

Corseting' and strangling'—two techniques sharing similar concepts to treat large venous malformations in the head and neck region

We congratulate Colletti et al. for introducing the 'strangling technique' in a preliminary report of two cases in the journal *Head & Neck* in October 2014¹. Although it shares a similar approach with the 'corset technique' in terms of the concept and the exposure of the lesion, it is very different from the corset technique². The term itself talks about a difference in collapsing the lesion using locked vertical mattress sutures. The following narrative clearly explains the differences between the two techniques.

In October 2014, Colletti et al. presented case reports showing the use of the strangling technique in two patients. We followed up a series of 90 patients who underwent corset suturing from 1999 to 2017. These patients were categorized into different groups as reported in the article by Nair et al. published in 2011³.

Colletti et al. describe the use of non-resorbable sutures involving the most superficial part of the venous malformation pexing into the underlying periosteum. The corset technique does not involve pexing the periosteum but only the bulk of the lesion. The suture used is PDS (polydioxanone). The advantage of PDS is that its tensile strength lasts for over 2 weeks and it undergoes resorption within 180 days. This allows sufficient time for internal thrombosis and fibrosis.

Colletti et al. use a technique of exposing the facial nerve from its exit through the stylomastoid foramen and its branches using an anterograde approach, which is not advocated in the corset technique. Dissecting around the nerve itself could cause a transient nerve palsy.

Many of our patients underwent surgical de-bulking once the reduction in size of these large venous malformations had been achieved using the corset technique.

In our recent paper on corseting, we presented the cases of 90 patients, 63 of whom were followed up over a period of 18 years; 25 had a minimum follow-up of 5 years and only two patients were lost to follow-up.

Below are our responses to the pointers for discussion introduced by Colletti et al., in sequence.

- Most patients in our study underwent a CT/MRI/ultrasound scan preoperatively. These were not presented in the paper, as the lesions were clinically

impressive enough to justify the use of the corset technique.

- It is agreed that MRI is the gold standard to diagnose and treat soft tissue venous malformations. However, our earliest cases were seen in 1999, a time when the availability of MRI in our country was limited. Furthermore, the added costs involved in MRI were prohibitive for many of our patients who have a low economic background. The above explains the use of a preoperative CT scan over MRI in this group of patients.
- We are in agreement that the term haemangioma should have been omitted to avoid confusion. Mention of haemangioma was thought to be relevant in select cases of low-flow venous malformations underlying residual haemangiomas, which were excised as a part of skin de-bulking during corseting.
- Venous malformations being complex in nature may present with a small arterial component although being mostly venous. There was no attempt made to isolate the arterial component and the lesions were corseted as a whole.
- The corset technique advocates suturing in a vertical looping fashion with polydioxanone sutures to incorporate the bulk of the lesion. The facial nerve was obviously avoided when visible, to minimize the chances of facial palsy. The vertical looping caused collapse of the transverse vascular channels, hence providing maximum benefit from this technique. Seven patients reported transient nerve palsy, which was reversed due to the resorbable nature of the sutures used.
- Many of our cases underwent surgical excision of the residual collapsed lesion to achieve a near total clearance and further minimize the chance of recurrence. The use of non-resorbable sutures poses the risk of leaving behind a source of infection and suture track formation. As mentioned previously, we advocate the use of polydioxanone sutures, which allow about 6 weeks for the lesion to collapse and enable further de-bulking as necessary.

Based on the above information, it is clear that although the two techniques share a similar concept, they differ in many ways. Furthermore, we have presented data based on 18 years of experience. Readers should not be confused between the two techniques, both of which could be used to treat these large complex venous malformations in the head and neck.

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Competing interests

None.

Ethical approval

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Patient consent

Not required.

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Regarding “Impact of crack cocaine use on the occurrence of oral lesions and micronuclei”

We read with interest the recent article by Antoniazzi and colleagues published in the *International Journal of Oral & Maxillofacial Surgery* entitled “Impact of crack cocaine use on the occurrence of oral lesions and micronuclei”¹. In their study, the authors detected an increased number of fundamental lesions and micro-

nuclei in the oral mucosa of crack cocaine users. However, there are certain questions related to data interpretation and accuracy that must be addressed with regard to this study.

First, it is important to stress that Giemsa is an inappropriate technique for detecting micronucleated cells, since it is not specific for staining nucleic acids. The Feulgen-fast green method has been considered the gold standard for this purpose and is highly recommended by the International Human Micronucleus Project on Exfoliated Buccal Cells². Giemsa stain can cause false-positive results due to the presence of keratohyaline granules and/or bacteria, thus the identification of micronuclei will be overestimated³. This may explain the high numbers of micronuclei presented in Table 3 (mean of 17.2 micronuclei in the experimental group and 3.8 micronuclei in the control group). Micronucleus is a rare event in the oral mucosa, whose formation arises in the dividing cells in the basal layer of the oral epithelium, with later detection in exfoliated keratinocytes following differentiation⁴.

Regarding cytotoxicity, the authors showed increased karyorrhexis and karyolysis in buccal mucosa cells from crack cocaine users. Karyolysis is closely associated with necrosis, whereas karyorrhexis is linked to apoptosis⁴. Independent of the biological phenomenon of cellular death, cytotoxicity participates in the non-genotoxic mechanisms of carcinogenesis, since it interferes with cell proliferation in the eukaryotic cell (promotion stage). Taken as a whole, the results of this study clearly demonstrate that mutagenicity and cytotoxicity are induced by crack cocaine in oral mucosa cells. These findings could explain the higher incidence of oral lesions in crack cocaine users, especially in some regions considered at high risk of oral cancer, such as the floor of the mouth and tongue. Following this rationale, it would be important to compare the frequencies of micronucleus, as well as other nuclear alterations indicative of cytotoxicity, among volunteers with and without oral lesions visible at clinical examination as experimental and control groups, in order to correlate the metanuclear alterations and the frequency of oral lesions. Such information would contribute towards the validation of the micronucleus assay as a putative biomarker for oral diseases in this vulnerable population and others as well.

Finally, the authors report that “Control individuals with history of street drug use were recruited from public schools or from a list for treatment at the dental school who required an examination before undergo-

ing treatment’’. It is well established that illicit drugs are able to induce genotoxic damage⁵. A control group not exposed to known genotoxins would also be interesting in this setting to confirm the cytogenetic damage mediated exclusively by crack cocaine in buccal mucosa cells⁵.

We hope that these comments are useful for better understanding the interesting article investigating the relationship between crack cocaine, cytogenetic damage, and oral lesions.

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Response to the Letter to the Editor regarding “Impact of crack cocaine use on the occurrence of oral lesions and micronuclei”

We are grateful for the interest in our study “Impact of crack cocaine use on the occurrence of oral lesions and micronuclei”¹. Firstly, regarding the criticism that Giemsa stain can cause false-positive results, we agree that this method may provide an overestimate of the presence of micronuclei. However, any misclassification would be non-differential, as a difference in the overestimation between groups is not plausible. Thus, this type of error would bias the results towards the null hypothesis² and therefore does not account for the differences described in our study between crack users and non-users. Moreover, despite its limitations, Giemsa solution has been used to evaluate the presence of micronuclei in different fields of dentistry, with articles published in high-impact journals^{3,4}.

Another criticism was that “it would be important to compare the frequencies of micronucleus, as well as other nuclear alterations indicative of cytotoxicity, among volunteers with and without oral lesions visible at clinical examination as experimental and control groups, in order