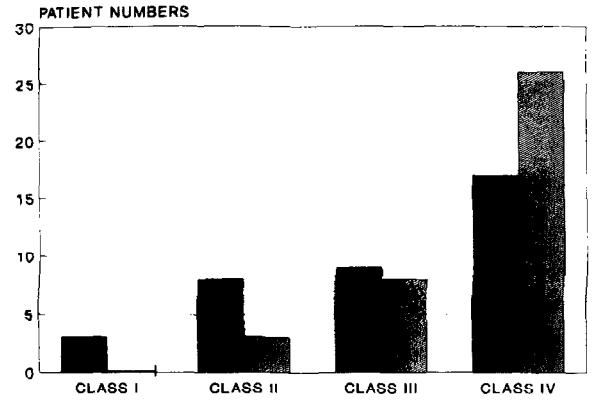

Fig. 2. The socio-economic class (I-IV) of patients <35 years (■) and >60 years (▨) with oral SCC.

Socio-economic class

Social class distribution showed a trend towards class IV in both the <35 and the >60 age groups. Although this feature was more obvious in the older population (Fig. 2), there were no statistically significant differences between young and older patients concerning social class.

Aetiological factors

The commonly identifiable aetiological factors were Pan chewing, smoking and alcohol. In the younger age group, only 10 of 37 patients had significant aetiological habits which included smoking (× 1), Pan chewing (× 2), smoking and pan chewing (× 3), Pan chewing and alcohol (× 2) and alcohol, Pan chewing and smoking (× 2) (Fig. 3). In the >60 age group, all except 1 patient had some significant aetiological factors; the habits included smoking (×3), Pan chewing (× 15), Pan chewing and smoking (× 13) and alcohol, Pan chewing and smoking (× 5). Thus, there were significantly more smokers ($P < 0.001$) and 'Pan' users ($P < 0.00001$), but not alcohol abusers, in the >60 age group compared with the <35 age group.

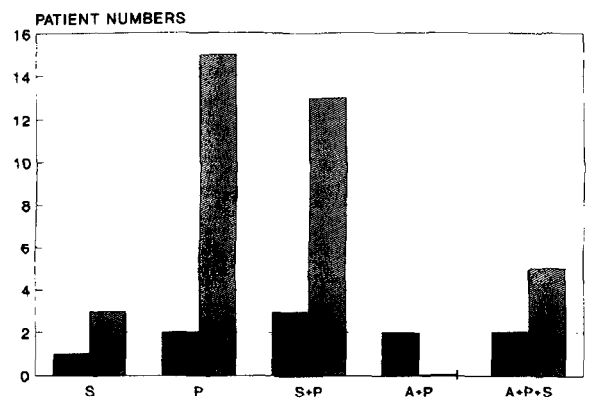

Fig. 3. Aetiological factors such as smoking (S), Pan chewing (P) and alcohol (A) in patients <35 years (■) and >60 years (▨) with oral SCC.

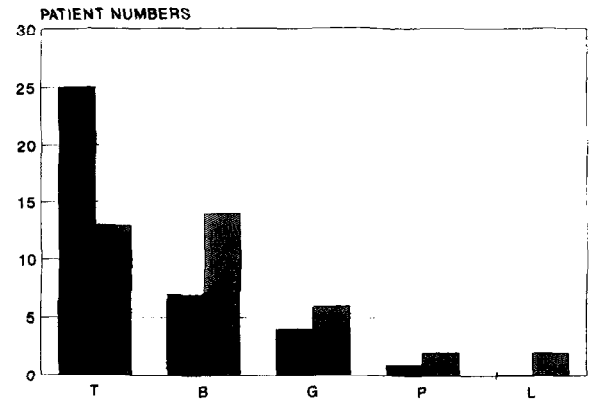

Fig. 4. The site distribution of oral SCC with respect to the tongue (T), buccal mucosa (B), gingiva (G), palate (P) and lip (L) in patients <35 years (■) and >60 years (▨).

Site

25 patients in the younger age group presented with lingual carcinoma but other sites included the buccal mucosa (×27), gingivae (× 4) and palate (× 1). By contrast, only 12 out of the 37 patients in the older age group presented with lingual carcinoma and the more common site was the buccal mucosa (× 14); other sites included the gingivae (× 6), palate (× 2) and lower lip (× 3) (Fig. 4). Thus, carcinomas in the >60 years age group occurred with similar frequency in the tongue and buccal mucosa but, in <35 years age group, tumours were more common in the tongue and these changes were statistically significant ($P < 0.04$).

Clinical stage

All patients were staged according to the UICC classification [21]. There were no significant differences in the distribution of the tumour stages between the two groups; the majority of the tumours in both groups were stage III (Fig. 5).

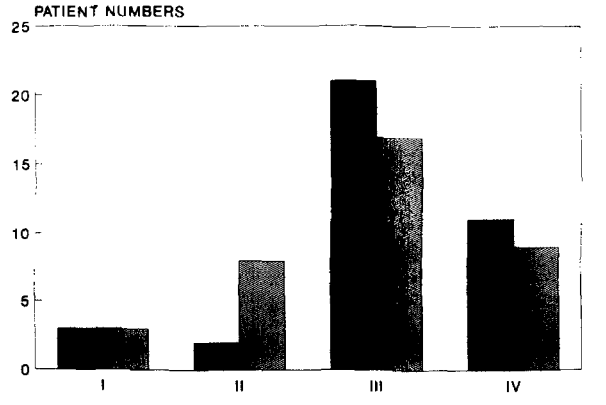

Fig. 5. The clinical stage of tumours (I-T1, NO, MO; II-T2, NO, MO; III-T3, NO, MO or T1–2, N1, MO; IV-T4, NO, MO or T1–3, N2/N3, MO or T1–3, NO–3, M1) in patients <35 years (■) and >60 years (▨) with oral SCC.

Despite there being an even distribution between the age groups concerning the staging of the tumours, tumours in the younger age group spread more commonly to the local nodes particularly as the tumour size increased. The difference in the incidence of nodal involvement in T3 tumours between the two age groups was statistically significant ($P<0.05$) (Fig. 6).

Pathological features

Ten tumours in the <35 age group were exophytic and 27 were infiltrative; in the >60 group, 31 were exophytic and only six were of the infiltrative type. Thus, infiltrative lesions were significantly ($P<0.0001$) more common in the <35 age group than the >60 age group (Fig. 7).

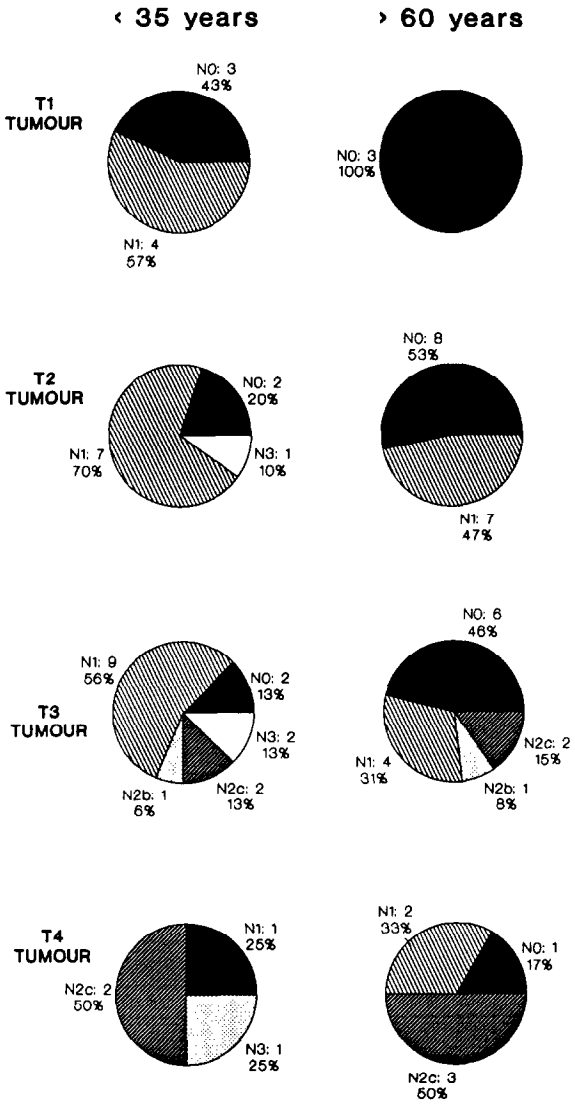

Fig. 6. Lymph node metastases in tumours <2 cms (T1), 2–4 cm (T2), >4 cm (T3) and fixed to adjacent structures (T4) with respect to patients <35 years and >60 years of age. Patients were classified as having no regional lymph node metastases (NO; ■), metastases in a single ipsilateral node of <3 cm diameter (N1, ▨), metastases in a single ipsilateral node of 3–6 cm diameter (N2a), metastases in multiple ipsilateral nodes and <6 cm diameter (N2b; ▩), metastases in bilateral or contra-lateral nodes and <6 cm diameter (N2c; ▨) and metastases in any node of >6 cm diameter (N3; □).

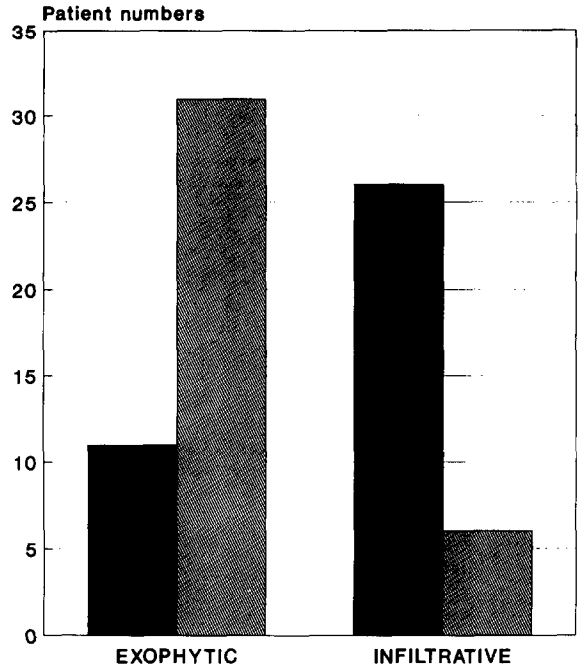

Fig. 7. The incidence of exophytic and infiltrative oral SCC in patients <35 years (■) and >60 years (▨).

Histopathological grading showed that the majority of patients in both groups had well-differentiated carcinomas. In the <35 age group, 22 were well-differentiated tumours, 10 were moderately differentiated and 4 had poorly differentiated carcinomas. In the >60 age group, 26 were classified as well-differentiated, 7 moderately differentiated and 3 poorly differentiated tumours. In 1 patient in this control group, tumour grading was not available. No statistically significant differences, therefore, were noted between the two age groups regarding tumour histology.

Treatment and treatment response

The treatment protocols used in both groups were the same. Of the 37 patients in the <35 age group, 11 received radiotherapy, 7 were treated by surgery, 7 by both surgery and radiotherapy, 10 by both radiotherapy and chemotherapy and 2 received all three modalities of treatment. In the >60 age group, 15 received radiation therapy, 3 patients underwent surgery alone, while 9 received surgery and irradiation and 8 radiotherapy and chemotherapy in combination. 2 patients in the >60 age group refused treatment.

The treatment responses were classified as complete response, partial response, progressive disease and stable disease (Fig. 8) and these assessments were made 6 weeks after completing the definitive treatment. 18 patients in the <35 age group showed complete response, whereas 11 showed partial response and 8 progressive disease. In the >60 age group, 22 showed complete response and 11 responded only partially to treatment. Only 1 patient in the >60 age group showed progressive disease and another had stable disease.

The patients were followed-up for at least 11 months (Fig. 9). In the <35 age group, 18 were still alive with no evidence of disease, 15 were alive with disease and 4 had died of cancer. In the >60 age group, 22 were alive with no evidence of disease, 12 were alive with disease and only 1 patient had died of cancer. 2 patients in this group, however, were not followed-up.

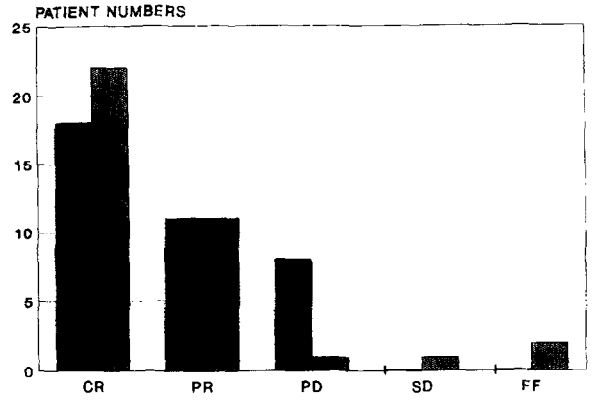

Fig. 8. The treatment response in patients <35 years (■) and >60 years (▨) with oral SCC. (CR, complete response; PR, partial response; PD, progressive disease; SD, stable disease; FF, failed follow-up.)

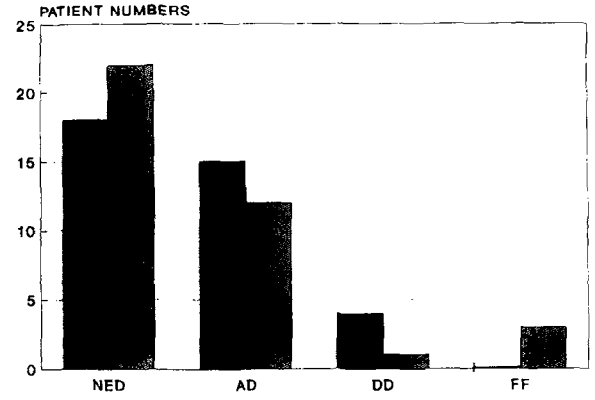

Fig. 9. The disease status after 11 months follow-up was classified as no evidence of disease (NED), alive with disease (AD), died of disease (DD) and failed follow-up (FF) (<35 years ■; >60 years ▨).

There were no statistically significant differences between the younger and older patients regarding their response to treatment.

DISCUSSION

The incidence of oral cancer shows considerable geographical variation. Whilst ranked the sixth commonest malignancy world-wide [22], in most western countries it constitutes 2–6% of all malignancies [23] and, in India, the rate is as high as 30% [1]. The incidence of oral cancer in a younger population, however, is more consistent and in the west varies between 0.4 and 5% of total oral cancer [11, 12, 19]; in the present series from India, the incidence was 1.3%.

Oral cancer is predominantly a disease of men in the sixth to eighth decades of life. Even though oral cancer is generally more common in males, it is interesting that in this series there was a slight predominance of female patients in the younger age group. Others have reported similar findings [16, 18, 24].

In the younger population examined in this study, oral carcinoma was apparent in all socio-economic classes but, in the patients over 60 years of age, it occurred more frequently in social classes III and IV. This may reflect the social class distribution of the population of India or may support the postulate that deficiency states, which are commonly seen in lower social classes, may be a predisposing factor of oral cancer particularly in the older age group. What is now important with regard to aetiological factors and socio-economic factors is to examine non-SCC patients; such studies are currently in progress.

Aetiologically oral cancer has been associated with tobacco smoking, Betel quid chewing, abuse of alcohol, chronic irritation from dental appliances and various deficiency states such as Plummer–Vinson syndrome [9, 10]. Whilst Betel quid chewing incorporating tobacco, tobacco smoking and alcohol abuse have been identified as major risk factors for oral cancer [25, 26] the results of the present study, and others [11, 12, 18, 19], indicated that no obvious aetiological factors characterised younger patients with oral cancer, especially those with cancer of the tongue. For example, some 88% (22/25) of patients aged <35 years with lingual carcinoma had no evidence of aetiological factors, whilst only 8.3% (1/12) of patients over 60 years with tongue tumours were characterised by the absence of causative agents. This lack of significant habits in young patients has prompted many to postulate factors such as immune deficiency and genetic factors in the aetiology of the cancers [27, 28]. Certainly there are rare genetic conditions which may predispose to oral cancer [13]. Whether other factors, such as viruses [29] or defects in Major Histocompatibility Complex Antigens [30, 31], are causal remains to be established.

Histopathological grading of tumours in this study showed no significant difference between the younger and older age groups; the majority of the tumours were well differentiated. Univariant analysis of survival and relapse-free periods by Holm and associates did not correlate with morphological grade [32] but others have suggested that cancer in younger adults tends to be more frequently anaplastic resulting in a more aggressive behaviour and poorer prognosis [12]. One of the significant findings in the present study was the difference in the morphological type of the tumour. 26 of 37 tumours in the <35 age group were infiltrative whereas, in the >60 age group, only 6 patients had infiltrative lesions. This may reflect, however, the higher incidence of lymph node metastasis and less favourable response to treatment in the <35 age group. Staging of the tumour showed an almost even distribution in both age groups. A critical analysis of the incidence of lymphatic involvement, however, showed that in the younger age group a higher proportion of the tumours metastasised early irrespective of the tumour size, a finding confirming previous reports (18).

Analysis of the response of the tumours to treatment revealed that in the <35 years group the cancers in 51.3% of patients resisted therapy. In patients >60 years, however, only 37.8% showed failure after treatment. Unfortunately, the patients in our series were followed-up for only 11–35 months, so a valid assessment of survival rate could not be made but, within this limited period, there was no significant difference in survival in the two age groups. It may be that in the older group the tumours, being exophytic, were readily controlled by surgery and radiotherapy. By contrast, tumours in the younger age group are infiltrative and the consequences of this disease process may have taken some time to manifest. A short follow-up period, therefore, may not have highlighted the differences between the age groups with regard to tumour behaviour. Review of the literature indicates conflicting reports of survival rates in younger age groups. Amsterdam and Strawitz reported a poorer survival outlook in T1,2 oral

cancer for the age group younger than 35 years [19]. Their 2-year survival was 57% for tongue and 75% for other oral cancers. On the other hand, Carniol and Fried reported comparable stage for stage survivals between the younger and older age groups [11].

In conclusion, the results of this study demonstrate marked differences in the manifestation of oral cancer in younger (<35 years) as opposed to older (>60 years) patients. The data indicates that tumours in younger patients behave more aggressively suggesting that more radical treatment modalities are necessary to control the disease.

1. National Cancer Registry. Annual report 1985. New Delhi: Indian Council of Medical Research.
2. Krolls SO, Hoffman S. Squamous cell carcinoma of the oral soft tissue: a statistical analysis of 14,353 cases by age, sex and race. *J Am Dent Assoc* 1976, **92**, 571–574.
3. Macfarlane GJ, Boyle P, Scully C. Rising mortality from cancer of the tongue in young Scottish males. *Lancet* 1987, **ii**, 912.
4. Binnie WH, Cawson RA, Hill GB, Soaper AE. Oral cancer in England and Wales. A national study of morbidity, mortality, curability and related factors. OPCS, *Studies on Medical & Population Subjects*, No 23 London: HMSO, 1972.
5. Szpak CA, Stone MJ, Frenkel EP. Some observations concerning the demographic and geographic incidence of carcinoma of the lip and buccal cavity. *Cancer* 1977, **40**, 343–348.
6. Tan KN. Oral cancer in Australia. *Aust Dent J* 1969, **14**, 50–56.
7. Depue RH. Rising mortality from cancer of the tongue in young white males. *N Engl J Med* 1986, **315**, 647.
8. Hakulinen T, Andersen AA, Malker B, Pukkala E, Schou G, Tulinius H. Trends in cancer incidence in the Nordic countries. *Acta Pathol Scand Suppl* 1986, **288**, 1–186.
9. Boyle P, Macfarlane GJ, Maisonneuve P, Zheng T, Scully C, Tedesco B. Epidemiology of mouth cancer in 1989: a review. *J Roy Soc Med* 1990, **83**, 724–730.
10. Boyle P, Zheng T, Macfarlane GJ, McGinn R, Maisonneuve P, La Vechia C, Scully C. Recent advances in the etiology and epidemiology of head and neck cancer. *Curr Opin Oncol* 1990, **2**, 539–545.
11. Carniol PJ, Fried MP. Head and neck carcinoma in patients under 40 years of age. *Ann Otol Rhinol Laryngol* 1982, **91**, 152–155.
12. Byers RM. Squamous cell carcinoma of the oral tongue in patients less than 30 years of age. *Am J Surg* 1975, **130**, 475–478.
13. Kraemer KH, Lee MM, Scotto J. Early onset of skin and oral cavity neoplasms in xerodema pigmentosum. *Lancet* 1982, **i**, 56–57.
14. Clark RM, Rosen IB, Laperriere NJ. Malignant tumors of the head and neck in a young population. *Am J Surg* 1982, **144**, 459–462.
15. Newman AN, Rice DH, Ossoff RH, Sisson GA. Carcinoma of the tongue in persons younger than 30 years of age. *Arch Otolaryngol* 1983, **109**, 302–304.
16. McGregor GI, Davis N, Robins RE. Squamous cell carcinoma of the tongue and lower oral cavity in patients under 40 years of age. *Am J Surg* 1983, **146**, 88–92.
17. Sankaranarayanan R, Mohideen MN, Nair MK, Padmanabhan TK. Aetiology of oral cancer in patients less than or equal to 30 years of age. *Br J Cancer* 1989, **59**, 439–440.
18. Venables CW, Craft IL. Carcinoma of the tongue in early adult life. *Br J Cancer* 1967, **21**, 645–650.
19. Amsterdam JT, Strawitz JG. Squamous cell carcinoma of the oral cavity in young adults. *J Surg Oncol* 1982, **19**, 65–68.
20. Holm LE, Lundquist PG, Silfversward C, Sobin A. Histological grading in squamous cell carcinoma of the oral tongue. *Acta Otolaryngol* 1982, **94**, 185–192.
21. TNM classification of malignant tumours. Geneva, International Union Against Cancer, 1988.
22. WHO, World Health Statistics Annual. Geneva: WHO, 1987.
23. Parkin DM, Laara E, Muir CS. Estimates of the world-wide frequency, of sixteen major cancers in 1980. *Int J Cancer* 1988, **41**, 184–197.
24. Son YH, Kapp DS. Oral cancer and oropharyngeal cancer in a younger population: a review of the literature and experience at Yale. *Cancer* 1985, **55**, 441–444.
25. Winn DM, Ziegler RG, Pickle LW, Gridley G, Blot WJ, Hoover RN. Diet in the aetiology of oral and pharyngeal cancer among women from the Southern United States. *Cancer Res* 1984, **44**, 1216–1222.
26. Jusawalla DJ, Deshpande VA. Evaluation of cancer risk in tobacco chewers and smokers: an epidemiological assessment. *Cancer* 1971, **28**, 244–252.
27. Wanebo HJ, Jun MY, Strong EW, Oettgen H. T Cell deficiency in patients with squamous cell carcinoma of the head and neck. *Am J Surg* 1975, **130**, 445–451.
28. Gangal SS, Tatake RJ. Immune status in head and neck cancer with special reference to cancer of the oral cavity. In Johnson NW ed. *Risk Markers for Oral Disease* Vol. 2. *Oral Cancer, Detection of Patients and Lesions at Risk*. Cambridge, Cambridge University Press, 1991, 114–154.
29. Scully C, Prime SS, Cox MF, Maitland N. Evidence for infective agents in the aetiology of oral cancer. In Johnson NW ed. *Risk Markers for Oral Disease*. Vol. 2. *Oral Cancer, Detection of Patients and Lesions at Risk*. Cambridge, Cambridge University Press 1991, 96–113.
30. Prime SS, Pitigala-Arachchi A, Crane IJ, Rosser TJ, Scully C. The expression of cell surface HLA heavy and light chain molecules in premalignant and malignant lesions of the oral mucosa. *Histopathology* 1987, **11**, 81–91.
31. Mutlu S, Matthews JB, Midda M, Scully C, Prime SS. MHC antigen expression in human oral squamous carcinoma cell lines. *J Pathol* 1991, **165**, 129–136.
32. Holm LE, Lundquist PG, Silfversward C, Sobin A. Histopathological grading of malignancy in squamous cell carcinoma of the oral tongue. *Acta Otolaryngol* 1982, **92**, 185–192.
33. Leete R, Fox J. Registrar General's Social Classes; Origins and Uses. *Population Trends* 1977, **8**, 1–7.

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