Idiopathic Hypertrophy of the Masseter Muscle

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Introduction

REGG (1880) was perhaps the first to report a case of recurrent swelling of the masseter muscle with cervical adenitis and a rather vague clinical picture associated with nausea and vomiting. Duroux (1905) described bilateral painless swellings in the parotid region with unknown aetiology. Boldt (1930) reported many cases of masseter swellings but only two of his cases had muscle hypertrophy. Thereafter a number of cases have been reported by various authors (Coffey, 1942; Gurney, 1947; Adams, 1949; Maxwell, 1951; Guggenheim, 1959, 1960 and 1961 and Joannes, 1971) and the condition is now recognised as a distinct surgical entity. To the best of the knowledge of the authors, this is the first Indian series on the subject.

Incidence

The total number of cases reported so far does not seem to exceed eighty. Barton (1952) came across thirty cases in his experience of ten years on tumor-boards. Guggenheim and Cohen (1959) collected twenty eight cases in a psychiatric clinic. Inspite of our vigilance both in the out-patients' department and in the wards, we have been able to collect only eight cases in as many years in our hospital which caters to an area of about five hundred square miles. Six of our cases were males and two females (Table I), showing a male preponderance. This is in conformity with the sex incidence in Mackinnon's series (1967). The disease was encountered most frequently in the fourth decade, only one case being forty-five years old (Table II). In none of our cases there was a familial history. In four the swelling was bilateral and in four unilateral (Table III).

Table I
Sex Incidence

Sex	Male	Female	Total No. of cases.
No. of cases	4	4	8

Table II
Age Incidence

Age in years	30-40	40-50
No. of cases	7	1

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Table III

Involvement	Unilateral		Bilateral
	Rt.	Lt.	
No. of cases.	2	2	4

Clinical Picture

Five out of the eight cases consulted us for fear the swelling might grow larger in size or progress to 'cancer'; two came for cosmetic considerations and one of the cases was discovered while he actually came for an altogether unconnected condition, chest injury. The duration of the swelling was one month in one case, in whom it followed an attack of cervical adenitis but persisted after the adenitis subsided. In two cases the duration of the swelling was two years while in the remaining five there was a long history of many years' duration. None of them suffered from pain or fatigue in the act of chewing. There was a positive history of chewing betal leaf/nut in six cases. There was no clear history of emotional disturbance in any of these cases. None of them complained of intermittent claudication. On examination the only positive feature of the condition was the presence of a firm muscular swelling situated over the angle and the adjoining part of the rami of the mandible, extending upwards to the zygoma. The swelling was present on one (Fig. 1) or both sides (Fig. 2). The swelling became tense and hard on clenching the jaws, the tenseness being both visible and palpable. None of them had dental pathology like excessive teeth-erosion or malocclusion and in none of them we could find an associated hypertrophy of the temporalis muscle.

Diagnosis

The swelling must be differenciated from parotid swellings which can be easily

Fig. 1

Fig. 2

done because of its contractility and also by the fact that the parotid swellings extend below and behind the ear lobule. Swellings arising from the bone whether solid or cystic are immobile and hard in consistency. Other tumors of the masseter like fibroma, myoma, haemangioma or sarcoma : inflammatory conditions like hydatids, guineaworms and trichinosis ; post-traumatic conditions like organised haematomas and myositis ossificans must be excluded. Muscular dystrophies are associated with weak bites and fatigue. Among other ancillary methods, alkaline phosphatase activity and electromyographic studies can be done but were not done in our cases. Ahlgren et. al. (1969) were able to demonstrate increased muscle power through electromyography. Muscle biopsy and routine staining did not reveal any abnormality and the muscle was indistinguishable from normal striated muscle (Fig. 3 and 4).

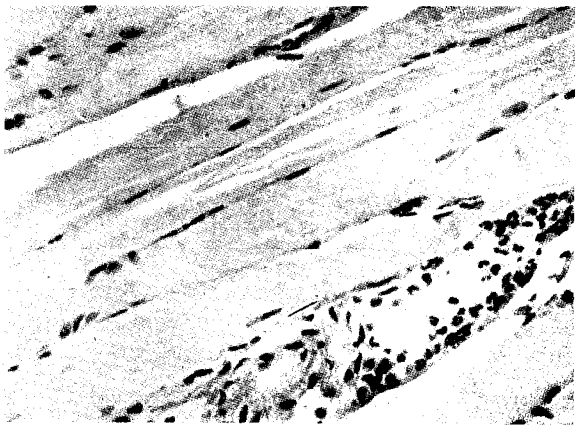

Fig. 3

Skiagrams

Consistently showed two types of changes :

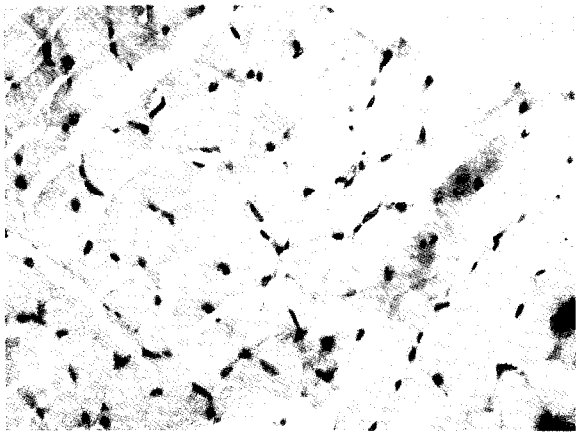

Fig. 4

1. An abnormally large bony overgrowth at the mandibular angle (Fig. 5).

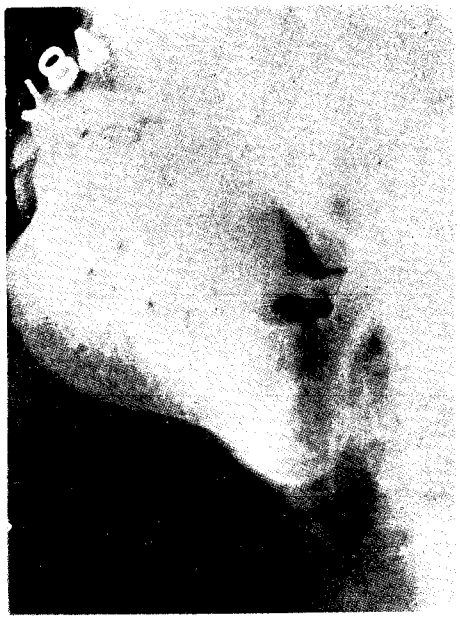

Fig. 5

2. A smaller mandibular angle (Fig. 6, 8). The normal angle is about 116°-126° (Fig. 7 and 10) whereas the average angulation in these cases is 90° (Fig. 9).

while the bony overgrowth appears to be

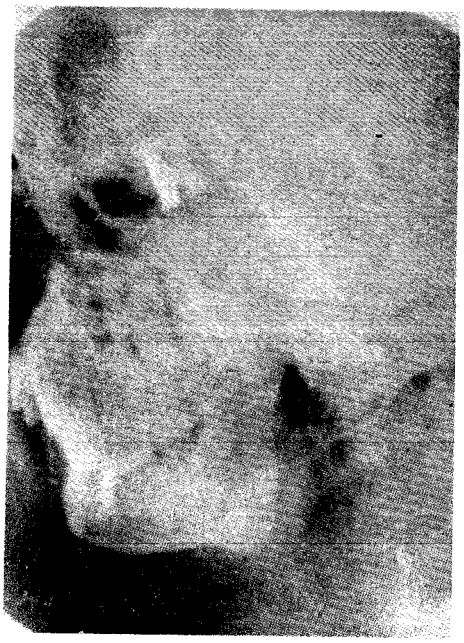

Fig. 6

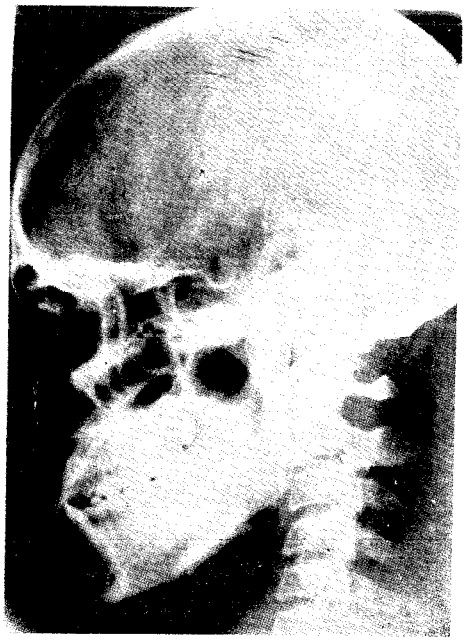

Fig. 7

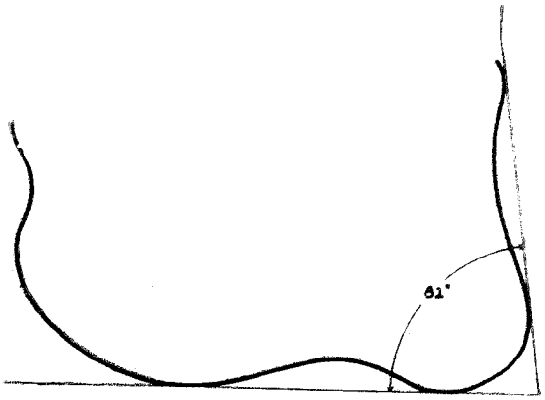

Fig. 8

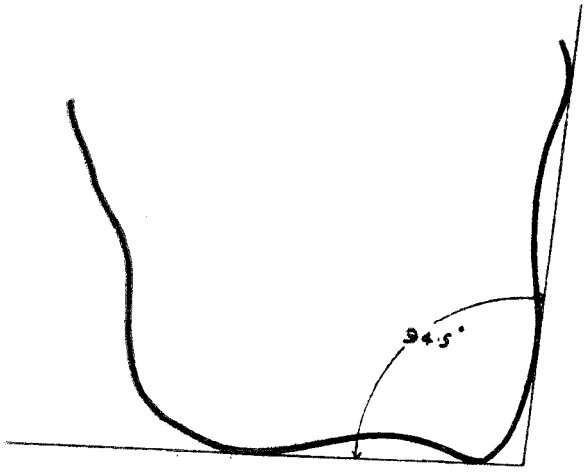

Fig. 9

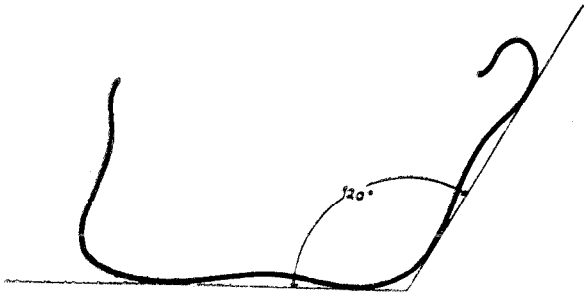

Fig. 10

the result of repeated microscopic trauma to the periosteum at the site of attachment of the muscle to the mandible followed by subperiosteal bone deposition, the angulation deformity is probably the result of the upward pull of the masseter muscle. Gelbke (1958), Ahlgren et. al. (1969) and Joannes (1971) are the only other authors who have mentioned the angulation deformity, found regularly by us. Many authors (Coffey, 1941 ; Tempest, 1951 ; Gurney, 1947 ; Guggenheim and Cohen, 1960) did not find any bony changes.

Morbid Anatomy

Macroscopically the hypertrophic muscle looked like normal healthy muscle. Histological examination revealed muscle cells of normal length and striations. There was no indication of muscle atrophy or loss of striation like those described by Goodwin et. al. (1967), nor was there any evidence of oedema or increased density of the collagen fibres.

Treatment

Our policy has been to operate on only those cases who were sufficiently bothered by the deformity to undergo surgery. 5 out of the eight cases were not operated because they did not opt for operation after being reassured that the swelling was not a cancer nor was likely to become one and that it would lead to no serious complication. One patient was dicovered during the course of examination for a chest injury. Two patients were keen for operation for cosmetic reasons and were operated on with gratifying results. Both intra-oral and submandibular approaches have their protagonists. Gurney (1947), who was the first to operate on the deformity used a four inch long incision under the mandible and removed a part of the muscle from its outer side. Adams (1949) excised a portion from its inner side with the argument that the buccal and mandibular branches of the facial nerve were susceptible to injury from the outerside. Dencer (1961) made a transverse incision over the muscle and the muscle fibres retracted irregularly to produce an uneven surface. Converse (1964) and Hankey (1968) also advocated the intra-oral approach and removed the hyperostotic bone. Ginestat (1959) finds the extra-oral approach unacceptable on cosmetic grounds. In those cases, in which we decided to operate, we used the extra-oral approach for the following reasons :

1. The approach is direct and easy.
2. The scar, situated below and behind the rim of the mandible is altogether inoccuous.
3. With care it is always possible to avoid injury to the branches of the facial nerve.
4. We do not advocate removing the medial portion of the muscle as the nerve supply as well as the vascular supply of the muscle come to it from its medial side and to that extent the operation is unanatomical. Instead we remove a slice of the muscle from its outside leaving a completely acceptable post-operative result (Figs. 11,12,13).
5. Removal of the bony mass where necessary is no more difficult from the

external approach.

Discussion

The condition has evoked considerable interest with regards to its causation. Guggenheim and Cohen (1960) after a histopathological and aetiological study suggested an arterio-venous origin. Clenching of the teeth due to emotional disturbance (Hersh, 1946) and work-hypertrophy due to constant chewing or grinding of teeth (Gurney, 1947) were believed to produce masseter hypertrophy but it is not accepted as the cause by Adams (1949), Master (1955), Gelbe (1958) and Dencer (1961). Temporomandibular joint disturbance, (Tempest, 1951), missing teeth (Barton, 1957) and bite deformity, bruxism and malocclusion are the other suggested causes. In one of our cases, the condition developed after an attack of cervical adenitis and did not subside after the glands settle down. This is of interest in as much as another author (Legg 1880) has reported a similar association in his case. In six out of the remaining eight cases, there was a history of betel-leaf/nut chewing, which to our thinking could contribute to the hypertrophy.

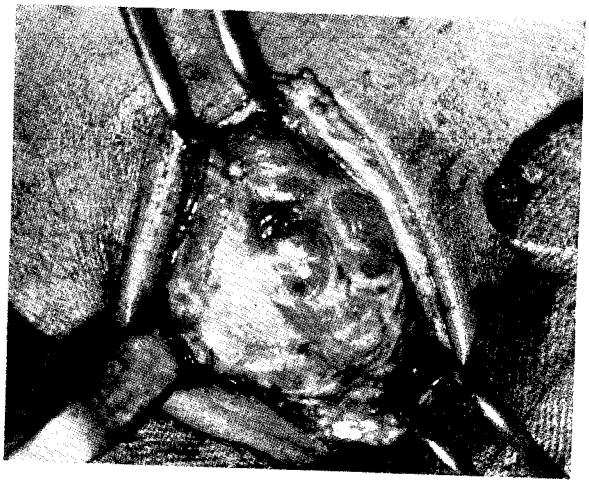

Fig. 11

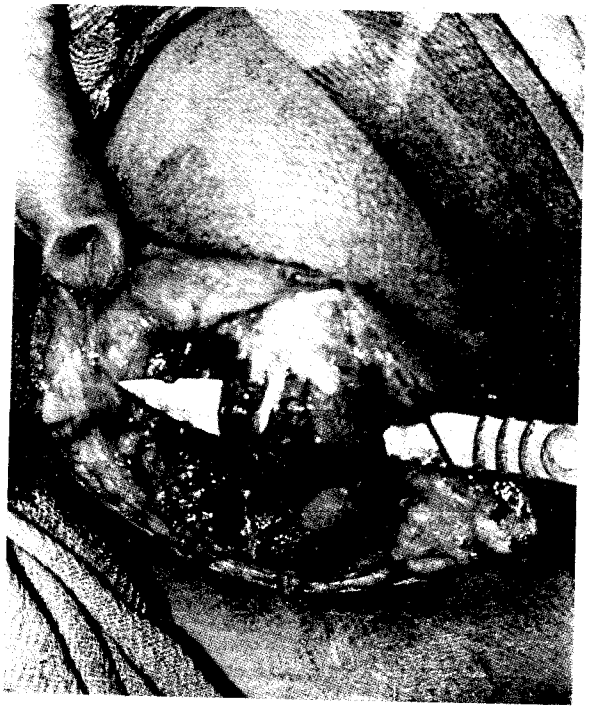

Fig. 12

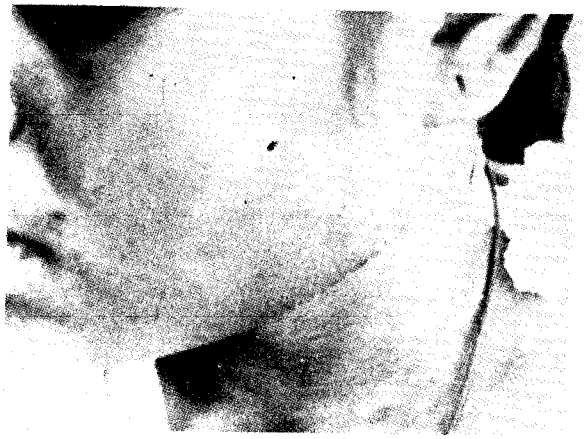

Fig. 13

Summary

A short summary of the literature on

masseter hypertrophy has been given along with the findings from our own series of eight cases. The salient features of our findings are that the condition is perhaps related to some type of work hypertrophy, in our cases betel-nut chewing ; that only for marked cosmetic deformity is surgical interference indicated ; we prefer the extra-oral approach with excision of the external portion of the muscle, which may be combined with excision of the hyperostotic bone where necessary ; ordinary histological examination does not reveal any changes in the muscle histology but special technique like silver staining could help. Radiology of the mandible shows definite changes in the form of abnormal bone growth at the mandibular angle and reduction in the angulation of the angle from an average of 120° to an average 90°.

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