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Erectile dysfunction following Nd-YAG visual laser-assisted prostatectomy (VLAP)

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We investigated if side fire Nd-YAG visual laser prostatectomy (VLAP) causes erectile dysfunction (ED) in patients who were sexually active prior to the procedure. The 36 study patients gave a detailed medical/sexual history: physical examination included bulbo-cavernous reflex (BCR) on rectal examination and routine blood tests. Lasing time, power of the applied laser beam (in watts), coagulation site and energy intensity were recorded. Patients with new onset ED during the one year study period underwent pudendal nerve conduction (PNC), color duplex Doppler ultrasonography, and NPT/RigiScan testing.

In 6 out of 36 (16.7%) patients reporting significant post-operative ED, there was a tendency towards higher energy applied and longer lasing time but no correlation between prostate size or the site of energy application. Patients reported loss of night and/or morning erections ($n = 5$), retrograde ejaculations ($n = 2$), loss of ejaculate ($n = 2$), and decreased sensation of orgasm ($n = 3$). Three had abnormal PNC, duplex Doppler showed abnormal blood supply in four, and all six had abnormal NPT/RigiScan.

We believe this is the first demonstration that VLAP may be associated with a high rate of ED and that the lasing time and intensity of applied laser energy may play a role in this outcome.

Keywords: laser prostatectomy; Nd-YAG laser; erectile dysfunction; impotence

Introduction

During the last decade, a variety of alternative methods have been introduced for treatment of benign prostate hyperplasia (BPH). Although transurethral resection of the prostate (TURP) still remains the 'gold standard', new developments have produced many alternatives for medical or minimal surgical treatment of BPH in an effort to decrease perioperative mortality and postoperative morbidity following TURP.[1–6]

Increasing enthusiasm has recently been shown for using laser technology for the treatment of BPH, and Nd-YAG visual laser prostatectomy (VLAP) has been accepted worldwide. It has a reported success rate comparable to that of TURP but with fewer perioperative complications, reduced hospitalization time, and reduced cost.[6–8] In this method of treatment, a high energy Nd-YAG laser beam is directed at the prostate inner surface and penetrates into the tissue. Testing of thermodynamics after VLAP in several in vitro and in vivo studies showed that tissue damage occurs after the laser beam application penetrates the prostate tissue to a depth of at least 0.4–1.4 cm.[1,2,9–12] However, the extent of the thermal damage outside the prostatic capsule has not been evaluated nor has the late complication rate of VLAP been well defined.

The incidence of ED following laser prostatectomy was estimated to be 0–5.4% of the patients whose preoperative sexual function had been normal.[1,2,4] The purpose of this prospective, open study was to detect whether side-firing Nd-YAG VLAP causes significant ED in patients who were sexually active prior to the surgery, and to identify any additional risk factors that may effect sexual function following this procedure.

Materials and methods

Out of 65 candidates for this study, 36 patients were recruited from our outpatient clinic had undergone VLAP due to symptomatic BPH, having had previously failed to obtain adequate improvement following medical treatment. All were potent and sexually active prior to the VLAP procedure. The mean age was 65.8 y (range 56–72 y). A detailed medical history including sexual function was

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obtained from all patients who also completed a sexual questionnaire and the AUA symptom score. All men underwent a complete physical examination including bulbo-cavernous reflex (BCR), routine blood tests, urinalysis including urinary culture, uroflowmetry, measurement of residual urine volume on abdominal US, cystourethroscopy for estimation of the prostate length and exclusion of other abnormalities, and prostatic volume measurement by TRUS. Those patients who complained of ED after the prostatectomy underwent pudendal nerve conduction (PNC), color duplex Doppler ultrasound, and NPT/RigiScan test. PNC was performed with the patient in a supine position with abducted legs. A Modeled/TECA (USA) Sapphire EMG apparatus was used and recordings by a concentric needle electrode were made directly from the striated bulbocavernous muscle. Stimulation of the penis was evoked by a bipolar stimulator, and stimulation of the pudendal nerve evoked reflective contractions of the bulbocavernous muscle. Both early and late latencies and responses of the reflexes were measured. The dynamic color duplex Doppler ultrasound evaluations were performed before and after intracavernous injection of prostaglandin E1 (PGE1) 10 μg. An NPT/RigiScan was considered as being abnormal if rigidity was less than 60% of the RigiScan relative scale (100% equals the rigidity of a wooden dowel) at the base and tip of the penis, and if it lasted for less than 10 min.

Operating procedure

All patients received the same perioperative antibiotic prophylaxis protocol. Twenty-four (67%) underwent the operation under local anesthesia. We used a side-firing laser (MyriadLase, Israel) and employed an operative technique similar to that described by Costello *et al*.[1] Four quadrant applications to the obstructive lateral lobes of the prostate were performed at the two, four, eight and ten o'clock positions. For glands over 3 cm in length, two sets of four quadrant laser applications were performed approximally 2 cm distal from each other. The applied laser power was 60 W and the lasing time applied at each point was 60 s, yielding a total 3600 J per spot application. The mean operating time was 27 min and mean total laser energy used was 39000 J. The average hospitalization time was two days and a catheter remained in the bladder for six days.

Results

During the one year follow-up period, 6 out of 36 (16.7%) previously potent patients (per history) reported new onset ED and related problems. Comparison of the demographic and technical surgical parameters showed that while the mean lasing time and total applied laser energy were higher in the group of patients with ED (43 000 J and 31 min *vs* 39 000 J and 25 min, respectively), the size of the prostate gland was quite similar in both groups (Table 1). The mean time of ED appearance was three months (range 1–6). Three patients reported complete absence of erection and three complained of inadequate erection for vaginal penetration. Five patients lost night and/or morning erections, two patients had retrograde ejaculations, two patients reported loss of ejaculate, and three patients had consistently decreased extent of the sensation of orgasm compared to the preoperative quality. Abnormal PNC was recorded in three patients (all of whom were treated with more than 30000 J for more than 30 min), duplex Doppler study showed abnormal arterial blood flow in three patients (peak systolic velocity [PVS] < 25 ml/s), and both arterial and venous blood flows were abnormal (PVS < 25 ml/s and end-diastolic velocity – [EDV] > 7 ml/s, respectively) in one patient. The NPT/RigiScan was abnormal in all six cases. There was no correlation between the exact point of direction of the beam and the appearance of ED.

Discussion

Laser prostatectomy is playing an increasingly important role in the treatment of BPH, with VLAP being the most frequently used procedure.[1–8] Although VLAP has been recognized as an effective and safe treatment, the urologist needs to under-

Table 1 Demographic and technical parameters of study groups

Parameters	*ED patients (n = 6)*	*No ED patients (n = 30)*
Mean age (y)	65.8	71.6
Mean prostatic volume (cc)	27	31.8
Mean catheter time (d)	5.5	6
Mean lasing time in min (range)	31 (10–40)	25 (10–48)
Mean laser energy in joules (range)	43 000 (14 000–48 000)	39 000 (11 000–119 000)
Median laser energy in joules	36 000	32 500

stand the possible effect of laser beam on prostatic and surrounding tissue, the effect of the lasing time and applying power, the characteristics of the fibers used, and the possible complications involved.

The reported postoperative incidence of ED following TURP is 4–35%,[13] while ED after VLAP prostatectomy is reportedly relatively low: although several authors stated that there was no effect of VLAP on erectile function, others have described ED after this operation at an incidence up to 5.4%.[1–7] Despite the lack of clarity, no study has specifically addressed the determination of the true incidence of ED using objective parameters, the characteristics of such a complication, and its exact etiology. While evidence of ED after laser prostatectomy in a canine model and possible etiological factors were presented by Breza *et al* and others,[10,14] our study is the first prospective one to delineate the incidence of ED and related abnormalities in patients who were sexually active prior the operation, indicating that ED and related abnormalities are much more prevalent (16.7%) than the earlier reported rate. The pathophysiology of ED and related abnormalities following VLAP has yet to be clarified. Various possible explanations have been suggested by different investigators, including damage to the penile innervation and vascularization because of the duration of time for laser beam application, the power of the energy delivered, the size of prostate gland, and the place of the applied laser beam. Psychological factors such as anxiety related to the operation and a negative influence of the perioperative stress may also inhibit the reinitiation of normal sexual life.[4,10–12, 14–17]

Nerve damage during laser prostatectomy can explain the evidence of the post-operative ED. The neuroanatomy of the penile erection has been well described by Lue *et al*,[18] who showed that the cavernous nerves are in intimate contact with the prostatic capsule in the distal prostatic urethra and at the apex, and that the distance between the capsule and nerve supply is only a few millimeters. It has been shown that higher laser power resulted in higher temperatures at greater depths and in larger coagulated volumetric lesions. The 60 W irradiations deliver 50% more joules than do the 40 W ones. It has also been shown that increasing the applied laser energy from 20 W to 60 W may increase the depth of coagulation lesions from 0.4 mm up to 1.4 cm, respectively, and that this may probably cause damage to the nerve supply.[4,6,10–16] We had routinely used 60 W of laser energy and this might have been one of the reasons for ED. That three patients showed abnormal PNC findings may support this hypothesis. The occurrence of ED in only in 6 out of 36 patients who were exposed to the same laser power indicates that other factors are culpable. It has been suggested that longer lasing time at high power energy results in larger lesion width, depth, and volume.[4,5,11,12,14–17] Thus, the high power density fibers heat more deeply in less time and at a lower wattage than the lower power density fibers. However, the depth of lesion was not always linearly proportional to irradiation time.[11,12,16] The operation time in four of six of our patients with ED was more than 20 min: this may be another explanation for the occurrence of ED.

Blood supply to the penis and to the prostate is mainly provided from the same arteries with some variations.[15] Latent compromise of vascular supply or vascular injury during the evaporation of the prostate can cause occlusion of penile vessels and may further compromise penile blood flow and erectile function. Duplex Doppler ultrasound in three of our patients demonstrated decreased arterial blood flow (PSV < 25 ml/s), and there was decreased arterial and venous blood flow (PSV < 25 ml/s, EDV > 5 ml/s) in another patient. Although we did not conduct preoperative duplex Doppler examinations in these patients, there were no signs of vascular abnormalities on the preoperative physical examination, which may support there having been new onset of vascular insufficiency.

Ejaculatory dysfunction following VLAP has been described in several publications as having occurred in 8–15% of cases.[2,6,7] We found this complication in 4 out of 36 patients (11.1%), indicating that although it occurs much less frequently than the reported incidence of ejaculatory dysfunction after TURP, this is still a significant difference. The cause of this complication is not well understood and we can offer no satisfactory explanation for it at this point.

In three of the six patients with ED, the size of the prostate was less than 30 cc. Although this is in correlation with the findings of several investigations which used potato and animal models[12,15,16] and showed that the depth of the coagulated lesion is in linear proportion with prostate size and configuration, we were not able to arrive at the same conclusion due to lack of any significant difference in the prostate size among all our study patients.

We failed to find any correlation between ED and site of laser energy application. We were also unable to find any report in the English literature explaining this particular relationship, and recommend more specific animal and human studies to shed light on this question.

Conclusions

We demonstrated a high incidence of ED after VLAP. Lasing time and intensity of applied laser energy may have an effect on erectile function. Urologists should be made aware of this complication. Patients undergoing laser surgery for BPH should be informed about ED, and this information should appear on the patient consent form.

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Editorial comment

Erectile dysfunction following Nd-YAG visual laser-assisted prostatectomy (VLAP)—by J Chen *et al.*

This manuscript is interesting, and, if the observation that 16.7% of men undergoing VLAP experience erectile dysfunction is correct, a potentially significant paper. The authors studied the six patients who complained of erectile dysfunction post-operatively and found that 'three had abnormal pudendal nerve conduction, duplex Doppler showed abnormal blood supply in four, and all six had abnormal NPT/Rigi-Scan.' Do these tests help us understand the etiology of erectile dysfunction reported in this series, or for that matter, in any series?

Pudendal nerve conduction (PNC) was performed by stimulating the dorsal nerve of the penis (a branch of the pudendal nerve) and recording evoked contractions of the bulbocavernosus muscle. Although the afferent portion of this reflex is a sensory nerve of the penis, the efferent (motor) portion does not control penile erection. We would like to measure the integrity of the cavernosal nerves, but currently we have no tests which can do this. Prior reports have indicated that penile dorsal nerve conduction times may be related to diabetes,[1] but, I, personally, do not believe that measurement of PNC is clinically helpful in determining the etiology or presence of erectile dysfunction in any patient. Even if the pudendal nerve were involved in the production of penile erection, injury to this nerve during VLAP would be very unlikely. 'The pudendal nerve does not contribute to the pelvic plexus but leaves the pelvis through the lower part of the greater sciatic foramen,...'.[2]

In addition to abnormal PNC, four patients had an 'abnormal' blood supply, and all six had 'abnormal NPT/RIGI-Scan.' I would not be surprised if four of six patients aged 65.8 y who said they were potent had peak systolic velocities < 25 cm/s following the intracavernous injection of 10 μg PGE1. How accurate is a cut point of 25 cm/s in this age group? The NPT/Rigi-Scan data is difficult to interpret since we don't know if they were 'abnormal' preoperatively when the patients reported normal potency.

I don't believe that our 'tests' used to determine the presence and etiology of erectile dysfunction are nearly as good as we think they are. If 16.5% of patients complain of new onset erectile dysfunction following VLAP, a problem exists. Our current ability to identify injury to the likely culprit, the cavernosal nerves, is very limited.

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