Ministry of Higher Education and Scientific Research

University of Baghdad
Institute of Laser for Postgraduate Studies

Response of melanocytic nevi to fractional CO_2 laser treatment

A Dissertation Submitted to The Institute of Laser for Postgraduate Studies, University of Baghdad.In Partial Fulfillment of the Requirement for the Degree of Diploma in Laser Medicine /Dermatology.

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2022 A.D 1443 A.H

بِسم الله الرحمٰن الرحيم

وَ سَخَّرَ لَكُمُ ٱلَّيلَ و ٱلنَّهَارَ وَ ٱلشَّمۡسَ وَ ٱلۡقَمَرَۖ وَٱلنُّجُومُ مُسَخَّرَٰتُۢ بِأَمۡرِهِۦٓۗ إِنَّ فِي ذَٰلِكَ لَأٓيَٰتٖ لِّقَوۡمٖ يَعۡقِلُونَ ۞

صدق الله العظيم

(سورة النحل: الاية 12)

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Dedication

To the spirit of my father…
To my great mother…
To my beloved wife and children.

Acknowledgments

I gratefully acknowledge the continuous encouragement offered by **Dr. Hussein Ali Jawad.** Dean of the Institute of Laser for Post Graduate Studies, University of Baghdad for his guardianship.

I gratefully acknowledge the continuous help and encouragement offered by Dr. Layla Mohammed Al-Ameri. the head of Biomedical Department/Iinstitute of Laser for Post graduate Studies, University of Baghdad.

I feel much indebted to, the supervisor, Assist.Prof. Dr. Mazin Hamid Ayyash. Department of Dermatology College of Medicine Tikrit University for his advises and precise supervision.

My gratitude and appreciation for the full cooperation from the staff of the Institute.

Finally, I wish to express my thanks to my colleagues in the department.

Abstract

Background: Acquired melanocytic nevi are common dermatological disease, many people regard this nevi unsightly and required removal, many modalities were used but with adverse effects.

Objectives: To assess the response of melanocytic nevi to fractional co_2 laser as a new treatment modality.

Methods: This study tried use fractional co_2 laser for removal of 35 nevi lesions in 17 patients and followed up for period 3-4 months.

Results: From 35 nevi lesions, 25 (71%) nevi were cleared with one session , 8lesions(23%) required two sessions while 2 lesion(6%) nevi required for three sessions respectively. All treated nevi showed complete clinical pigmentation clearance at the end of the last session. Side effects: Fortunately, most side effect were mild and transient were seen in 8 patients (47 %), At the 3 months follow-up , hyperpigmentation were in 3 (17 %), atrophic and hypertrophic scars in 2 (12%), recurrences in 2 (12%), and hypopigmentation in 1 (6%) these side effects treated accordingly.

Conclusions: Fractional CO_2 laser produced excellent cosmetic results, so it is good modality for treatment.

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List of Abbreviations:

cm	Centimeter
CO_2	Carbon dioxide
°C	Celsius degree
KW	Kilo watt
W	Watt
Er:YAG	Erbium: Yttrium Aluminum Garnet
UV	Ultraviolet
nm	Nano meter
µm	Micrometer
SPTL	Selective photo thermolysis
RTD	Resistance temperature device
LLLT	Law level laser therapy
CW	Continuous wave
Q-CW	Quasi-Continuous wave
MW	Megawatts
mj	mill joules
`ms	Mill second
mm	Mill meter

Chapter One

Introduction
And
Basic Concepts

1-MELANOCYTIC NEVI

1.1.1Definition of Nevus: A benign growth on the skin that is formed by a cluster of melanocytes (cells that make a substance called melanin, which gives color to skin and eyes). A nevus is usually dark and may be raised from the skin. Also called mole.[1]

Melanocytes are pigment-producing cells in the skin and typically reside within the epidermis, at the dermo epidermal junction and within hair follicles. Several benign neoplasms are derived from melanocytes and are typically the result of individual oncogenic mutations.[2] Many adults have nevi, but their abundance varies tremendously from individual to individual, ranging from just a few nevi up to hundreds of lesions per person. Nevi are rarely present at birth and when they are, are known as congenital nevi. Rather, most nevi form later on in life, typically during the first and second decades.[3] Total nevus number in any given individual is thought to peak during the third decade of life. This peak is due to reduced formation of new nevi (which becomes less common after 30 years of age) combined with the clinical regression of some existing nevi. Clinical regression of nevi is a poorly understood process during which nevi involute and can disappear entirely. The frequency of nevus regression increases with advancing age.[4]

Compared with other clinically apparent, benign, but potentially precancerous lesions, melanocytic nevi are unique as they arise relatively early in life. In contrast, for example, actinic keratosis, which can be a precursor of cutaneous squamous cell carcinoma, are uncommon prior to the age of 40 and become much more prevalent with advancing age, even into the 80s and 90s. The reason(s) why nevi arise primarily during the first two decades of life and less so with advancing age is unclear. The reason why some individuals get only a few nevi, whereas others get hundreds are also

not well understood. In terms of abundance, a combination of inherited causes and ultraviolet radiation and other environmental mutagens, are likely at play. Germline mutations such as in *CDKN2A*, which affects both nevus size and total nevus counts, underlie this phenotype in a small subset of patients. [5,6]

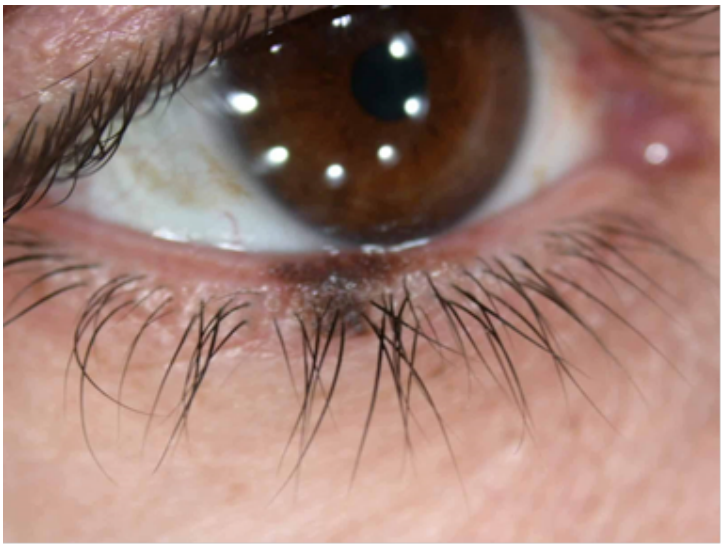


Fig 1.1: A melanocytic nevus occurring within conjunctival epithelium.[7]

1.1.2Clinical and histopathologic features

Nevi are most often 2–6 mm in size and have a uniform color and symmetric architecture clinically. Nevi are grouped into one of three major categories: junctional (melanocytes confined to the epidermis only), intradermal (confined to the dermis only) and compound (both an epidermal and a dermal component) , fourth type is combined (compound and blue nevus in the center) ,as shown in(fig 1.2).[7]

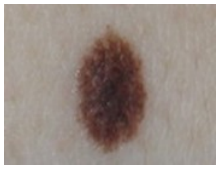
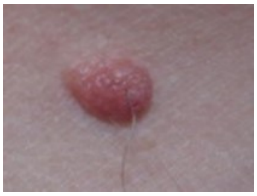
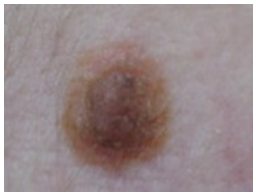
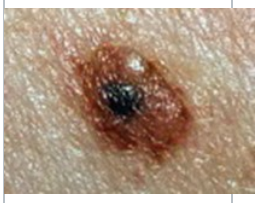
A junctional nevus	**A dermal or intradermal nevus**	**A compound naevus**	**A combined nevus**
has groups or nests of nevus cells at the junction of the epidermis and the dermis. A flat mole.	has nevus cell nests in the dermis. A papule, plaque or nodule with a pedunculated, papillomatoses (Unna nevus) or smooth surface (Miescher nevus).	has nests of naevus cells at the epidermal-dermal junction as well as within the dermis. A central raised area surrounded by a flat patch.	has two distinct types of mole within the same lesion – usually blue nevus and compound nevus.
			

Fig. 1.2 The pathological classification of melanocytic nevi relates to where nevus cells are found in the skin.[7]

. The relationship among these three different types of nevi and what factor(s) result in the formation of one type versus another are not well understood. $BRAF^{V600E}$ mutations, which are found in the majority of nevi, appear to occur with relatively similar frequencies in all three types, but may be slightly more common in nevi with a dermal component.[6]

Despite the heterogeneity in clinical and histologic appearance of these types of acquired nevi, all are thought to share a relatively similar natural history and relationship to melanoma. For the purposes of this review, all three types will be considered together. It should be noted that additional types of benign melanocytic nevi such as: blue nevi, Spitz nevi and deep penetrating nevi exist, however, are relatively less common.[8-10]

Microscopically, nevi are well circumscribed, symmetric and are composed of melanocytes with a monotonous, banal cytology. Two cardinal

histopathological features of nevi are nesting and maturation. Nesting refers to the tendency of nevus melanocytes to form small clusters of cells within tissue. Maturation is a feature of nevi with a dermal component and refers to a gradual and progressive change (from superficial to deep) in nest architecture and melanocyte cytology. As one goes deeper into the lesion, nest size decreases, cell and nuclear volume decreases, pigment production decreases and changes in cell shape occur. [11]as shown in (Figure 1.3).

The Cytologic features of maturation have been used to divide the melanocytes in individual nevi into three groups, types A, B and C. Type A melanocytes are most similar in morphology to normal epidermal melanocytes and are found in nests in the most superficial portions of nevi, including the epidermis and superficial dermis. Type B melanocytes are found in the mid dermis in relatively smaller nests and are also relatively smaller in size and rounder in shape. Type C melanocytes are found primarily as individual cells in lower portions of the dermis and have a more spindled/fusiform morphology. The complex architecture observed in nevi suggests that both cell intrinsic and extrinsic factors act in concert to shape nevus formation, prevent uncontrolled growth and maintain homeostasis. In melanoma, organized nesting and maturation tend to be lost. It is possible that nesting and/or maturation reflect poorly understood tumor-suppressive interactions within the tissue microenvironment, however, there is currently no data to support what (if any) active role these processes have in constraining nevus growth.[12]

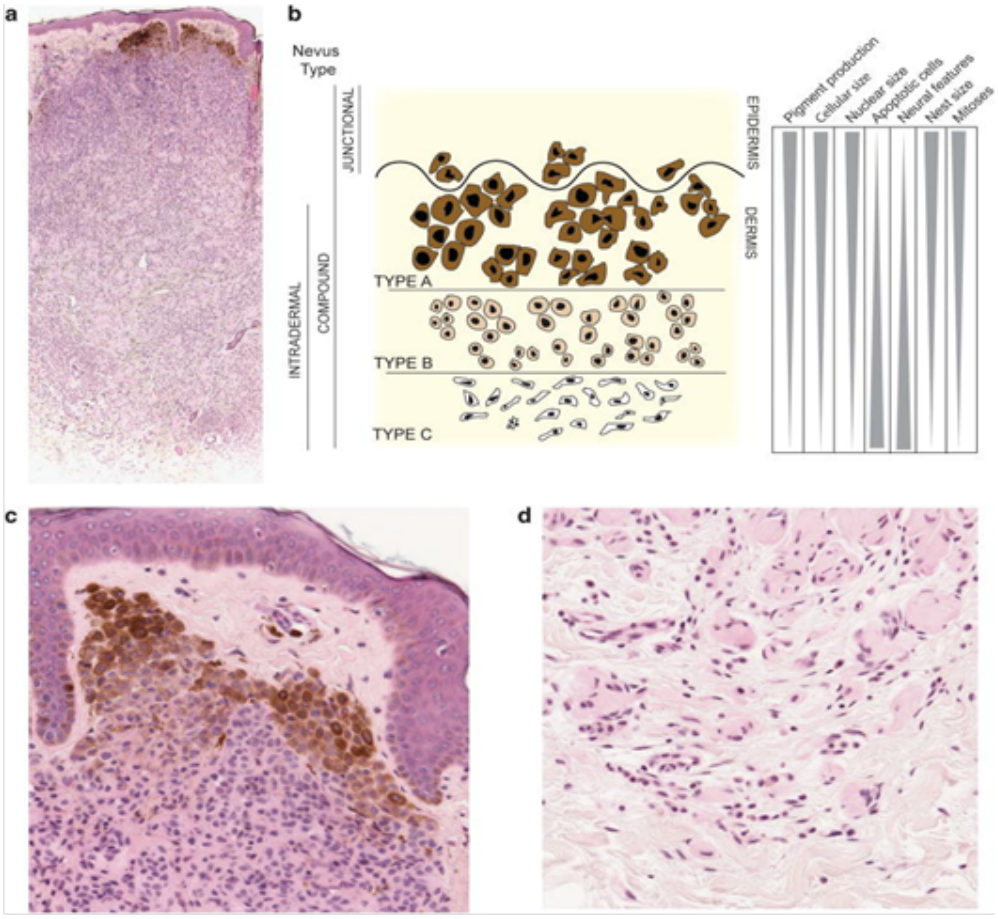


Fig 1.3: Schematic of melanocytic nevus architecture. (**a**) Low power image of an intradermal melanocytic nevus stained with hematoxylin and eosin (H&E). The nevus shows features of maturation. (**b**) Junctional nevi are confined to the epidermis and appear as pigmented macules. (**c**) High power images of type A melanocytes in the most superficial portion of the nevus. H&E-stained section. (**d**) Type C melanocytes in the deepest portion of the nevus showing neural (Schwannian) differentiation. H&E-stained section.[4]

1.1.3Etiology:

The etiology of melanocytic nevi remains unknown. No established genetic or environmental influences are known to contribute to the development of congenital nevi. However, after studying 144 children in Naples, researchers concluded that development of melanocytic nevi early in

life is the result of complicated relationships among nevus evolution, anatomic location, and environmental and constitutional factors. The specific genetic factors that contribute to the development of acquired melanocytic nevi also remain unknown. However, data suggest that the propensity for developing large numbers of nevi, such as multiple dysplastic nevi, might be inherited as an autosomal dominant trait.[13]

Patients with the familial atypical multiple mole and melanoma syndrome (also known as the dysplastic nevus syndrome) develop dozens to hundreds of melanocytic nevi and have an elevated lifetime risk for the development of melanoma. As the name implies, this disorder is believed to have an inherited basis.[13]

As noted previously, population-based evidence suggests that ultraviolet irradiation may trigger the development of acquired melanocytic nevi. The number of melanocytic nevi in childhood is inversely related to the degree of skin pigmentation and is high in children with poor sun tolerance. The mechanism of this induction has not been adequately investigated, but such induction could represent an example of tumor promotion by ultraviolet light.[13]

1.1.4 Epidemiology

The prevalence in ethnic groups with dark skin is lower than that observed in individuals with fair skin. If ultraviolet exposure represents an inducing agent for the development of melanocytic nevi, then this is unsurprising. Some individuals of northern European extraction, especially those from northern Germany, Holland, Belgium, and the United Kingdom, not uncommonly have large nevi (≥ 1 cm in largest diameter), often of large

number (>50, up to several hundred), with a red-brown color. These nevi have been called atypical moles or dysplastic nevi.[14]

1.1.5 Race

Melanocytic nevi are common lesions in patients with light or fair skin and are less common lesions in dark-skinned individuals. This difference in prevalence is in part attributable to the fact that identifying moles in dark-skinned patients is often difficult, especially if the lesions are macular (flat).[15]

Some authorities have suggested that melanocytic nevi are in part stimulated by exposure to sunlight.[, 15] If so, then individuals with dark skin might have fewer nevi because of the protective properties of melanin. Evidence indicates that broad-spectrum sunscreens attenuate the development/evolution of melanocytic nevi when used in children; therefore, dark-skinned individuals probably have inherent protection against the development of moles because of their cutaneous melanization.[16]

1.1.6 Sex

No clear sex predilection is reported for the development of melanocytic nevi. However, melanocytes have been postulated to exhibit some degree of sex hormone responsiveness. The findings associated with melanocytic nevi during pregnancy support this conclusion. Melanocytic nevi commonly darken and/or enlarge during pregnancy. Melanocytes have been shown to have cytosolic receptors for estrogens and androgens, and melanogenesis is responsive to these steroid hormones. Some melanomas seem to respond to hormones, an observation that might be explained by these cytosolic receptors. Subtle differences may exist in the prevalence of melanocytic nevi between women and men. Judging the incidence and

prevalence based on available biopsy data is difficult because women may be more likely to seek medical attention. If sex-specific variations in incidence do exist, the differences may be site specific. For example, specific histopathological features are commonly observed in melanocytic nevi that occur within genital skin. These features are noted almost exclusively within biopsy specimens from women, although similar alterations can occasionally be observed in melanocytic nevi from males.[17]

1.1.7 Age

By definition, congenital melanocytic nevi are present at birth or soon thereafter, although some small congenital nevi are clearly tardive in their clinical presentation. Current opinion holds that some elements of such nevi are present at birth but remain inconspicuous until some later date.[17]

Nevus subtype incidence may also vary with ages. According to a study by Zalaudek et al, melanocytic nevi of mixed pattern with a peripheral rim of dark globules are sufficiently rare in persons older than 50 years that they should be considered suspicious.[17]

1.1.8 Pathophysiology

Melanocytes are present in the basal layer of the epidermis and exhibit a certain degree of territoriality. Non-neoplastic melanocytes typically exhibit contact inhibition to each other, and thus pigment cells are usually not found as contiguous cells. With certain forms of stimulation, such as the exogenous administration of ultraviolet irradiation, the density of melanocytes in normal epithelium may increase. Normal melanocytes may also involve adnexal epithelium, most notably the bulbs of follicular papillae.[18]

Melanocytic nevi represent proliferations of melanocytes that are in contact with each other, forming small collections of cells known as nests.

Melanocytic nevi commonly form during early childhood. Their onset is believed by some authorities to be, at least in part, a response to sun (ultraviolet) exposure. However, genetic factors are also clearly involved in the development of some types of melanocytic nevi. Some kinships express an autosomal dominant condition (the so-called dysplastic nevus syndrome or the familial atypical multiple mole and melanoma syndrome), in which members have a large number of large nevi, sometimes hundreds, scattered over the integument.[18]

Melanocytic nevi have also been observed to develop or spread rapidly after blistering events, such as second-degree thermal burns, severe sunburns, or Toxic Epidermal Necrolysis or in persons with genetic blistering diseases such as Epidermolysis Bullosa. In such instances, the development of so-called eruptive melanocytic nevi appears to be propagated by a traumatic stimulus, with scattering of melanocytic nevus cells over a large area within a zone of blistering and with the subsequent development of multiple independent melanocytic nevi within the injured area. Growth factors, such as basic fibroblast growth factor, have been suggested to be released by proliferating keratinocytes and could contribute to stimulation of melanocyte proliferation in this context. In summary, the exact etiology behind the development of melanocytic nevi is complex and multifactorial and is incompletely understood.[18]

Acquired melanocytic nevi are considered benign neoplasms. In contrast, congenital melanocytic nevi are perhaps best interpreted as congenital malformations. Melanocytes are of neural crest origin, and congenital nevi probably represent an error in the development and migration of these neuroectodermal elements. Evidence of errant embryological migration can be seen histopathologically within giant congenital melanocytic

nevi. In this context, melanocytes may be found distributed throughout the dermis, around and within the walls of blood vessels, around adnexal structures such as hair follicles, within the subcutis, and sometimes within skeletal muscle, smooth muscle bundles, nerves, or sebaceous glands.[18]

Errant embryological migration is also believed to be the source of melanocytic nevus cell "rests," which can be observed in the capsules of lymph nodes. Occasionally, rests of melanocytes can also be found in the subcapsular space or within lymph node trabecula. The importance of melanocytic nevus rests is that they can sometimes be mistaken for metastatic deposits because of their extracutaneous location.[18]

These rests are not uncommonly associated with agminated blue or cellular blue nevi or with large congenital melanocytic nevi. However, with the advent of sentinel node evaluation, nodal rests of melanocytic nevi clearly are not uncommon and can be found in association with a variety of melanocytic and nonmelanocytic lesions. These rests of cells are sometimes referred to as benign metastases because these cell clusters may represent the end result of an intra lymphatic migration of benign melanocytes. Note, however, that benign nodal melanocytic nevi are almost invariably within the capsule, while melanoma metastases are commonly subcapsular.[18]

1.1.9 History

Melanocytic nevi are common lesions that can be found on the integument of almost all individuals. Some patients present with few lesions, while others have hundreds. The number on a given individual increases in rough proportion to the degree of skin pigmentation. Melanocytic nevi can be broadly divided into **congenital** and **acquired types**. Determining if a lesion is congenital or acquired is generally easily accomplished by direct query of

the patient, although, as noted above, some small congenital melanocytic nevi are tardive and may be perceived by the patient as acquired.[19]

When evaluating the nature of a melanocytic lesion, a number of attributes must be assessed. Further commentary describing physical attributes can be found in Physical. Whether a lesion has become symptomatic (eg, itchy, painful, irritated, or bleeding) is considered an important indicator of potential malignant change. Not all melanocytic nevi that change are malignant, especially if change is noted in a person younger than 40 years. However, change that is perceptible over a short time is an indicator of potential malignancy and designates a lesion deserving of biopsy. An Australian study found that 16% of benign lesions changed over an interval of 2.5-4.5 months. The proportion of benign lesions that changed was higher in persons aged 0-35 years than in those aged 36-65 years but rose again in the elderly (age >65 y). Acquired melanocytic nevi are typically less than a centimeter in diameter and evenly colored.[20]

Melanocytic nevi most commonly are tan to brown, but coloration can be variable, ranging from skin-colored (nonpigmented) to jet black. The deep pigmentation associated with dark melanocytic nevi often stems from associated intracorneal pigmentation. The spectrum of "hypermelanotic" melanocytic nevus includes lesions with heavy epidermal pigmentation.[20]

1. **Dysplastic melanocytic nevi** have also been referred to as Clark nevi. The designation "dysplastic" was applied to such lesions because of an early belief that such lesions might be biologically unstable and represent common precursors of melanoma. Further study has not definitively confirmed that this is the case. Dysplastic nevi present clinically as flattish, pigmented macules or thin papules. Often, a "fried egg" configuration is

apparent, with a central papular area that is flanked by a macular zone of deeper pigmentation.[21] as shown in figure 1.4 .

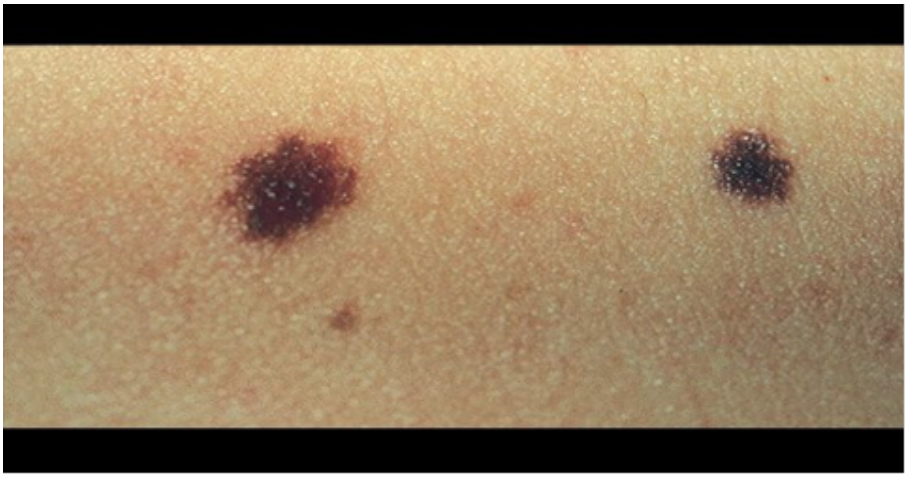


Fig 1.4. Dysplastic melanocytic nevi,[22]

2. **Spitz nevus** represent another distinctive variant of melanocytic nevus. In decades past, such lesions were referred to as "juvenile melanomas," but now they are recognized by specific microscopical features and are believed to be benign. Although Spitz nevi tend to manifest as pink papules on the head of a child, they can be clinically indistinguishable from conventional nevi in some instances; they also can be heavily pigmented. Heavily pigmented Spitz nevi may also be referred to as "Reed nevi" or "pigmented spindle cell nevi." Many Spitz nevi exhibit considerable associated vascular ectasia and, thus, display a hemangioma like clinical appearance. as shown in(figure 1.5).[22]

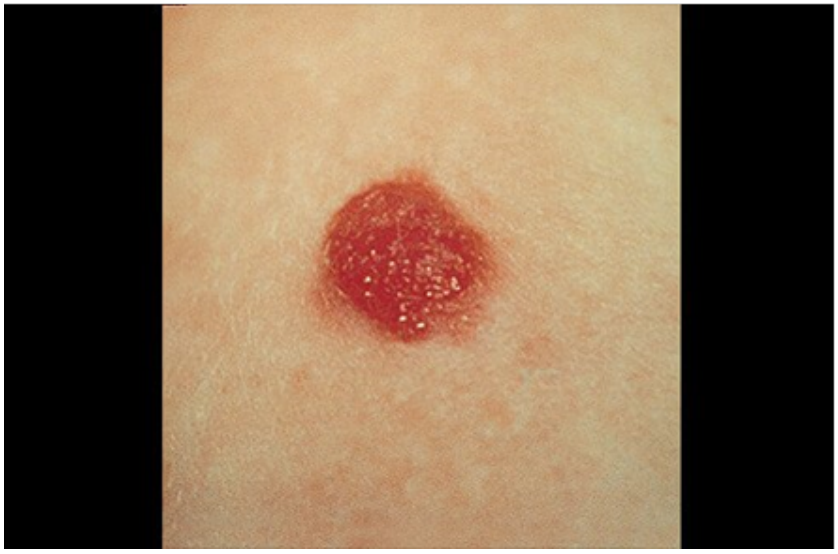


Fig 1.5: Spitz nevus.[22]

3. **Blue nevi** represent melanocytic nevi with a dermal distribution of cellularity and spindled cytomorphology under the microscope. Such lesions are typically heavily pigmented. Because of the presence of deep pigmentation within a refracting colloidal medium (namely, the skin), the brownish-black pigment present contributes a bluish cast to such lesions, thereby explaining the name. The optical effect that accounts for clinical blueness is known as the Tyndall phenomenon. The Tyndall phenomenon is not unique or exclusive to blue nevi. Any melanocytic nevus with deep pigmentation may present clinically with a blue hue. Not all blue nevi are blue. Some present with various shades of gray, brown, or black. The clinical appearance varies depending on the degree of clinical pigmentation. Indeed, some blue nevi may be wholly amelanotic. Because of the fact that the term blue nevus is not always reflective of the true clinical appearance of the lesion, some dermatopathologists name blue nevi based on the cellular morphology present. The terms spindle cell melanocytic nevus or dendritic melanocytic nevus represent morphological terms that refer to the spectrum of blue nevi. Despite their variability in

coloration, blue nevi are usually relatively small and reasonably symmetric, as typically is the case in benign lesions. Some blue nevi may be large and nodular with high cellularity under the microscope. Blue nevi typically occur on the distal extremities or scalp, but they can occur at anybody site.[7]

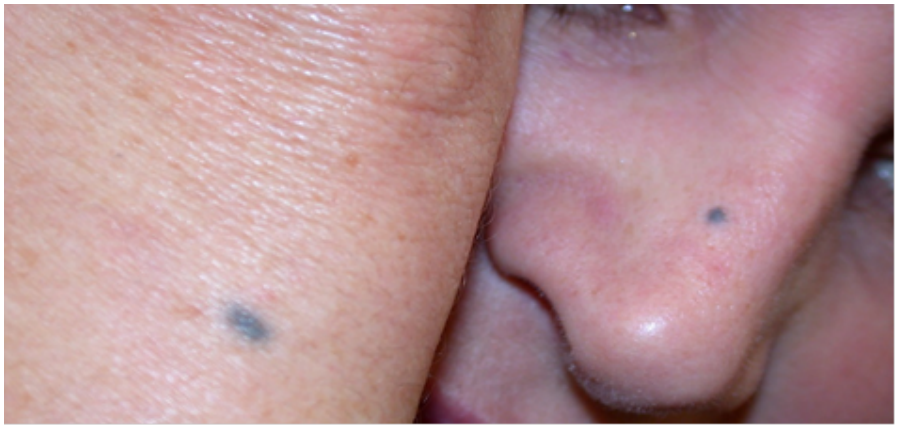


Fig 1.6: Blue nevi.[22]

1.2 Physical Examination

Physical examination involves, at a minimum, careful visual inspection of the lesion in question; in some instances, an examination of the entire skin surface should be performed. Importantly, document the dimensions and coloration of any lesion evaluated and record its exact location. A simple drawing of the lesion and the overall topography can be helpful. Many dermatologists use topographic charts to record the location of multiple lesions that are monitored from visit to visit. Some dermatologists enumerate individual lesions to facilitate follow-up. For some patients, especially those with multiple melanocytic nevi, photographic documentation

of lesions (including both distant views that demonstrate topography and close views that capture subtle features of a particular lesion) can be valuable. When examining melanocytic nevi, the physician should examine the scalp (possibly with the aid of a hair dryer), the palms, the soles, between the toes, and the genitalia.[7]

1.3 Treatment / Management

1.3.1Medical Care

Medical treatment is typically ineffective and inappropriate for the management of a benign neoplasm such as a melanocytic nevus.

1.3.2Surgical Care

Melanocytic nevi can be surgically removed for cosmetic considerations or because of concern regarding the biological potential of a lesion. Melanocytic nevi removed for cosmesis are often removed by tangential or shave excision. **Punch excision** can be used for relatively small lesions. **Large lesions** may require complete excision with sutured closure, even if known to be benign, because lesions exceeding 1 cm in diameter often are not amenable to the shave technique. A simple conservative excisional biopsy with a sutured closure is usually the most expeditious means to diagnosis if concern exists regarding the possibility of melanoma. If the lesion is found to be benign, then, ordinarily, no further treatment is required. Providing the pathologist with a complete excisional specimen affords him or her the best opportunity to make an accurate diagnosis because all available criteria (including low-magnification attributes such as size, circumscription, and symmetry) can be applied to the lesion. If incisional biopsy is obtained, information regarding the size and appearance of the lesion that underwent

biopsy should be forwarded to the interpreting pathologist or dermatopathologist.[23]

1.3.3 Differential Diagnosis:

- Atypical Mole (Clark Nevus or Dysplastic Nevus)
- Cafe Au Lait Spots
- Cockarde nevus
- Cutaneous Melanoma
- Nevi of Ota and Ito
- Nevus spilus.[24]

1.4 CO2 Laser

The CO_2 laser was one of the earliest of the laser systems to appear. It was first developed in 1964 by Patel and colleagues working Bell Labs in the USA. It was quickly recognized as an ideal surgical laser because of its high-water absorption, and many indications were pioneered by the late Professor Isaac Kaplan and other.[25] This is a gas laser, which emits a wavelength of 10,600 nm in the infrared portion of the spectrum. The radiation that is therefore produced is Invisible. The lasing medium is actually a combination of helium (60 – 80%), nitrogen (~ 25%) and CO 2 (~ 5%), and the external energy source is usually either an electrical charge, such as from an electrical outlet, or a radio frequency field.[26]

It is considered to be the most versatile and safe medical laser, because of its limited depth of penetration. As soft tissue is 80% water, and the CO 2 laser is highly absorbed by water, deep penetration is prevented, as long as there is intra and extracellular water to be vaporized. Ultimately, the depth of penetration is determined by the water content of the target tissue,

but is generally limited to 0.1 – 0.5 mm, with a lateral thermal damage of 0.5 mm.[27]

Optical scattering occurs in tissues when wavelengths of less than 1,000 nm are used. Because of the long wavelength produced by CO 2 lasers, there is little optical scattering, which helps to minimize lateral thermal damage ,It is also depend on tissue types so higher scattering observed in soft and fatty tissues.[28]

As described earlier, heat is delivered by the laser, and used to elevate the temperature of the target tissue. At temperatures over 100 ° C, vaporization of the intracellular water occurs, with conversion of the cellular components to smoke. $C0_2$ lasers can be made to produce emissions of up to several kilowatts (kW), however, most medical applications only require 10 – 20 W. They are highly efficient, with 10 – 15% of input power converted to laser emission.[29]

High power densities have often been required to maximize vaporization, and minimize tissue necrosis, which has led to the development of super-pulsed techniques. By pulsing the laser at extremely high rates, one can effectively slow down operating speed, while still maintaining high average power.[30]

Now, with the development of photonic band-gap fibers for guiding the light, the CO_2 laser can be easily adopted for endoscopic application. In fact, the flexible fibers can be coupled to conventional medical CO_2 lasers. As in the case with traditional delivery systems, the beam can easily be defocused by changing the distance from the target tissue, allowing for cutting, ablation and coagulation. Some of its other limitations include the inability to coagulate vessels 1.0 mm, and its propensity to create both a

plume and oxidized char. Once tissue has been charred, vaporization of underlying structures is impeded. Additionally, CO_2 laser is absorbed by blood, limiting its effectiveness to dry surgical fields.[31]

1.5 Laser Tissue interaction:

When laser energy is delivered to tissue four specific responses of this tissue to the laser light can occur as shown in (figure 1.2): Reflection, scattering, transmission or absorption. The extent of each depends on the wavelength of the laser, fluence and tissue types.[31,32]

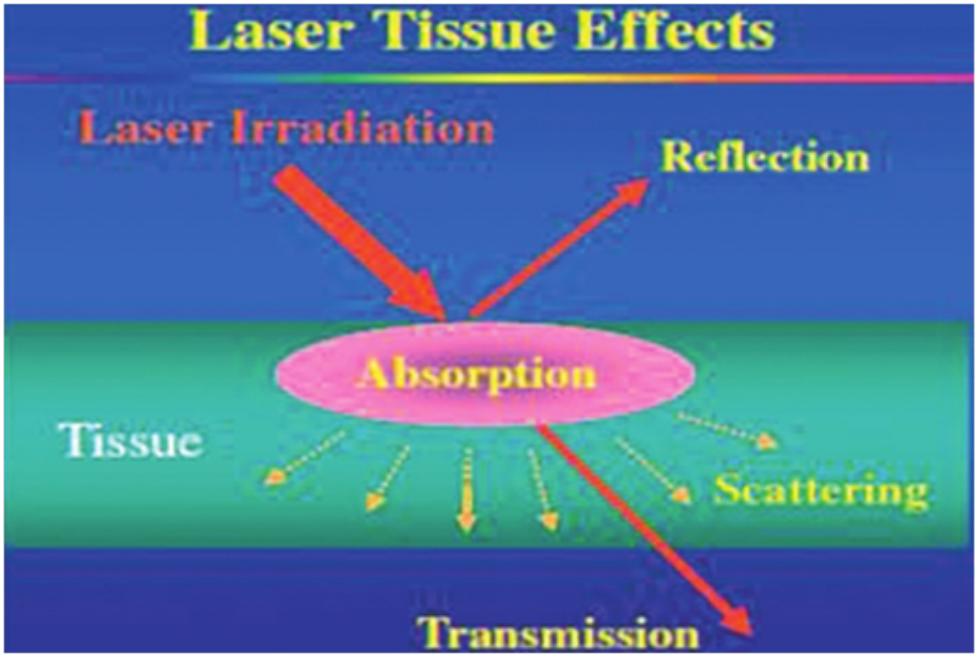


Figure 1.7: Action of tissue on laser light.[33]

Each tissue has specific absorption characteristics based on its composition and chromophore content. The principal chromophores present in mammalian tissue are: Hemoglobin, melanin, water and protein. Infrared

light is absorbed primarily by water, while visible and ultraviolet light are primarily absorbed by hemoglobin and melanin respectively .[32]

The chromophores of mammalian are shown in(figure1.8).[34]

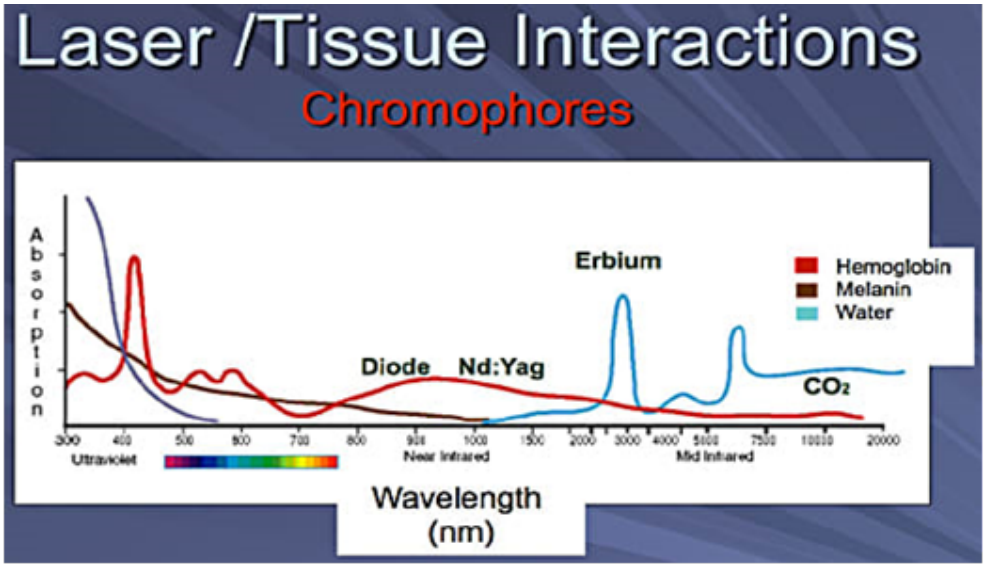


Figure 1.8: Laser tissue interaction.[34]

The most common interaction mechanisms, meaning the effect of the laser light on the tissue, for therapeutic and surgical applications may be divided into two main categories:

1. Wavelength dependent interaction mechanism,
2. Wavelength independent interaction mechanism.[35]

1.6 Mechanisms of laser in surgery and medicine

Lasers devices generate light energy in the form of a radiation of photons emitted from the laser medium, which usually gives the laser its name and determines the precise wavelength produced by the laser. For example, a ruby crystal gives the ruby laser its name, and emits at 694.3 nm: the CO_2 laser has carbon dioxide gas as its medium, and emits energy at

10,600 nm. Current medical lasers emit wavelengths from the ultraviolet to the mid-infrared portions of the spectrum. The medium is activated, or "pumped", with some form of energy, which is usually either light from a flashlamp, or electricity. The stimulated emission of photons occurs in the medium, which are then are amplified in the laser cavity, consisting of the medium bounded in the front and rear by mirrors, and emitted from the front mirror in a beam of unique light energy: the beam is more or less parallel, known as collimation; the photons are precisely in step temporally and spatially, known as phase; and the photons are absolutely identical, all one color, known as monochromaticity. The sum of these three components is coherence, and its coherence which gives a laser beam its uniquely high photon intensity, and allows a laser beam to be focused to very small spots. The main biological targets such as blood, melanin and water, absorb light energy very differently and have optimum absorption spectra depending on the wavelength of the incident photon energy as shown in(Fig 1.9). For visible light lasers and some near-infrared lasers, the main target chromophores are hemoglobin (consisting of oxy- and deoxy hemoglobin) and melanin, the former being found in vascular lesions and the latter in melanogenic lesions. For the CO_2 laser at 10,600 nm, the only chromophore is water, as is also the case with the Er: YAG laser.[29]

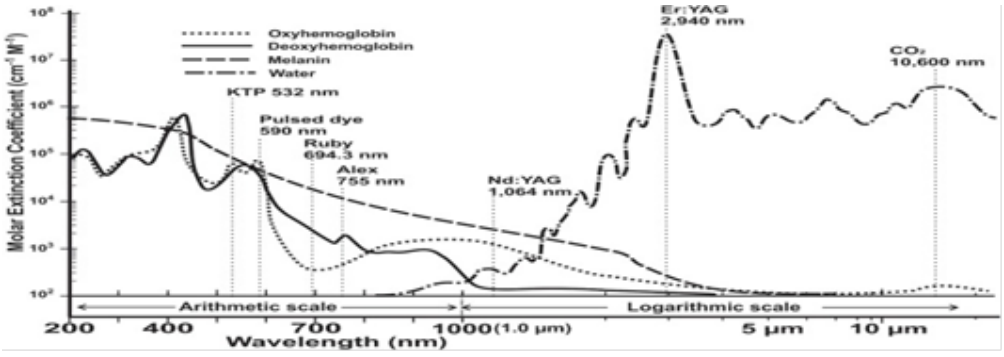


Fig. 1.9 : Absorption spectra of chromophores.[29]

When lasers are used in surgery and medicine, especially for the removal of any kind of pigment, the optimum approach is based on the principle put forward by Anderson and Parrish, known as 'selective photo thermolysis (SPTL)' which means using laser energy at high peak powers and short pulse widths to destroy the intended target alone while inflicting as little damage as possible on the surrounding tissue,[29]

Three key conditions must be met to achieve this:

1) The target must contain chromophores that absorb a specific laser wavelength;

2) These chromophores should not be found in the surrounding tissue; and

3) Minimization of damage to the surrounding tissue is achieved through the high peak powers and short pulse widths of the laser energy in the millisecond, microsecond or nanosecond domain, the latter being achieved by Q-switching the laser beam. The CO_2 laser does not meet the criteria for SPTL because its chromophore, water, exists uniformly in soft tissue, not just in the target zone. CO_2 laser surgery could therefore be described as non-tissue-selective since only the target tissue is ablated with minimal involvement of surrounding normal tissue. The advantage of this is that the CO_2 laser is not pigment-selective, and so both pigmented and non-pigmented skin lesions can be targeted with the CO_2 wavelength.[36]

1.7 Fundamentals of the CO_2 laser

The CO_2 laser was one of the earliest of the laser systems to appear. It was first developed in 1964 by Patel and colleagues working Bell Labs in the USA. It was quickly recognized as an ideal surgical laser because

of its high-water absorption, and many indications were pioneered by the late Professor Isaac Kaplan and others.[26] If a focusing lens is fitted to the handpiece as shown in Figure 1.10a, it will bring the collimated beam to a fine spot, concentrating all the photon energy of the beam into that area. The CO_2 laser operates in the invisible infrared waveband, and an aiming beam is required to see where the treatment beam will impact tissue more recently from a visible red diode laser. The clinician can use the aiming beam to position the CO_2 laser beam at the point of focus on the tissue. Focusing the laser produces an extremely high irradiance or power density, for example 3 W of CO_2 laser energy focused to a 100 μm spot will have an irradiance of over 38 kW/cm^2. This is sufficient for instant vaporization and ablation and if the handpiece is moved linearly, the target tissue will be cleanly incised, thus giving the CO_2 laser its reputation as a laser scalpel. As an important point regarding the safety of the clinician, patient and all in the operating room when a CO_2 laser is being used, this explosive reaction disperses a laser plume into the air consisting of steam and particulate matter, so a dedicated laser smoke evacuator is always recommended when using the CO_2 laser.[25]

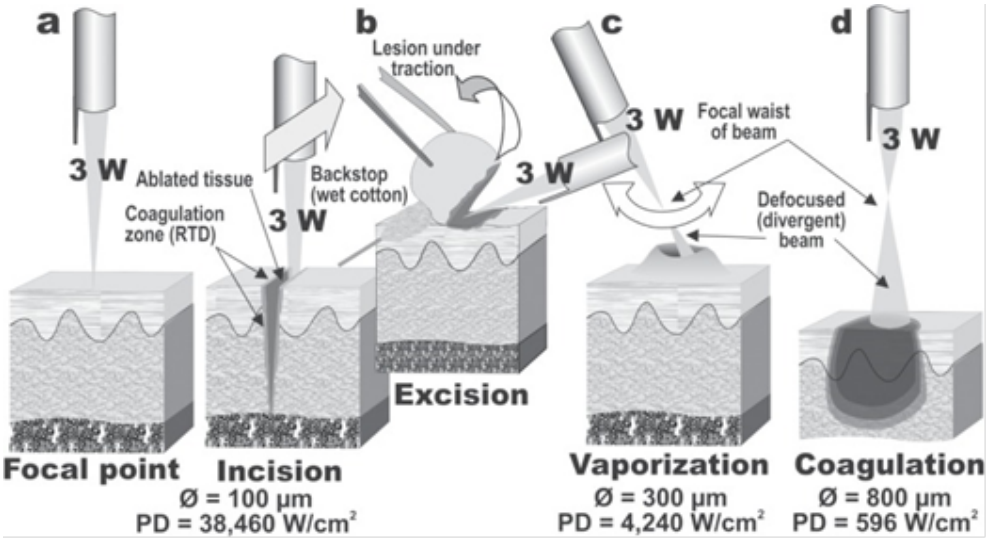


Fig. 1.10: The biological effect of the laser beam through moving the handpiece towards or away from the focal point of the beam.[29]

The irradiance of a laser beam decreases in an inverse square ratio to the beam diameter, as the area of a laser beam is calculated using the formula $\pi \cdot r^2$, where π (Greek letter pi) is the constant 3.142, and *r* is the radius, or one-half of the diameter, of the spot size. Accordingly, simply by moving the handpiece away from the tissue and defocusing the beam, the clinician can make dramatic changes in the irradiance and instantly change from incision/excision mode (Figure 1.10b) to bulk vaporization (Figure 1.10c) to coagulation (Figure 1.10d) while leaving the output power of the laser unaltered.[29]

When we consider the theoretical side of the CO_2 energy/tissue reaction, the absorption coefficient of the 10,600 nm CO_2 laser in water is around 5×10^2 cm^{-1} (reciprocal: 0.2×10^{-2} cm), and the theoretical depth of penetration is therefore from 2×10^{-5} m (20 µm). In other words, this means that when the carbon dioxide laser is used on biological tissue *in vivo*, all of

the incident energy is absorbed in the tissue water down to a specific depth, thus making the CO_2 laser a comparatively "safe" system, since the water in tissue quenches the beam and prevents deeper tissue damage. The actual depth reached and the damage volume (irradiated area × depth) will depend primarily on the irradiance and secondarily on the energy density, the latter being calculated in J/cm^2 as irradiance (W/cm^2) × exposure time (s).[36]

The boiling point of water, which comprises about 70% of soft tissue, is 100°C. When the aqueous component of tissue is raised to 100°C by the incident CO_2 laser energy, it is immediately converted to water vapor and gasifies, the volume expands, and as a result the skin (or other target) with its connective tissue, cells, cell membranes and intracellular organelles, is removed by fragmentation, vaporization, and ablation. There is always some degree of residual thermal damage at the borders of a CO_2 laser ablated zone caused by decreasing levels of photothermal reaction in the tissue. Thermocoagulation occurs in this zone, at least to some extent, and small blood vessels are cauterized together with the surrounding tissue, achieving hemostasis and therefore a dry field. Larger blood vessels may not be cautery*zed, however, and hemorrhage will occur. In this case the quickest method is to use electrocautery to control the bleeding, because, even with defocusing, all of the CO_2 energy will be absorbed in the water at the surface of the blood and will not reach the cut wall of transected vessel to coagulate it. As has been pointed out by Ohshiro in his laser apple concept, all of the incident energy is not quenched in ablation of the tissue, and there is a series of temperature-dependent bioreactions in tissue associated with each laser shot which become less destructive as decreasing levels of the photons penetrate into the tissue from the point of impact.[37]

At the very perimeter of the beam, there is insufficient photon intensity to raise the temperature of the tissue, and this is the thermal photoactivation zone, al*so referred to as the simultaneous LLLT zone, occurring concomitantly with the photo destructive effects.[38]

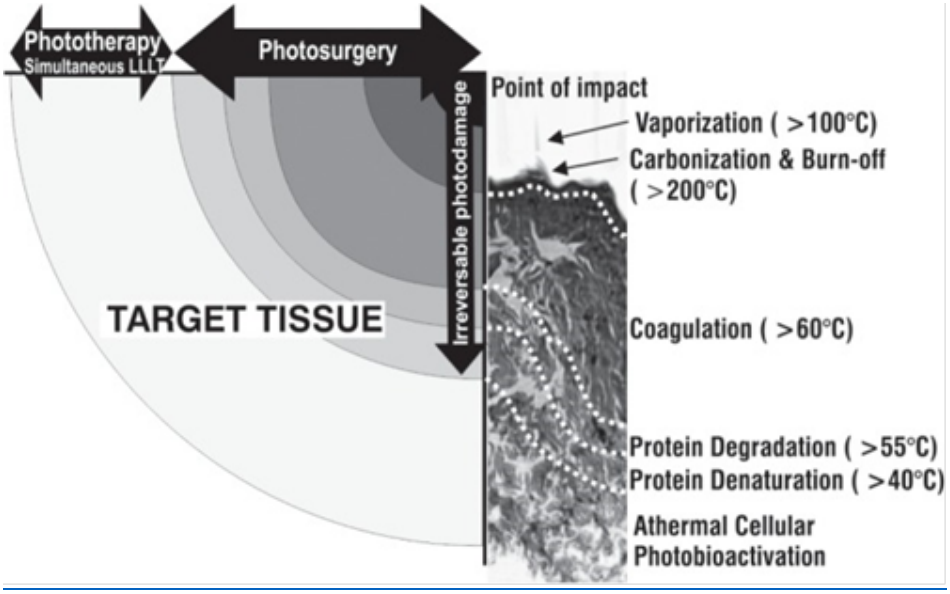


Fig. 1.11:Temperature-dependent bioreaction zones from a surgical CO_2 laser impact illustrated schematically with a typical histological pattern shown alongside the schematic. [29]

The CO_2 laser has a number of temporal beam modes, each of which induces a different reaction in the tissue. The simplest mode is continuous wave (CW), in which the laser beam is emitted, operated for a specific time, and turned off, producing a pattern as in (Figure 1.12a). CW beam can be mechanically or electronically gated or chopped to provide a series of square waveforms as shown in (Figure 1.12b). The output power is measured in watts and the irradiation time in seconds. More recent CO_2 lasers

have trains of very short high peak power pulses with a very long interpulse interval: this is referred to as quasi-CW, because the tissue ‘sees’ the average power, measured in W, although the peak power of each pulse might be in megawatts (MW) (Figure 1.12c). Quasi-CW has the advantage of allowing cleaner incision or bulk vaporization with less charring because each pulse is shorter than the thermal relaxation time of the target tissue (1 ms in the case of skin), so that optimum ablation is achieved with minimum heat build-up in the adjacent tissue. Quasi-CW can also be referred to as “super pulsing” or “ultra-pulsing”, depending on the peak power, pulse width and interpulse interval.[39]

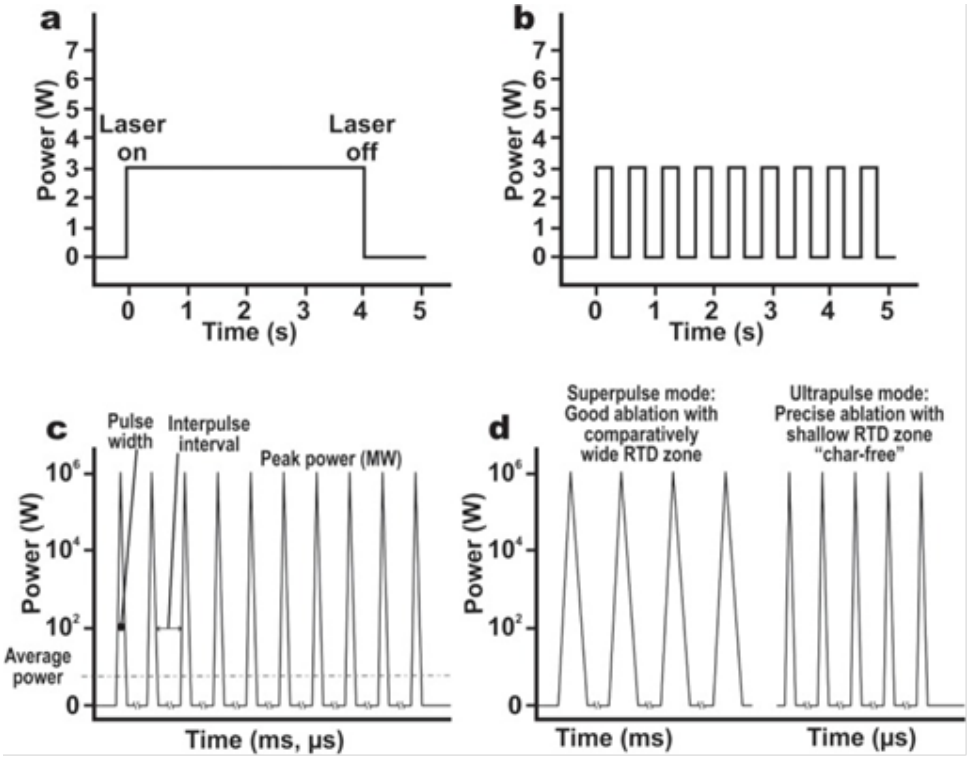


Fig. 1.12: Temporal mode of a laser beam illustrated schematically.[29]

1.8 Safety with the CO_2 Laser

Safety with lasers is paramount for the well-being of the patient and all in the operating room. This is particularly so for the CO_2 laser given its range of use in deliberate destructive indications. As the eye is the most vulnerable target for optical energy, eye protection is mandatory for all in the treatment room. However clear glass or plastic will stop the CO_2 beam completely: this is why normal quartz fibers cannot be used as a beam transmission system for the CO_2. Ordinary prescription lenses can be used, but dedicated glasses or goggles incorporating lateral shields are always recommended. The 10,600 nm beam is also a "safe" wavelength for the eye, or at least the retina, because all of the energy will be absorbed in the water in the cornea at the front of the eye. This makes the CO_2 laser marginally safer than the visible and near

infrared lasers, because the latter will pass straight through the cornea, be focused by the lens, and severely damage the macula/fovea complex in the retina. Protecting the patient is also a priority, and for the CO_2 laser this means draping the skin around the target area with damp drapes or gauze, and keeping these materials damp throughout the procedure because the CO_2 energy is absorbed in water. If a beam of CO_2 energy strikes dry gauze or cotton, the potential for starting a fire is very high. When using the CO_2 laser to excise tissue held under traction, a backstop of some kind should be employed, either a dampened wooded tongue depressor or a damp cotton bud depending on the site. Finally, as already discussed, the laser plume created by the CO_2 laser can contain potentially harmful substances such as viable viral particles, so the use of a dedicated smoke evacuator is extremely important. The use of masks alone will not suffice.[,37;39,40]

Aim of the study:

To assess the response of melanocytic nevi to fractional CO_2 laser treatment.

Chapter Two

Patients,Materials And Methods

Introduction:

In this chapter, patient's selection, sample preparation (patients) and procedures are mentioned. Materials including laser system and its accessories used in this work are discussed. Evaluation criteria and measurement will be illustrated. Also, laser safety measures will be discussed.

2.1Patient's selection:

Examination of all the patients were done by two dermatologists to diagnosis the melanocytic nevi and their suitability for fractional co_2 laser therapy .

2.1.1: Inclusion criteria:

1. Acquired melanocytic nevi.
2. Blue nevi.
3. Spitz nevi

2.1.2. Exclusion criteria:

1- Atypical looking nevi with features such as: asymmetry, bleeding, infection, irregular pigmentation and border, or personal or family history of melanoma.
2- Patients age less than 15 years.
3- Pregnant female in spite of safety in pregnancy
4- History of scar and keloid tendency.
5- Congenital melanocytic nevi.

2.2 Patients:

Seventeenth patients with melanocytic nevi were enrolled in this case series study,11 men and 6 women (aged from 18 to 56 years with average age of 32.3) with total number of lesions 35, to evaluate the response

of melanocytic nevi to treat by fractional CO_2 laser, in treatment of nevi in Iraqi patients. As shown in Table (2.1).

Samples included in this study were selected from patients presented to a private dermatologic clinic and get treatment in a private dermatologic center, during the period from September 2021 to December 2021

Recruited patients had melanocytic nevi that were present for more than 2 years and who had little benefit from other conventional therapy or not treated at all.

Diagnosis of melanocytic nevi was based on history and clinical examination, and consensus of college dermatologist.

2.3 Materials:

2.3.1 Laser System:

The laser employed in the present work is fractional CO2 laser system, (KES MED-870+fractional CO2 Laser Therapy System manufactured in China by Beijing KES Biology Technology Co.Ltd.) using micro pulsed mode

Its clinical data is the following, as shown in the catalogue:

Wavelength	10600nm Laser
Power	40W U.S. RF Metal Tube
Spot Size	0.12mm and 1.25mm (adjustable(
Spot Density	Up to 102400 dot
Scan Size	Up to 20×20mm
Scan Shapes	Square; Rectangle; Circle; Triangle; Rhombus;
Scan Modes	Standard; Random; Scatter
Aiming Beam	5mW red diode laser, 635nm, adjustable

Beam Delivery	360° Rotation Articulated 7 Joint Arm
Operating System	Fractional co_2
Cooling System	Air Cooling, self-contained; closed cycle
Display	10.4 Inch True Color LCD Touch Screen
Voltage	220V±10% 50/60Hz, 110V±10% 50/60Hz.
Dimension	52×38×117cm (W*D*H) Net

Fig 2.1:Fractional CO2 laser system

2.3.2 Laser Accessories:

- Articulated arm
- Power cable
- Footswitch
- Interlock key
- Fuse
- Googles for doctor
- Blocked eyepiece for patient

2.3.3 Laser parameters:

Energy: 30 mj,

Pulse duration: 1ms,

Distance: 0.2mm,

Spot size:0.12mm

Fluence 26.5 mj/mm^2 ,scan size: variable according to the size of nevi from 1-10mm, Wavelength:10600 nm

Fig. 2.2 Display panel of the system

2.4 Procedure:

This includes preparation of patient before, and after laser treatment and the relative and absolute contraindication to laser treatment.

Before laser sessions:

1. Complete history from patient was taken.
2. The site of lesions is identified.
3. Age of patients.
4. Color of lesions.
5. The duration of lesions.
6. History of previous treatments modalities.
7. Complete drug history (steroid, Isotretinoin, anticoagulant).
8. The field of procedure thoroughly cleaned and disinfected and gloves were warned.
9. Photograph by digital camera had been taken to each patient before any session and after completion the study for comparing.
10. Apply a local anesthetic injection of lidocaine 2% Made in England gsk company.

The treatment sessions were 1-3 sessions at 4 weeks interval between sessions.

During, and post procedure:

The laser hand piece is positioned in a perpendicular manner to the lesion and spot size adjusted and changed according to size of nevi, shots slowly till all the lesions in area hated by laser beam. The laser causes instant charring to the lesion that should be removed after each pass by cotton gauze. Between 3- 20 passes of ablation were done in depth 100 μm ablation for each pass and 120 μm thermal ablation width for each application, but for

large protruded nevi the number of passes were may be more than 20 passes. If we were detecting structure of the nevus the laser was performed at two months intervals until complete clearance was obtained. This meant that up to 10-20 passes were performed into the reticular dermis for removal of as much of the pigment as possible. During laser application, residual tissues on the wound surface were removed with a gauze cotton sponge.

Healing cream (antibiotic, fucidic acid 2% - leo company + soothing, bepanthen5%-Bayer company) is applied locally twice daily for one weeks. The patients were asked to avoid sun exposure by using wide spectrum sun-blocking cream.

2.5 Evaluation Criteria: -

a. Clinical assessment.

b. Subjective method.

Pain during procedure Grade 0 = no pain

Grade I = mild pain

Grade II = moderate pain

Grade III = sever pain

2.6 Safety measures (Class IV):

In the present work, the laser employed was Class IV laser which include any continuous-wave device with energy outputs above 500mw. These lasers can cause damage with direct intra-beam exposure and from specular or diffuse reflections. Additional performance requirements and safety measures must be taken to provide protection from the energy emissions of these lasers. Some of these precautions include.

1. All personnel were asked to wear protective glasses appropriate to the procedure to eliminate the risk of eye damage. These glasses designed with special wavelength and optical density for the CO2 laser system, with wavelength 10600 nm.
2. In addition to protective glasses, the eyes of the patient were covered with mops of wet cotton.
3. To avoid the reflection hazard avoid placing reflection materials such as glass, metals and polished plastic in the laser room.
4. Explosion hazard, avoid using flammable or fume emitting substances e.g. (ether, iodine solution, and alcohol in operative field

Table 2.1 Age of patients, gender, site of lesion, number of lesions, follows up duration, and number of sessions.

Patient	Age	Gender	Position and number of lesions	Number of sessions	Follow up duration/months
1	56	F	2 foreheads	1	2
2	43	F	1 cheek	1	2
3	27	M	1 cheek	1	2
4	25	M	1 cheek,1 forehead	1	2
5	28	M	3 cheek,1 forehead	2	3
6	50	F	1 cheek	1	2
7	28	M	3cheek	1	2
8	25	M	3 cheek,4 forehead,1 nose	2	4
9	41	M	1 cheek,1 forehead	1	2
10	18	M	1 nose	1	2
11	21	M	1cheek	1	2
12	35	F	1 upper lip	1	2
13	36	F	1 forearm	1	2
14	37	F	1 upper lip	2	3
15	38	M	1upper lip ,1cheek	3	4
16	23	M	1 neck	1	2
17	28	M	1 cheek,1 forehead	1	2

Chapter Three

Results,
Discussion, Conclusions

3.1. Results

The patients consisted of 11 males (65%) and 6 females (35%) between 18 and 56 mean 32.35 years. All patients had Fitzpatrick's skin types II -IV. Skin types were classified as Type II (in 5, 29%), Type III (in 11, 65%), and type IV (1,6%) Most of the patients were in skin type III. Diameter ranged from 2 to 10 mm (mean 4.55 mm)

Localization of nevi: cheeks in 18 (51 %), were the most commonly involved followed by forehead 10 (29%), lips 3(8%), nose2 (6%); forearm 1(3%), and neck 1(3%)

3.1.2Clinical inspection

The total lesions were 35 melanocytic nevi, 25 (71%) nevi were treated with 1 session followed by 8 (23%) nevi were treated with 2 sessions and 2 (6%) nevi required for 3sessions. All treated nevi showed complete clinical pigmentation clearance at the end of the last session.

Three patients treated with 1 session were shown in Figure 3.1. Nevi were evaluated from the time of operation to re-epithelization and the duration period of post-treatment erythema. Transient crusting and erythema were observed immediately after treatment. In general, re-epithelization occurred within two weeks post treatment.

Post treatment erythema lasted for 3 to 4 weeks in 90% of the treated patients, then subside gradually without treatment or with local corticosteroids.

Erythema lasted for 12 weeks in 10% of the treated melanocytic nevi then subsided gradually without treatment. Figure3.2 shows that clinical

photography images of one patient before and after one month of treatment with erythema.

3.3Satisfaction

According to researcher (investigator) after one month from treatment, 60% of the patients were rated as excellent, 20%patients were rated as good, 12% patients were fair, and 8% were poor with respect to response to fractional CO_2 laser In contrast, for satisfaction of patients at one month were 59% rated treatment as excellent, 32 % good, 5% fair, and just 3% of the patients rated treatment as poor, as shown in Figure-3.4.

Satisfaction	Percentage
Excellent	75-100 %
Good	50-75 %
Fair	25- 50 %
Poor	0-25 %

3.4Side effects

Fortunately, most side effect were mild and transient were seen in 8 patients (47 %), at the 3 months follow-up, hyperpigmentation was in 3 (17 %), atrophic and hypertrophic scars in 2 (12%), recurrences in 2 (12%), and hypopigmentation in 1 (6%). As shown in Figure 3.3ABC

For the pain felt by the patients during and after the procedure was mild in 70% of the cases, moderate in 30% of patients, while no one out of pain nor sever pain (table 3.1).

Table 3.1 Patient`s Pain Results

Type of pain	Grade	No. of patient	Percentage
No	0	0	0
Mild	I	12	70%
Moderate	II	5	30%
Sever	III	0	0
Total		17	100%

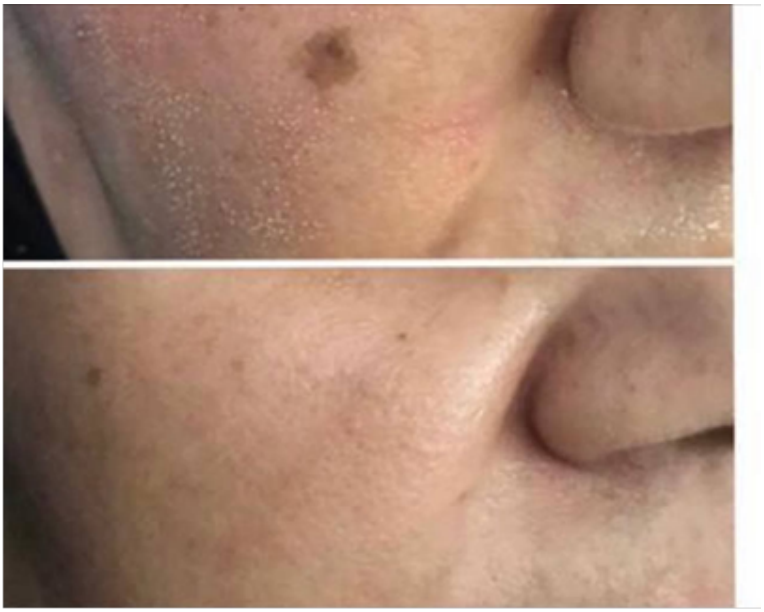


Fig 3.1A: Forty-three years female patients before and after nevus removal in the right check in one session (seen after one month).

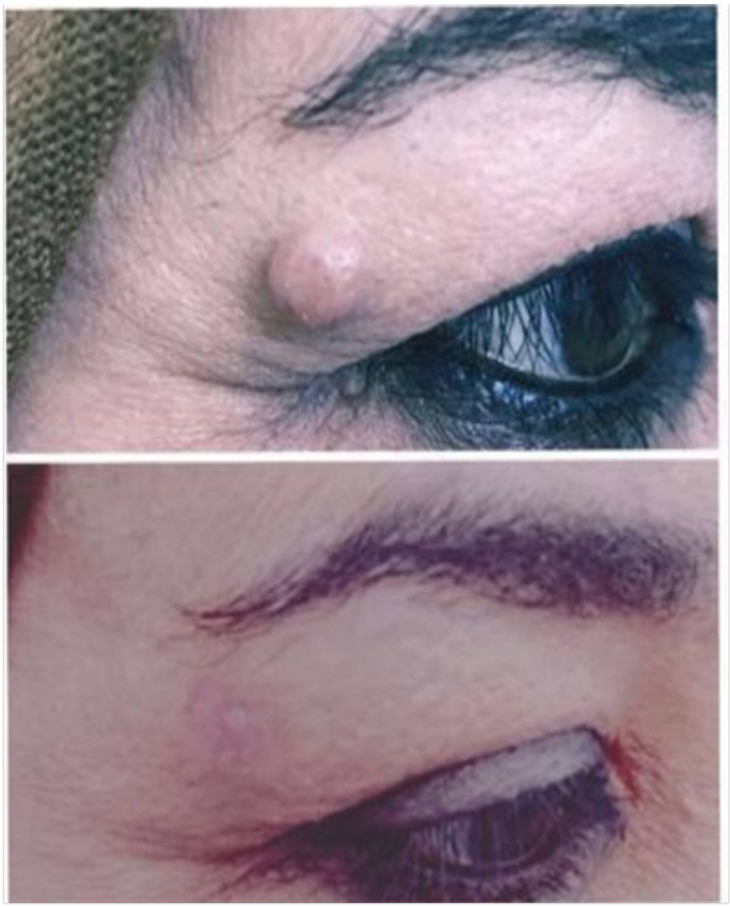


Fig 3.1 B: Fifty years female patients before and after nevus removal in the right eyelid in one session (seen after one month) and the result improved more after two months.

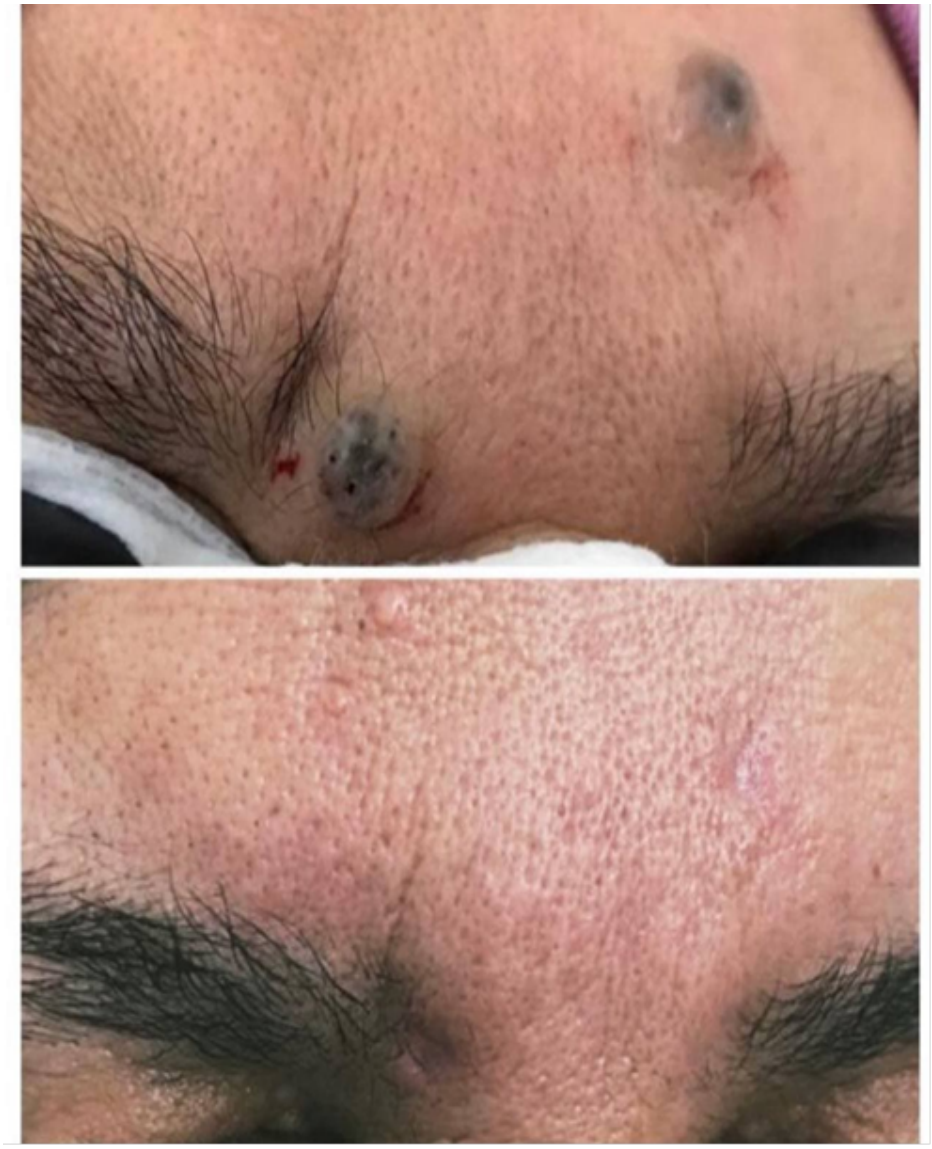


Fig 3.1C: Fifty-six years female before and after removal of two nevi in forehead in one session (seen after one month).

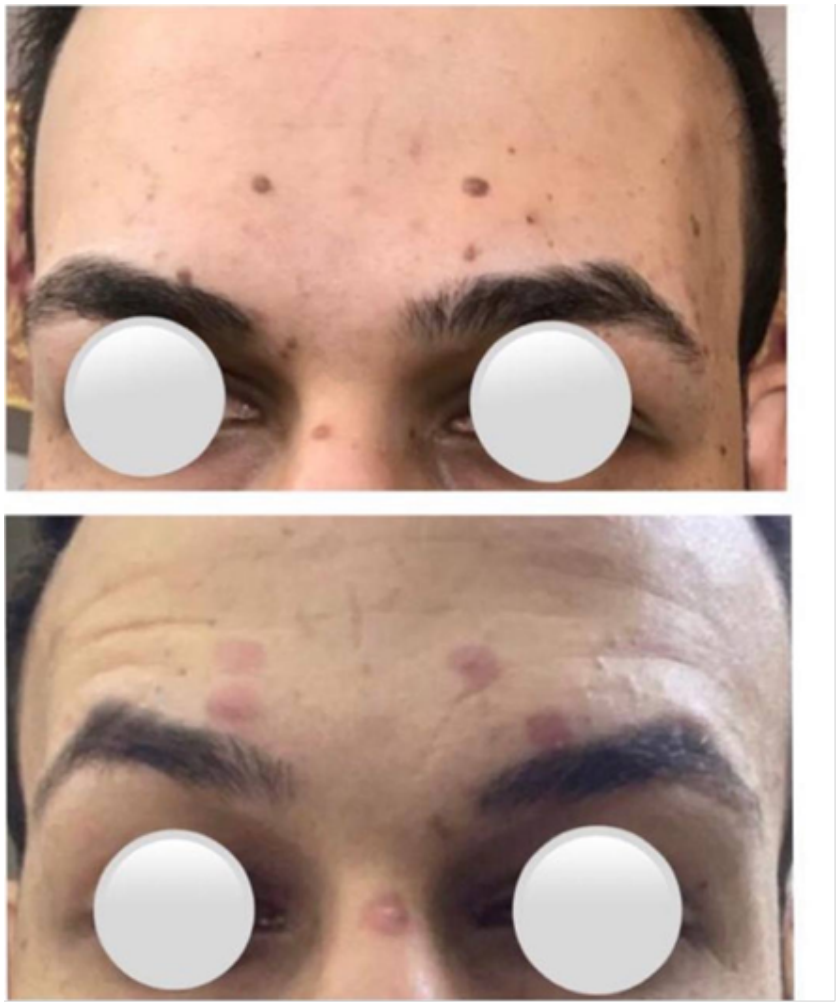


Fig 3.2: Twenty-five years male patient with erythema after 30 days from fractional co2 laser as transient side effect that treated by topical hydrocortisone 2.5 % for 3 weeks and it decrease gradually and disappear completely after 2 months.

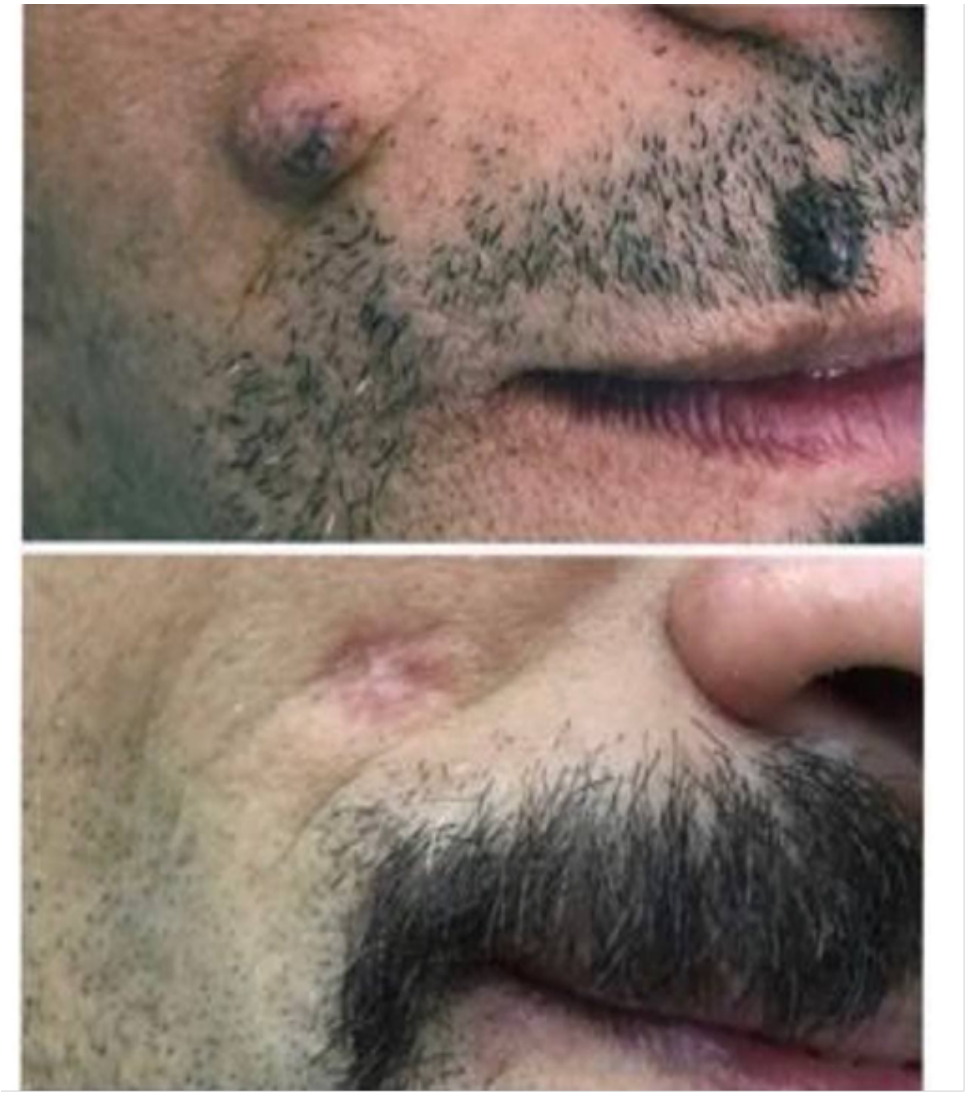


Fig 3.3A: Thirty-eight years male patient with two nevi removal one is completely disappear without complication, other one leave atrophic scar after one month which moderately decrease after two months by using micro needling treatment.

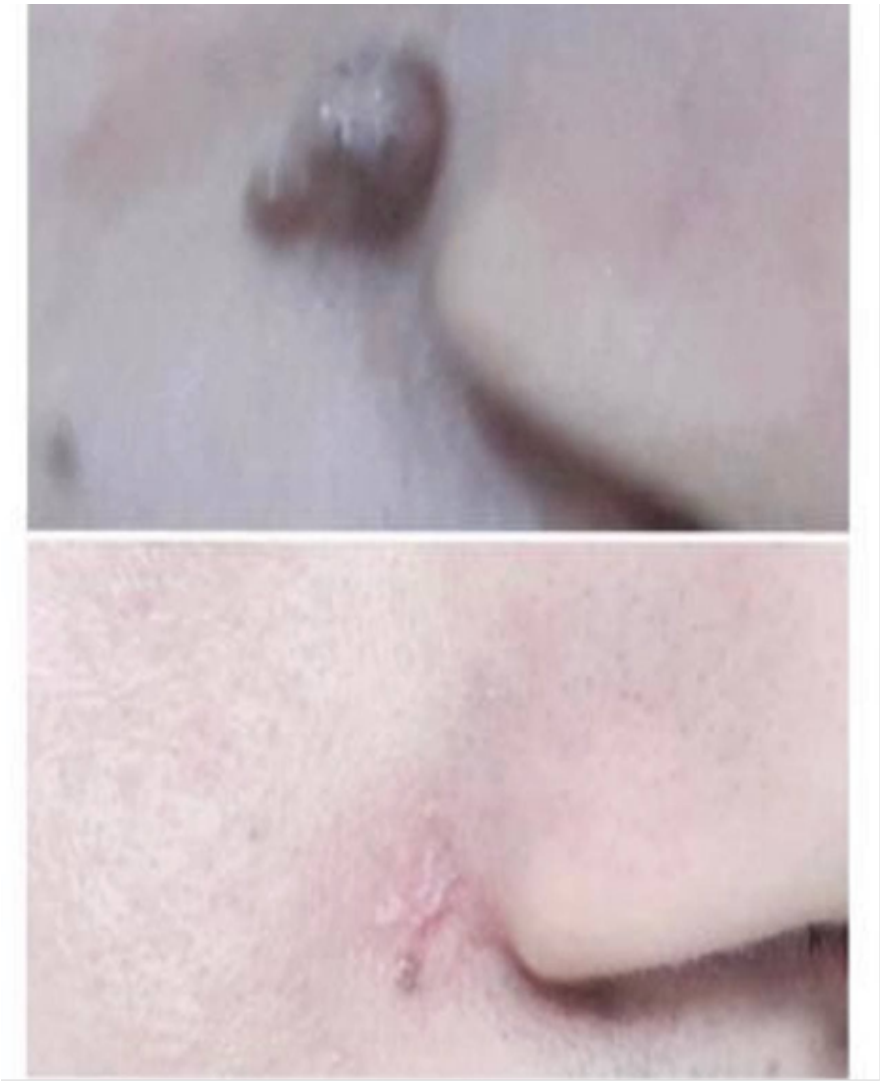


Fig 3.3 B: Twenty-one years male patients develop hypertrophic scar after one month, improved after fractional CO2 resurfacing.

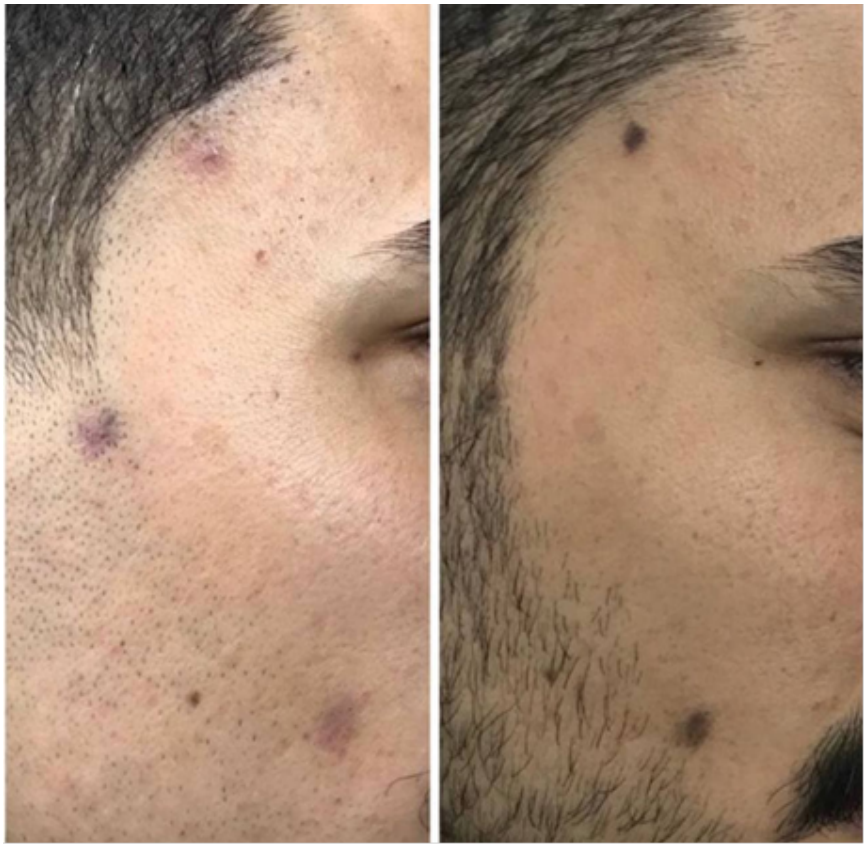


Fig. 3.3 C: Twenty eight years male patient with hyperpigmentation that last3 months and disapear after treatment by topical hydroquinone cream 4 % with hydrocortisone cream 1% for one month .

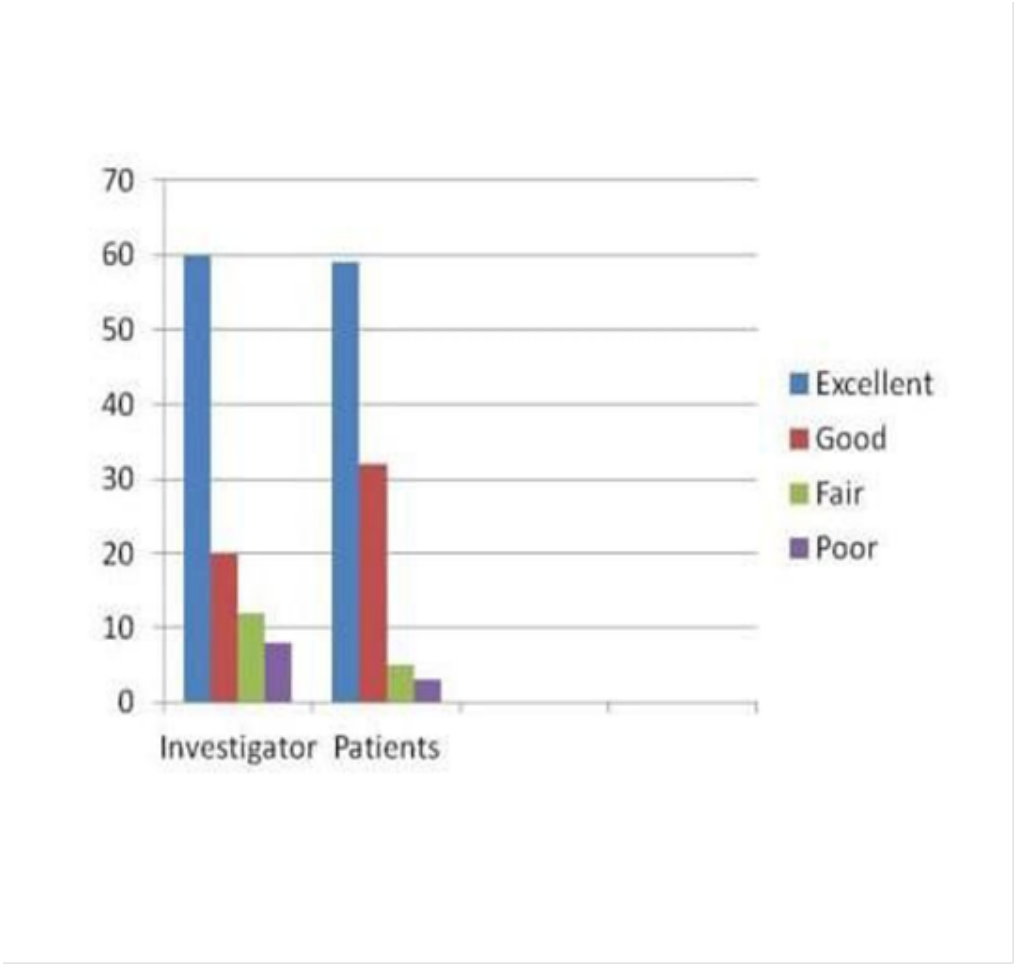


Fig 3.4: Satisfaction of investigator and patients

3.2Discussion

There are many treatment options for melanocytic nevi, these include dermabrasions, cryosurgery, surgical excision, and laser treatment.[41] Surgical excision is the most common option, but it leads to post-operative linear scarring. Many types of lasers have been used, including either pigment or ablative lasers or a combination of the two groups of lasers.[41,42]

CO_2 laser has been widely used for skin rejuvenation in a cosmetic procedure. This laser uses a wavelength of 10.600 nm and penetrates into the epidermis. Water is the main chromophore that absorb 10.600nm CO_2 laser in human tissues, and induce the tissue evaporation of the tissue. The main modes of CO_2 laser action are ablation, fragmentation, and vaporization. Fresh ulcers had occurred after ablational fractional CO_2 laser, followed by the healing of the primary wound. After that process, re-epithelization is generally completed in around four weeks.[43-45]

In cases of excessive irradiationl, laser treatment can be associated with scarring. We started laser energy between 25 to 30 mjoule and fluence 70 mjoule/cm2 at first session followed by decreasing energy (20–25 mjoule). Fortunately, most side effect were mild , transient were seen in 8 patients (47 %), at the 3 months follow-up, hyperpigmentation was in 3 (17 %), atrophic and hypertrophic scars in 2 (12%), recurrences in 2 (12%), and hypopigmentation in 1 (6%).

While in a study carried by Köse O. the hyperpigmentation was found in 13 (2%) of the patients, this because Köse O started with a CO_2 laser energy of between 2 and 5 mjoule and flounce 20 mjoule/cm2 at the first

session. Then energy (1–3 mjoule) was decreased gradually if the nevus cell structure was detected in the ajmn. At each pulse, ablation of the epidermis was performed with approximately 25 to 50 µm. In generally, 100–200 µm ablation will not cause scarring, and skin healing occurs in a short time, indicating that ablations were performed stratum Basale on the epidermis. He can conclude in general that the slight ablation done by CO2 fractional laser can achieve good results without any side effects.[46]

Ozaki et al. performed CO2 fractional laer for facial compound and dermal melanocytic nevus with 5- and 10-mm diameter. CO2 fractional laser was found effective without any side effects for nevus.[44]

In the current study, 71% of nevi were treated with one session, which is in agreement with that found by Kim et al. used the Q switched Nd:YAG laser (1064 nm) to remove melanocytic nevi in 2064 patients .They concluded that about 70% of the nevi were removed in one session.[47]

Fractional CO2 lasers focus the same amount of energy into the skin in microscopic thermal zones(spot size) as small as 120 µm , which create fluences of nearly 100 times those of traditional(surgical) lasers. This results in tissue ablation past the epidermis and through the papillary dermis into the reticular dermis in these narrow zones while leaving the surrounding epidermis intact.[48]

Conclusions:

In conclusion, to our knowledge, this study is the first study that use fractional CO2 laser in removing melanocytic nevi in Iraqi patient. In this study, we report that fractional CO2 laser is regarded as one of the best modality for treatment of acquired melanocytic nevi due to its simple post-treatment care, fast recovery, lack of bleeding, and no serious side effects. It can be suggested that multiple nevi can be treated in a short time with fractional CO2 lasers, and this method showed a good cosmetic outcome with a high degree of satisfaction as reported by both the physicians and the patients.

Recommendations

- Further studies ,more number of patients with longer time of follow up.
- Recommended to do comparative study of fractional CO2 with fractional Erbium-YAG laser.

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ملخص البحث

الخلفية:

وحمات(شامات) الخلايا الصباغيه المكتسبه هي مرض جلدي شائع يعتبره كثير من الناس شئ بشع ومطلوب ازالته ؛ وقد تم استخدام العديد من الاساليب ولكن مع اثار ضاره.

الاهداف:

يهدف هذا العمل لتقييم استجابة الوحمات (الشامات) الصباغيه لليزر ثناني اكسيد الكربون التجزيئي كطريقه جديده للعلاج.

الطرق:

تحاول هذه الدراسه استخدام ليزر ثناني اكسيد الكربون التجزيئي لازالة 35 شامه في 17 مريض ومتابعتها لفترة من 3-4 شهور.

النتائج:

نتائج35 شامة ، 25(71%) شامه تم ازالتها بجلسه واحده ، 8 (23%) ، 2 (6%) شامات تطلب ازالتها اثنان وثلاث جلسات على التوالي ، اظهرت جميع الشامات المعالجه اختفاء التصبغ الكلينكي الكامل نهاية اخر جلسه. **التأثير الجانبي**: لحسن الحظ كانت معظم الاثار الجانبيه خفيفه ومؤقته في 8 مرضى (47%)، من خلال المتابعه في 3 اشهرحيث كانت الحالات هي زيادة التصبغ في 3 مرضى(17%) ، الندب الضموريه والمتضخمه (12%) ، ورجوع الشامات في 2(12%)، وفقدان الصبغه قي 1(6%).

هذه التأثيرات الجانبيه عولجت كلا حسب الحاله.

الاستنتاجات:

يعطي ليزر ثناني اكسيد الكربون التجزيئي نتائج تجميليه ممتازه ؛ مما ادى الى ارضاء المرضى بدرجه عاليه ؛ ويمكن اجراؤه بسهوله.

وزارة التعليم العالي والبحث العلمي

جامعة بغداد

معهد الليزر للدراسات العليا

استجابة الشامات الصباغية للعلاج بليزر ثنائي اكسيد الكاربون

دراسة مقدمه الى معهد الليزر للدراسات العليا / جامعة بغداد
كجزء من متطلبات دراسة الدبلوم العالي لتطبيقات الليزر
في الطب / الجلدية

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خالد ابراهيم جاسم

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استاذ مساعد في كلية طب تكريت

1443هـ 2022م