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Laser Treatment of Venous Malformations

Oren Sarig, MD, Sol Kimel, PhD, and Arie Orenstein, MD

Abstract: Venous malformations (VM) are developmental errors comprised of enlarged dysplastic blood vessels. Clinically they manifest as either a faint blue patch or a soft blue vascular mass. Treatment options include a sclerosing agent (such as alcohol or ethiblock), surgery or laser therapy. A review of the literature concerning VM and laser treatment yielded a few sporadic reports describing series of 3 to 46 cases. In this retrospective study we reviewed the files of the relevant cases. The data included the extent of the lesion, the different anatomical sites, age of patient and the extent of clearance of the lesion as the treatment's end point. This study includes 56 cases which makes it the largest series presented.

The success rate of laser treatment was 92.8%. The immediate complication rate was very low (approximately 3.57%) including minimal scarring and deformity. No long term complications were noted. Our study concludes that treatment of VM is a difficult task due to the nature of the lesions. Laser treatment of these lesions enables one to obtain good results with a very low incidence of complications. Surgery and other treatment modalities are not always satisfactory, yield similar or less efficient results and have a higher complication rate. Laser treatment can play an important role in the treatment of VM and in fact may be the treatment of choice in some settings.

Key Words: venous malformations, lasers, treatment, primary laser malformations

(*Ann Plast Surg* 2006;57: 20–24)

Venous malformations (VM) are developmental errors usually present at birth.[1] Treatment options include a sclerosing agent, surgery, or laser therapy.[2]

The aim of this article is to present a large series (the largest to our knowledge), with the longest follow up time. We define a new term, namely, primary VM.

Our work shows good results, with a low complication rate when compared with other methods of treatment. We suggest that this may in fact be the treatment of choice in some settings.

We herewith present our results in the treatment of primary VM using the YAG laser.

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MATERIALS AND METHODS

Patient Profile and Treatment Procedures

In this retrospective study we reviewed the files of the relevant cases (our definition of primary VM). All available patient data were extracted from the charts. The data included the extent of the lesion, the different anatomic sites, age of patient, and the extent of clearance of the lesion as the treatment's end point.

We defined 4 categories and scored them accordingly: class 1, excellent result (4 points), complete resolution of the lesion; class 2, good result (3 points), more than 50% improvement in the final size of the lesion after treatment; class 3, intermediate result (2 points), less than 50% improvement in the final size of the lesion after treatment; class 4, bad result (1 point), no improvement at all or the lesion returned to its initial size.

Other data gathered from the charts included time of onset, depth of the lesion, age of the patient, and follow-up time.

Heterogeneity of lesion,[4] sites, and initial sizes dictated treatment by a number of laser modalities. Some patients were treated in an outpatient setting using topical anesthesia (EMLA, lidocaine, or both), while others underwent procedures under general anesthesia (for example, in the case of giant VM in the region of the glans penis, repeated sessions were performed at 3-month intervals).

In some cases, the YAG laser was used by passing the fiber directly into the lesion.

Equipment Used

Continuous wave (CW), NdYAG laser of L.B.T. (Israel). Parameters used: 20–25 W, pulse duration: 1–2/s. Spot size was 0.8 mm. The number of pulses used was according to the size and thickness of the lesion, ranging from 2 to 50. The energy was transmitted to the vascular lesion by direct contact of the YAG laser fiber (800-μm-diameter, manufacturer).

The "lovely" system of Msq Ltd. (Israel). In this system, a long pulsed 1064-nm handpiece is used. The parameters used were a 3-mm spot size, 10 m/s pulse duration, and a fluence of 250 J/cm^2.

RESULTS

A total of 56 cases of primary VM, located at different anatomic sites, were treated.

Success Rate

Forty patients comprising a total of 71.4% were ranked as class 1 (4 points), 12 patients (21.4%) were ranked as class 2

FIGURE 1. Laser treatment of VM of the cheek.

FIGURE 2. Laser treatment of VM of the cheek.

FIGURE 3. Laser treatment of VM of the cheek.

FIGURE 4. Laser treatment of VM of the cheek.

FIGURE 5. Laser treatment of VM of the lower lip.

FIGURE 6. Laser treatment of VM of the lower lip.

(3 points), 4 patients (7.2%) were ranked as class 4 (1 point). None were scored as class 3, as shown in Tables 1 and 2.

Complications

Almost no long-term complications were noted. Among the few complications that occurred was a scar in the lower lip region without distortion of the lip. In another case, a patient treated in the region of the lower lip developed a granuloma a few weeks later. The granuloma was ablated as part of the second treatment without further sequelae (the patient underwent a total of 4 sessions).

FIGURE 7. VM of the lower lip. This patient had been treated with radiotherapy during childhood as well.

FIGURE 8. VM of the lower lip. This patient had been treated with radiotherapy during childhood as well.

FIGURE 9. VM of the lower lip. This patient had been treated with radiotherapy during childhood as well.

In 1 case, a patient with a lower-lip lesion, treatment was terminated after 8 sessions due to a concern that further treatment would distort the lip. The final grading score of this patient was good (3 points).

FIGURE 10. VM of the lower lip. This patient had been treated with radiotherapy during childhood as well.

FIGURE 11. VM of the lower lip. This patient had been treated with radiotherapy during childhood as well.

Four patients had undergone surgical treatment during childhood. Two patients had previously undergone radiotherapy. The remainder of the patients had received no previous treatment of the lesions.

DISCUSSION

VM are developmental errors composed of enlarged dysplastic blood vessels lined by quiescent endothelium. They are usually present at birth. Under a light microscope, they are seen as channels and sacs filled with blood, organizing thrombi, and phleboliths. The dysplastic venous networks drain to adjacent veins, many of which are varicose and deficient of valves. Clinically, they manifest as either a faint blue patch or a soft blue vascular mass.

They are usually solitary. They are easily compressible and exhibit increased swelling when dependent. Patients often complain of pain and stiffness in the affected area, especially in the morning. They usually exhibit a proportionate growth during childhood but often enlarge during puberty. In the face, they are usually unilateral and, due to a mass effect, may cause asymmetry and a progressive distortion of

FIGURE 12. Laser treatment of VM of the penis.

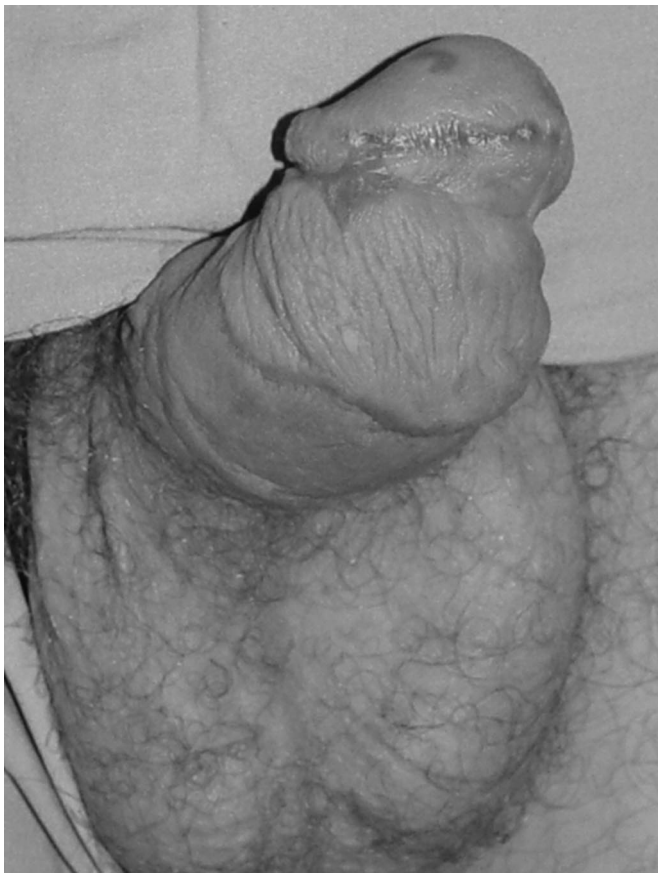

FIGURE 13. Laser treatment of VM of the penis.

TABLE 1. Distribution of Anatomic Sites and Treatment Scores

Anatomic Site	No. Cases	%	Average Score
Lips (upper and lower)	36	64.2	3.58
Cheeks	6	10.6	3.67
Tongue	1	1.8	4
Nose	2	3.6	2.5
Eyelids	1	1.8	4
Neck	2	3.6	4
Trunk	3	5.4	2.66
Limbs	3	5.4	4
Penis	2	3.6	4
Total and weighted average	56	100	3.57

The mean age was 37.5 yr, with a range of 4 to 79 yr. The average follow-up time was 5 yr, with a range of 6 mo to 14 yr. The average number of treatment sessions was 3.25 (ranging from 1 to 13). The average weighted treatment score was 3.57. Scores according to anatomic site are listed in Table 1.

TABLE 2. Distribution of Scores

Class	No. Patients	%
Class 1 (4 points), excellent	40	71.4
Class 2 (3 points), good	12	21.4
Class 3 (2 points), intermediate	0	0
Class 4 (1 points), bad	4	7.2

facial features.[1] It is important to note that they have a propensity for recanalization and recurrence.[2]

In the literature, various terms are used to describe VM, sometimes causing confusion.[2,4,10,11] Therefore, it is difficult to find a reliable series pertaining to VM.[2–11]

VM arise from 2 distinct pathways. The first pathway is what we define as primary VM. By this we mean that the defect seen on the patients was formed by the process mentioned above and enlarges due to increasing hydrostatic pressure (a physical mechanism). The second pathway starts from a cavernous hemangioma formed due to faulty angiogenesis (a biologic mechanism), which regresses with time. This anomaly may further develop into a secondary VM, a capillary malformation, a combination of both, or be dissolved completely. These 2 forms of VM can be differentiated by proper history taking. The distinction is important since in this article we deal only with primary VM.

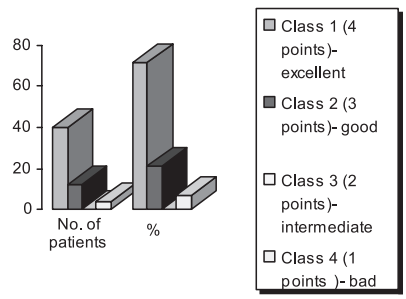

FIGURE 14. Distribution of scores.

Treatment options include a sclerosing agent (such as alcohol or ethiblock), surgery, or laser therapy. Surgery may be technically difficult since such lesions may be very large at presentation. The borders of the lesions may be poorly defined, bleeding may be profuse, and deformities may occur as a result.

Sclerotherapy may be used alone or as an adjunct to surgery but may be accompanied by various adverse effects such as prolonged local swelling, skin necrosis, neural damage, renal toxicity, and cardiac arrest. Surgery also causes severe pain, large scars, deformities, and often requires general anesthesia and hospitalization.[2]

A review of the literature concerning VM and laser treatment yielded a few sporadic reports describing series of 3 to 46 cases.[2–12] Other articles published on this topic included a small number of cases reported in each, the largest of which contained 46 cases but with a mean follow-up of only 6 to 24 months.[9] In another article describing 10 patients with lesions of the lip, 3 had posttreatment complications. They included bleeding that required surgical intervention, an ulcer, which healed with conservative management, and a commissural contracture, which required correction by z-plasty.[5]

In cases of penile lesions, operative treatment is not suggested because of the high frequency of postoperative bleeding and poor healing (scar tissue). In this setting, our data, together with the work of Ramos et al,[7] suggest that laser is the treatment of choice. Other complications reported in the literature were superficial burns and 1 case of lip herpes eruption after treatment.[2]

VM may be difficult to cure and may be treated by various methods such as surgery and sclerotherapy, though both have their limitations. Laser treatment carries great promise. Unfortunately the reports in the literature are sparse and for the most part anecdotal. Our data suggest that laser treatment of VM, when employed properly, is in fact the method of choice in some settings due to its high success rate and very low incidence of complications.

The primary mechanism by which the laser exerts its effect is by nonspecific thermal coagulation, which occludes and shrinks the lesion.[2]

The challenge of treating VM is to get enough photons through the enlarged blood vessel and cause immediate shrinkage and occlusion. The choice of wavelength, energy fluence, and pulse duration of laser exposure are all related to type and size of target vessels and tissue conduction scattering phenomenon. Large-diameter blood vessels (as in VM), require long pulse duration to allow diffusion of heat evenly through the cylindrical vessel lumen. Delivery of energy should occur without causing a shock wave, including gentle but fast heat conduction, to prevent posttreatment hemorrhage and purpura. Energy should pass through the epidermis and dermis without being absorbed so as to reduce the harmful thermal effects on these layers.

These requirements can be achieved by using the Nd:YAG (1064 nm) laser, which has deep penetration in tissues since it is poorly absorbed by water molecules, which are the main chromophores in the near infrared. It lacks absorption by melanin, thus enabling uniform pan vessel heating. To minimize epidermal damage, cooling by a spray device may be used. Cooling enables use of higher fluences, thus allowing more potential for producing pan endothelial vascular destruction.[13,14] The damaged vessels are replaced by normal connective tissues.[6]

The technique of introducing the fiber tip of the YAG laser through the epidermal and dermal layers enables the emission of laser energy directly into the blood vessels. This facilitates in reducing loss of energy to the superficial layers of the skin, thus increasing efficacy of blood vessel occlusion, and helps to reduce scars over the epidermis.

CONCLUSIONS

Treatment of VM is a difficult task due to the nature of the lesions. Laser treatment of these lesions enables one to obtain good results with a very low incidence of complications. Surgery and other treatment modalities are not always satisfactory, yield similar or less efficient results, and have a higher complication rate. Laser treatment can play an important role in the treatment of VM and in fact may be the treatment of choice in some settings.

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