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Combined Gingival Enlargement on the Treatment of Glioma and Seizures - A Rare Case Report

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Abstract:- The risk of developing epilepsy in patients with gliomas or neuroepithelial tumours is extremely significant. Low-grade and high-grade gliomas are the two types of gliomas. Low-grade brain tumours, as well as tumours located in the temporal and insular cortex, have a higher proclivity for developing epilepsy. Seizure control is much improved by surgical treatment of the tumour, particularly gross tumour removal, especially in neuroglial tumours. Low-grade gliomas are more likely to cause refractory epilepsy, with 30–35 percent experiencing it. The therapy of combined drug-induced gingival expansion in a patient surgically treated for low-grade glioma is described in this case report.

I. INTRODUCTION

Glioma is a brain/spine tumour that emerges from glial cells, accounting for 30% of all brain and CNS malignancies and 80% of malignant brain tumours. The development of seizures is most usually linked to neuroepithelial malignancies. Low-grade brain tumours have a larger proclivity for epileptogenesis than more malignant brain tumours (Table 1)[1]. These developing tumours include structural abnormalities linked with cortical dysplasia, implying that structural lesions in the brain parenchyma have been present for a long time, favouring conditions that contribute to the development of seizures..

Seizures are common in low-grade glioma, ranging from 65 percent to 85 percent of the time, and they are the first clinical symptom in 70-90 percent of cases. At the time of presentation, the typical age is 38-40 years old (Table 1)[1].

During follow-up, 6-11 percent of the initial patients who have never had a seizure will eventually acquire epilepsy[2]. It could be focal seizures without altering consciousness (23.7 percent of the time), focal seizures with altering consciousness (6.6 percent of the time), or secondary generalised seizures (6.6 percent of the time) (69.7 percent). Seizures are less common in low-grade gliomas involving midline structures. Studies have shown that excision of the epileptogenic zone due to a brain tumour can result in significant seizure control in 70-90 percent of these difficult patients when combined with medication therapies. [3,4].

Medication interactions are serious issues that can cause problems with compliance, tolerance, and side effects.

Merrit and Putnam were the first to propose phenytoin sodium as an antiepileptic medication in 1938[5]. Phenytoin-induced gingival overgrowth was first reported in the literature a year after it was first used in clinical trials[6]. Other anticonvulsant drugs, such as Valproic acid, Carbamazepine, and Phenobarbitone, have since been demonstrated to cause clinically significant gingival overgrowth. The mechanism that causes drug-induced gingival overgrowth isn't totally understood. In vitro, however, several investigations have found evidence of reduced collagen destruction, extracellular matrix synthesis by gingival fibroblasts, and cell proliferation[7,8].

	Neurogliomas	Gangliogliomas	Low gradegliomas	Glioblastoma
Age at Presentation	15 years	17-21 years	38-40 years	60 years
Seizures (%)				
At Presentation	100	60-95	65-85	68
Incidence		80-90	70-90	30-60
Type of epilepsy (%)				
Focal	51	86		
Without LOA	na	12	24	20-27
With LOA	52	74	7	3-10
Secondarily generalized/mixed (%)	49	43	70	35
Location of tumor (%)				
Temporal	79	35-46	37	24-27
Extra temporal	21	24	63	73-76
frontal	14	7-13	71	28-43
Parietal	3	6-10	9	19-27
Insular	na	na	21	na
occipital	3	2.0-3	0	5-9

Table 1 : Seizure characteristics of neuroepithelial tumours

II. CASE REPORT

A 36year old male patient was referred to the Department of Periodontics and Oral Implantology, A.J Institute of Dental Sciences with a chief complaint of gingival enlargement and difficulty in speech, mouth opening and swallowing. (Figure 1)

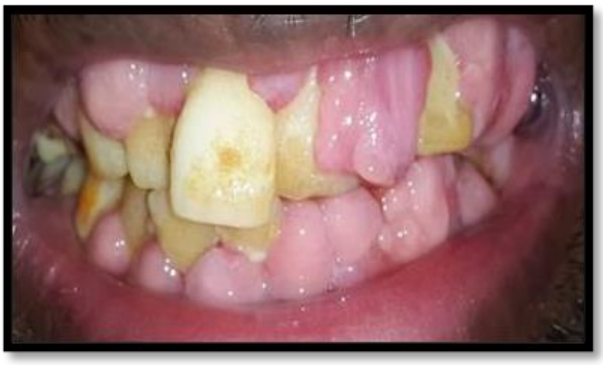

Fig 1: Pre-Operative View of The Patient

His medical history revealed a frontal glioma (low grade glioma) for which he was operated at NIMHANS, Bangalore two years back. Patient was on prophylactic antiepileptic drug, Phenytoin Sodium (100mg BID) since the time of surgery. He eventually developed generalised gingival overgrowth 6months back which was extensive, covering most of the occlusal surface of the teeth. Patient had difficulty in chewing and complained of pain and due to constant friction, there was bleeding from gingival tissues. Patient had an altered consciousness, thus a deficiency in plaque control measures and generalised inflammation was evident resulting in combination of DIGO and complicating the inflammation caused by bacteria.

Neurologist consent was obtained and patient was posted for surgical excision of gingival tissue. Oral prophylaxis was performed on the first visit and 2% chlorhexidine mouthwash was prescribed for 15 days. (Figure2)

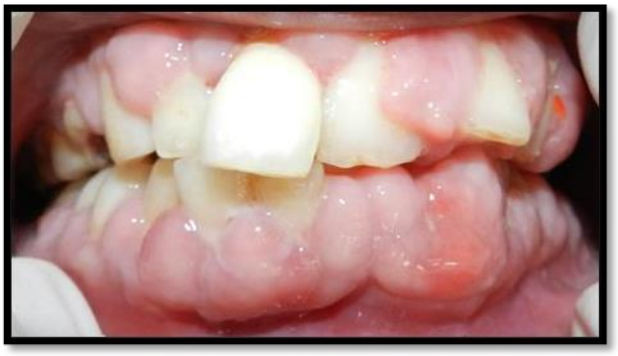

Fig 2: One Week after Scaling

Patient was briefed about the surgical procedure and written informed consent was obtained. Surgical gingivectomy procedure was carried out at 4 sitting under local anaesthesia. During the excision tissue appeared overtly inflamed which contributed to the local factors. Post operative instruction were given and anti-inflammatory medications was prescribed. (Figure 3)

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Fig 3: Gingivectomy procedure performed for theexcision of the gingival overgrowth using No: 15 blade.

Healing was uneventful. Successful follow up was made for 2 visits after completion of the treatment. (Figure 4)

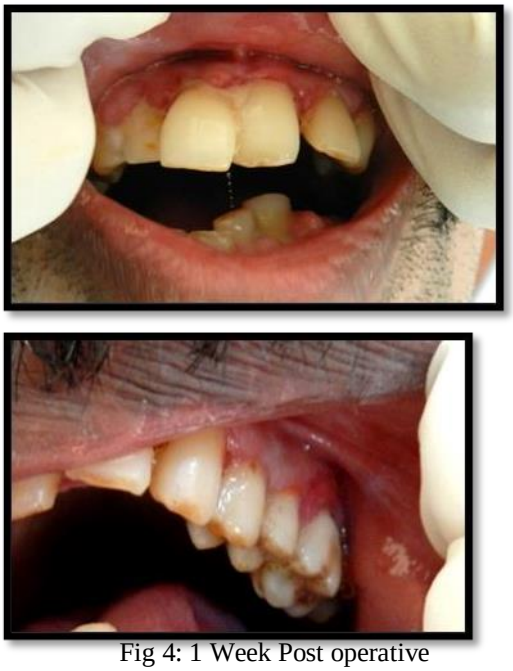

Fig 4: 1 Week Post operative

III. DISCUSSION

One of the most commonly prescribed seizure medications is phenytoin sodium. Gingival enlargement caused by phenytoin has been known to occur for numerous years and affects 50% of those who take the drug[9]. Its prevalence and severity are not always linked to the dosage given once a certain threshold level has been exceeded[10]. In vitro, fibroblasts from phenytoin-induced gingival overgrowth produce more sulphated glycosaminoglycans[11] and less collagen breakdown due to the generation of inactive fibroblastic collagenases[12].

A genetic tendency has been hypothesised as a factor in determining whether or not a person taking phenytoin will experience gingival hypertrophy. DIGO recurrence is inevitable in all surgically treated cases[13]. Plaque control measures (soft toothbrushes), Chlorhexidine mouth rinses, and professional oral prophylaxis on a regular basis will help. However, since the medication in the present case is given for control of seizures post surgically, recurrence rate is considerably significant.

IV. CONCLUSION

Because the topic of preventative treatment for patients with brain tumours is so complicated, every instance of gingival growth should be treated with great care. Following consultation with the treating physician and surgical therapy, supporting periodontal care should be administered at regular intervals.

REFERENCES

[1]. Melissa Kerkhof, and Charles J. Vecht. Seizure characteristics and prognostic factors of gliomas.Epilepsia2013;54(Suppl.9):12–17.

[2]. Van Breemen MS, Rijsman RM, Taphoorn MJ, Walchenbach R, Zwinkels H, Vecht CJ. Efficacy of anti-epileptic drugs in patients with gliomas and seizures. J Neurol 2009;256:1519–1526.

[3]. Britton, J. W., G. D. Cascino, et al. Low-grade glial neoplasms and intractable partial epilepsy:efficacy of surgical treatment. Epilepsia 1994; 35(6): 1130-1135.

[4]. Zentner, J., A. Hufnagel, et al. Surgical treatment of neoplasms associated with medically intractable epilepsy. Neurosurgery 1997;41(2):378-386:discussion 386-377.

[5]. Merritt H, Putnam T. Sodium diphenyl hydantoinate in the treatment of convulsive disorders.J Am Med Assoc 1938;111:1068-1073.

[6]. Glickman I, Lewitus M : Hyperplasia of the gingiva associated with Dilantin sodium Diphenyl hydantonate therapy. J Am Med Assoc 1941;28:1991.

[7]. T.G Modeer, M. Brunis, M. Linuma and U.H Lerner. Phenytoin potentiates interleukin -1 induced prostaglandin biosynthesis in human gingival fibroblasts. The British Journal Of Pharmacology 1992;106.(3)574-578.

[8]. T. Modeer, I. Anduren, A. Bengston and G. andersson, Interleukin -1 beta and phenytoin reduce alpha 1 procollagen mRNA expression in human gingival fibroblasts. Journal of Periodontal Research 1996;31(8)563-569.

[9]. Jones JE, Weddell JA, McKnown CG. Incidence and indications for surgical management of phenytoin-induced ginigival overgrowth in a cerebral palsy population. J Oral Maxillofac Surg 1988;46: 385–390.

[10]. Katz J, Givol N, Chaushu G, Taicher S, Shemer J. Vigabatrin-induced gingival overgrowth. J Clin Periodontol 1997;24:180-182.

[11]. Kantor ML, Hassell TM. Increased accumulation of sulfated glycosaminoglycans in cultures of human fibroblasts from phenytoin-induced gingival overgrowth. J Dent Res 1983;62:383-387.

[12]. Morton RS, Dongari-Bagtzoglou AI. Regulation of gingival fibroblast interleukin-6 secretion by cyclosporine A. J Periodontol 1999;70:1464-1471.

[13]. Rees TD, Levine RA. Systematic drugs as a riskfactor for periodontal disease initiation and progression. Compendium 1995;16:20,22,26.