

## Editorial Commentary

# Vascular Birthmarks - Need a conceptual change in their understanding and management

Not too long ago I had the privilege of reviewing a book 'The Management of Haemangiomas and Vascular Malformations of the Head and Neck' authored by Prof. K.S. Goleria and I will rely solely on the contents of this book while writing this commentary. These lesions owe their identity to the presence of various channels, it is the behaviour of the dominant channels that decides the behaviour of a particular lesion, which in turn is dependent upon their location in the vascular tree, their size and the rate of flow through them. This in turn decides both - the investigative modalities to identify these lesions and the therapeutic modalities they would respond to like lasers, radio frequency (RF), embolization and surgery.

Investigative modalities like Ultrasound, Doppler, CT, MRI, angiography and DSA can readily differentiate the various types of vascular malformations and haemangiomas into four clinically identifiable management oriented groups – the small channel high flow lesions (congenital haemangioma of infancy or CHI), small channel low flow lesions (capillary, lymphatic, venule combination CLVC), large channel low flow lesions (vascular malformations – VM) and large channel high flow lesions (arterio-venous malformations - AVMs).

In Venous Malformations- the large channel low flow lesions it is of vital importance to arrive at a correct diagnosis of the predominant component of the lesion in order to avoid surprises like profuse bleeding per operatively. In venous malformations the input must be ligated before the output and the dissection should be in

a centripetal fashion. Sclerotherapy has been advocated for small and residual lesions in this group.

The large channel high flow lesions however grow relentlessly and treating them by blocking the main blood vessels without treating the nidus spells doom as it results in a severe re growth with multiple new feeders. Ablation of the feeders along with the nidus by embolization, surgery or a combination of both is a must for results to be lasting and recurrence free. The nidus, which is a fast flow system involving both arteries and veins, a conglomeration of many A-V fistulae is the cause of 'steal', which in turn promotes the nidus. Hence, the only way to treat AVM is to totally eliminate the nidus.

Another modality that we should not forget is the laser. This causes photocoagulation resulting in thrombosis of the channels and involution of the lesions with less morbidity and minimal scarring. The target chromophores - haemoglobin, oxy-haemoglobin, melanin and water get hit by lasers and the heat energy thus produced coagulates blood, chars vessel walls and produce thrombosis best in small channel low flow lesions.

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