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Original Article

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Correlation of Outcome of Scars with their Pathogenesis Following Er YAG Laser

Nishad K[1], Ravi Kumar Chittoria[2], Padmalakshmi Bharathi Mohan[3], Imran Pathan[4], Shijina K[5], Neljo Thomas[6]

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Abstract

The scar is a sequela of trauma, burns or surgery. The abnormal scar can influence an individual's well beingness, so the prevention as well as treatment of scar is important. The concern for scar makes a good percentage of consultations in any Plastic Surgery out-patient department. There are many methods to manage a scar, laser Therapy using Er YAG laser is one method of management of scars that is widely used in western countries. In India, Er YAG laser therapy is relatively a recent addition, hence the data available on scar management using the same is few. This study was conducted in a Tertiary care Institute in South India under the Department of plastic surgery, aim of which was to find out if there is any relation of etiopathogenesis of scars with their outcome following Er YAG Laser Therapy or in other words to find out how different types of scars responds to the Er YAG Laser therapy. There are studies which evaluated the response of laser therapy based on the time of starting the therapy and location of the scar but the data based on the etiology is few. A Total of 73 scars were included in the study with a follow up period of 6 months.

Keywords: Scar management; Scar Etiology; Scar Etiopathogenesis; Er-YAG Laser.

Introduction

The concern of scar is a very common problem for which patients seek consultation from any plastic surgeon, either it is for preventing abnormal scar formation or for the management of abnormal scars. The symptoms of scars that brings a patient to any medical care facility is, disfigurement, pain, pruritis and disablement due to it in the form of restricted movement of joints, neck, eyelids, lips, finger etc. Scars can influence the symptoms and signs based to their location, color, consistency, or size (height). There are many methods known for the prevention as well as management of scars. Though the scars are not completely avoidable, they can certainly be made better with meticulous initial management. In this study we give more importance to the management of scars and scar modulation. There is no method which can be cited as the single best method in managing the scars. Some of the commonly used methods are scar massage, silicone gel or silicone sheet application pressure garments, medications for local application containing allantoin, heparin etc.,

intralesional steroids, surgical scar revision, and Laser Therapy. Laser therapy can be low-level laser therapy or high-level laser therapy. In this study we are more focused on High-level laser therapy. Initial high-level lasers in use were CO_2 and Pulsed-dye laser, which were known for many adverse effects. Due to which there was always a search for newer lasers, equally effective, lesser adverse effects as well as lesser downtime to achieve the desired clinical effects like changes in size (height), consistency, color (pigmentation) or vascularity.

Though the Er-YAG laser was used in western countries for many years, in India Er YAG is a relatively newer addition to the armamentarium in scar management, hence the data of the efficacy of the Er YAG in managing unsightly scars in Indian skin type is few. In this study, we have used the Er YAG for fractional ablative resurfacing of the post-trauma and burns scars and studied the effect of the Laser on each scar parameter. The efficacy of the Laser on pigmentation is studied in special interest as pigmentation is one parameter that can attract a beholder's attention to any scar.

Materials and Methods

Table 1: Scars Included in the Study and their Etiology.

Scar Type	(n %)
Post Burn Scar	18(24.7)
Post Traumatic Scar	8(11)
Amputation Stump Scar	5(6.8)
LSCS Scar	2(2.7)
Electrical Burn Scar	7(9.6)
ALT Flap	4(5.5)
STSG	7(9.6)
FTSG	3(4.1)
Fasciotomy Scar	4(5.5)
Healed Scar Face	5(6.8)
Venesection Scar	2(2.7)
Post-operative Scar	4(5.5)
Keystone Flap	1(1.4)
SFJ Ligation	1(1.4)
Ischial rotation	1(1.4)
PBC release scar	1(1.4)
Total	73(100)

This study was conducted in the Department of Plastic Surgery at a tertiary care center after getting the departmental ethical committee approval. Informed written consent was taken from each patient for Er YAG laser therapy and photography of the scars. Total 73 scars were enrolled into the study randomly (Table 1) post-trauma, post-surgical and post burns scars were included. The scars were evaluated only twice during the study using the Vancouver scar scale scoring system, which included the following parameters and scores; vascularity (normal=0, pink=1, red=2, purple=3), pigmentation (normal=0, hypopigmentation= 1, hyperpigmentation=2), pliability (normal= 0, supple=1, yielding=2, firm=3, banding=4, contracture= 5), and height (normal=0, <2 mm=1, 2~5 mm=2, >5 mm=3) and clinical photography, once pre-treatment and next one month after the completion of the laser therapy. The laser therapy was given for four sessions each at a one-month interval. Er: YAG Laser therapy using already existing equipment in the department, Twain 2940, Quanta System S.p.A., Italy, in ablative as well as thermal mode, at a wavelength of 2,940nm, fluence was set to 1 to 2 J/cm2, pulse width used was 300 microseconds using spot diameter of 4mm.

During each session, two laser passes of 400 mJ in short pulse mode (pulse duration 0.30ms) and one pass of 800 mJ in long pulse mode (pulse duration 1 ms) were performed. The types of scars and response of each scar after the completion of the fourth sitting of the Er YAG were compared and statistical analysis was done using IBM statistical software, SPSS Statistics version 27 (IBM Inc.). Normally distributed data were expressed as mean ±SD. Data were expressed as median (interquartile range, IQR), when the assumption of normality was violated (Shapiro Wilk test, $P<0.001$). Paired T-Test was used and wherever needed one-way repeated measure ANOVA was done to determine whether there are any statistically significant differences between the means of three or more levels of a within-subjects factor over time. A P value<0.05 was considered statistically significant. And it was noted if there is any difference in response between scars of different etiologies to the Er YAG Laser.

Result

Table 2: Preprocedural and Post Procedural Changes of Vancouver scar scale variables.

VSS Parameter	Pre Procedural	Post Procedural	P Value
Vascularity	2+0.8	1.3+0.7	0.001
Pigmentation	1.47+0.6	1.2+0.6	0.006
Pliability	2.1+0.8	1.5+0.7	0.001
Height	1.6+0.7	1+0.6	0.001
Total Score	6.6+1.6	3.2+1.9	0.001

The mean age of patients was 35.2±7.8 (range, 18-50 years). The preprocedural and postprocedural

Vancouver scar scale parameters are compared (Table 2) and the response is plotted as graph (Figure 1). There was a significant difference in vascularity, pigmentation, pliability, and height after laser application (paired t-test, P=0.001, 0.006, 0.001, 0.001 and 0.001, respectively).

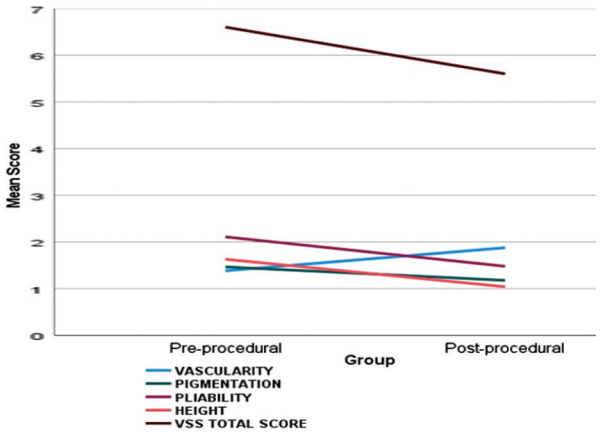

Fig. 1: Graph Showing the changes of VSS variables.

Discussion

The scar is defined as fibrous tissue that replaces the wound[2] During the process of healing the wound develops a bridge of collagen fibers with a thin epithelium, forming an immature scar.[3] The process of wound healing comprises three phases, the inflammation phase which lasts for a few days, the proliferation phase lasting for weeks, and the maturation phase takes several months or years. Hypertrophic scars begin to develop 6 to 8 weeks after wound healing, it grows for 3 to 6 months, and then regress after 6 months.[4] An immature scar is red, raised, rigid, and hypopigmented, During the process of maturation the scar becomes pliable, flatter, less vascular and color is normalized. The difference between the normal scar, immature scar lies in the difference in their extracellular matrix composition. A normal scar when mature consists of 80% type-I collagen with 10-15% type-III and a minimal amount of type-V collagen. This composition is altered in an abnormal scar with an increased ratio of type-III to type-I collagen and abnormal scar consists of around 33% type-III, 10% type-V, and around 60% type-I collagen. Apart from the composition of the collagen, the arrangement of fibrils and interfibrillar space also is different in an abnormal scar compared to the normal mature scar. The cellular function of fibroblasts and keratinocytes is also altered in an abnormal scar making them pro-fibrotic. The expression of cytokines is also altered in an abnormal scar. The balance between matrix metalloproteinase (MMPs) and tissue inhibitors of metalloproteinase (TIMPs) is altered and is moved towards the pro-fibrotic side. Transforming growth factor-β (TGF-β), connective tissue growth factor (CTGF), platelet-derived growth factor (PDGF), and insulin-like growth factor 1 (ILGF-1) are up-regulated, meanwhile interferon-α (IFN-α) and interferon-γ (IFN-γ) are down-regulated.[5]

There are many methods known for management of scars like scar massage, silicone gel or silicone sheet application pressure garments, medications for local application containing allantoin, heparin etc., intralesional steroids, surgical scar revision, and Laser Therapy. The first LASER machine was devised in 1960 by Maimon, which was a Ruby laser. Dr. Leon Goldman a dermatologist is the father of laser medicine. The first laser that was specifically designed for use in a medical condition was Pulsed Dye Laser (PDL), which was used for port-wine stains. Since then, laser technology has evolved with newer concepts of pulsed therapy, fractionated laser therapy, Q-switched mode, etc. being added to the list.

The principle of any laser is photo thermolysis, which was proposed first by Anderson. Each laser has a specific target on which it acts, known as chromatophore. The laser selectively acts on its chromatophore and produces thermal ablation of the target tissue. Fluence, pulse width, spot size, and stacking are variables that are to be adjusted according to the individual requirements The mechanism by which a laser affects scar remodeling is not fully known, but ablative fractional resurfacing may lead to the production of various cytokines and growth factors by stimulating a variety of not fully known cellular responses Fractional photo-thermolysis produces controlled and limited dermal heating which triggers a cascade of events in which leads to normalization of the collagenesis-collagenolysis cycle.

The present study was designed to find out if there is any difference in response to Er YAG Laser based on the etiology. There are studies which found factors that influence the response to the Laser based on location and the time on initiating the Laser therapy. But aim of the study was to find out if there is any difference based on etiology or the causative factors. Interestingly in our study we did not find any statistically significant difference.

Conclusion

The study shows that Er -YAG Laser therapy is

an effective method in the management of post-trauma scar. All the scars in the study showed good response to the Er YAG Laser Therapy and there is no statistically significant relation of etiopathogenesis with outcome. No adverse effects were noted during the study. The limitation of the study is that most of the scars were of maturation phase, many scars on the same individual were enrolled into the study, laser was given by a single specialist and follow up was for 6 months. We suggest large volume and multi-center study and longer duration of follow up to get a better picture of the effect of Er YAG laser.

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Original Article

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Clinicopathological Evaluation of Tympanosclerosis

Gangadhara Somayaji[1], Mukesh Edward[2], Harsh Suri[3]

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Abstract

Introduction: Tympanosclerosis is a disorder associated with calcareous deposits in various parts of the middle ear. It is an irreversible end result of any unresolved specific or nonspecific inflammatory disease of the middle ear characterized by anatomical distortion resulting in conductive type of hearing loss.[1]

Objective: This study was conducted in a tertiary care teaching hospital to know the incidence, to study clinical features, histopathology and the quantum of hearing loss secondary to tympanosclerosis (TS) in cases with chronic otitis media (COM) undergoing surgical treatment.

Methodology: All patients with chronic otitis media (COM), either mucosal or squamosal type undergoing surgery were included in the study.

Results: Out of 160 cases operated for COM, 52 cases (32.5 percent) had tympanosclerotic plaques. In 52 cases with TS plaques, 5 cases (all are males) were squamosal type with histopathological evidence of cholesteatoma and rest 47 cases were of mucosal type (24 males & 23 females). On histopathological evaluation of the TS plaques, only 7 cases showed extensive hyalinization and the reminder showed fibrotic changes, granulation tissue, dense collagen, scattered fibroblasts and inflammatory cells.

Conclusion: TS is a common association, clinically and pathologically with COM more with mucosal type. TS causes significant hearing loss compared to similar cases without TS. TS patches over critical areas like stapes foot plate, facial nerve canal and semicircular canals may be difficult to manage because of their strategic location.

Keywords: Tympanosclerosis; Chronic otitis media; Cholesteatoma.

Introduction

Tympanosclerosis is a condition associated with hardening of the middle ear cleft. (Tympanum-middle ear; sclero-hardening; osis-condition). It is a non-functional and non-changeable inert repair phenomenon.[1] The condition is characterised by calcareous deposits in the tympanic membrane, middle ear cavity, ossicular chain and occasionally in the mastoid.[2] Von Troltsch (1873) was the first person to coin the term Taukensklerose to describe sclerotic changes in the middle ear mucosa. Zollner (1955) later called it tympanosclerosis.[3] The fibrosis occurs in the submucosal connective tissue layer covering the auditory ossicles, lining the bony walls of the tympanic cavity and forms the middle fibrous layer of the tympanic membrane.[4] Sclerosis is the final stage of the dynamic inflammatory process.[1] TM(Tympanic membrane) is the most common site for tympanosclerotic patches where it is called myringosclerosis. It is considered as a long term sequelae of Chronic Otitis Media (COM).[5]

It is proposed that TS is not usually associated with active mucosal disease and with cholesteatoma.[6] As tympanosclerosis has a mysterious nature, this study was planned to know the incidence at our place, the clinical features, histopathology, level of hearing impairment and to plan the management of tympanosclerosis in both mucosal and squamosal type of chronic otitis media.

Materials and Methods

Study design

Patients coming to the ENT outpatient department of our tertiary care referral hospital, diagnosed with chronic otitis media either mucosal or squamosal type and posted for surgery, were included in the study. Patients with otosclerosis and non-chronic otitis media cases undergoing surgery, those with intracranial complications were excluded from the study. The study was conducted for a period of 18 months.

Sample size

A minimum of 50 patients was the sample size proposed for the study. However, the final sample size was 160 in 18 months.

Methodology

Ethical clearance was obtained from the institutional ethics committee. Relevant clinical and demographic data were obtained from the patients. A detailed clinical examination was performed. Informed and written consent was taken from all the patients. Audiological assessment (pure tone and impedance audiometry), radiological assessment (X ray bilateral mastoids lateral oblique view and in selected cases, HRCT temporal bone), laboratory investigations (complete blood count, bleeding time/clotting time, random blood sugar, blood urea, serum creatinine and urine analysis) and histopathology of excised plaques were done.

Results

Out of 160 cases operated for chronic otitis media, 52 (32.5%) had histopathological evidence of tympanosclerotic plaques with slightly higher preponderance in males. (56%) 5 cases were associated with cholesteatoma. (10%) All these 5 cases were males. Rest of 47 cases with TS patches were having male to female ratio of 24: 23 (Table 1).

Table 1: Gender distribution of squamosal disease (SPSS version 22.0).

	Male	Female	Total	p-value
TS patches associated with cholesteatoma	5	0	5	0.04* Fisher's Exact
TS patches not associated with cholesteatoma	24	23	47	test *P<0.05 statistically significant
Total	29	23	52	

Out of 52 cases, 29 underwent tympanoplasty. 16 underwent cortical mastoidectomy and tympanoplasty. 4 cases underwent modified radical

Table 2: Types of Surgeries (SPSS version 22.0).

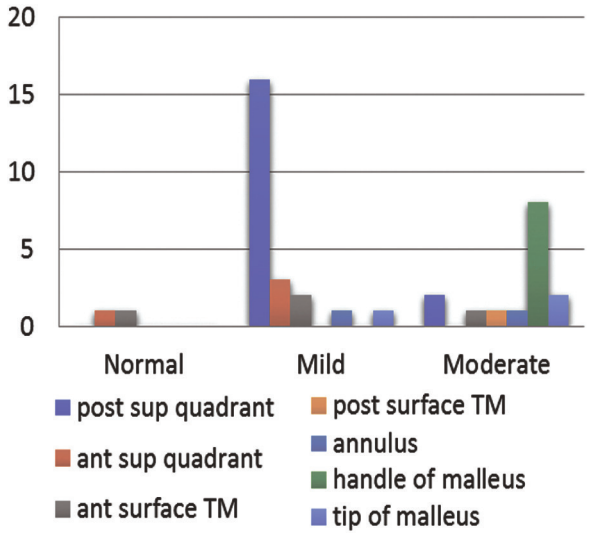
	Tympanoplasty	Cortical + Tympanoplasty	Intact Canal wall Mastoidectomy + Atticotomy + Tympanoplasty	MRM + Tympanoplasty	
Type 1`	25	12	2	1	0.048*
Type 2	4	4	1	1	Fisher's Exact test.
Type 3 & above	0	0	0	2	*P<0.05 statistically significant

mastoidectomy and tympanoplasty. Other 3 cases underwent intact canal wall mastoidectomy with atticotomy and tympanoplasty (Table 2).

Out of 52 intraoperative cases with TS patches, 40 cases (77%) had intact and mobile ossicular chain. Seven cases (13%) had partially eroded malleus, three cases (6%) were having absent malleus, one case (2%) both malleus and incus were absent and other one case (2%) where all the ossicles were absent except foot plate of stapes.

Hearing loss and distribution of TS patches where ossicular chain was intact and mobile in 40 cases were studied. (figure no.1) TS patches were more in posterosuperior quadrant of TM with mild CHL in 16 cases and moderate CHL seen in 8 cases with distribution of TS patches in handle of malleus. (Graph 1 -Y axis denotes number of cases).

Graph 1: Hearing loss & distribution of TS patches.



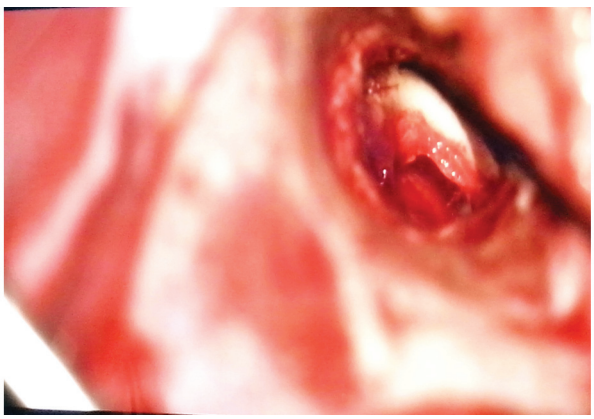


Fig. 1: Intraoperative TS patch in the anterosuperior quadrant of TM with intact handle of malleus.

Considering degree of hearing loss in 52 patients, out of the 5 cases associated with squamosal disease, 3 cases showed moderately severe mixed hearing loss and one each with moderate and mild CHL. Rest of the 47 cases had hearing pattern as per table no.3. The middle ear mucosa was healthy in 16 cases who underwent only tympanoplasty. Remaining cases of mucosal disease had thickened and oedematous mucosa. The mucosa was unhealthy in all the squamosal cases.

Table 3: Degree of hearing loss (SPSS version 22.0).

Hearing Status	Without Cholesteatoma	Cholesteatoma + TS patches	Total
Normal hearing	2	0	2
mild CHL	24	1	25
moderate CHL	14	1	15
moderately severe mixed HL	7	3	10
Total	47	5	52
	Fisher's exact value = 4.95, p= 0.128(NS) Fisher's exact test P<0.05 statistically significant p>0.05 non-significant, NS		

Regarding histopathology of TS patches, fibrotic changes were more evident with fragments of keratin material. Cases associated with Cholesteatoma showed granulation tissue, dense collagen, scattered fibroblasts and inflammatory cells. Only 13 cases showed extensive hyalinization and calcification, on which 2 cases were cholesteatoma associated. Two cases had associated refractory foreign material with nonspecific granulation tissue surrounded with chronic inflammatory infiltrate.

Discussion

Cassebohm described this entity as `chalky patches' in the tympanic membrane as early as in 1734. Gibb AG (1976) called this as the 'cinderella of the middle ear disease.[1] The presentation of the disease is variable and aetiologies may be different. There are too many hypothesis on the aetiology of TS. It is proposed to be secondary to infection associated with necrosis and granulation, ischemic alterations, and/or stasis of infected materials. It could be caused by immunologic reaction. Connective tissue components are stimulated because of infection, inflammation or trauma and this initiates the local immunological reaction in the submucosa.[7]

Tympanosclerosis though a relatively benign condition, causes significant conductive type of hearing loss. The severity of hearing loss depends on the extent of the tympanosclerosis plaques and their location. which can be judged only after formal exploration of the middle ear.[5] The sclerotic changes

may damage the middle ear both anatomically and functionally.[8] It is usually because of inflammation, but rarely may appear after trauma or surgery or any non-treated inflammatory process may be the cause.[9]

The incidence of tympanosclerosis in chronic otitis media has been reported to range from 9% to 38%. It appears histologically as an acellular hyalinization of the subepithelial connective tissue of the tympanic membrane and middle ear associated with calcification and neo osteogenesis. Ossicular fixation occurs most frequently in the attic and is associated with the heads of the malleus and body of incus. When plaques occur within the tympanic membrane, they are limited to the lamina propria.[10] Microscopically, there is hyalinized collagen in the submucosa with a lamellar arrangement. Mature fibrocytes may be seen between collagen fibers.[11]

Under light microscopy, four different phases in the development of tympanosclerosis can be observed histologically. During the early phase, vesicles of around 50-300 nm in diameter are formed in the fibroblasts, inflammatory cells, and epithelium cells of the extracellular collagenous matrix followed by the appearance of crystalline like inclusions called calcospherules in these vesicles. Calcium and phosphate precipitate in these spherules and mineralization begins. During the last phase, masses that are completely mineralized appear and they are then called plaques.[12]

Under otoendoscope or microscope, plaques are classified as type I (soft), type II (moderately hard) and type III (very hard).[13] In type I, fibroblasts and collagen fibers are equally abundant in typical loose connective tissue. A few small calcium crystals are also seen. In type II TS, large bundles of collagen fibres, proliferation of fibroblasts and focal calcification points are seen. In type III, round shaped chondroblast like cells located in lacunae and intense calcification points are evident.[13]

Another way to classify TS is based on their morphological and histological aspects. Histological classification is based on the maturation of the tissue, and thereby helps to grade the disease. In type I tympanosclerosis, even after surgical excision, the underlying process may go on and new sclerotic tissue formation can be expected. Type III sclerotic tissue is associated with limited and inactive disease. Progress of the disease and the patient's benefit from surgery can be interpreted according to this classification.[13]

Radiologically tympanosclerosis appears as unifocal or multifocal punctate or web like calcifications in the middle ear cavity or on the tympanic membrane. New bone formation (fibro-osseous sclerosis) is usually seen in the attic and is the least common manifestation. Thick bony webs or generalized bony encasement may be seen in computer tomography (CT).[14]

Tympanoplasty and ossicular reconstruction in ears with tympanosclerosis carry higher risk of cochlear damage than in other middle ear diseases because of the extensive dissection that is required in these ears and the coexistence of labyrinthine fistula.[10] The dense plaque on the tympanic membrane reaching the perforation edge should be removed from the medial aspect to facilitate vascularization of the graft.[15]

In tympanosclerosis with intact tympanic membrane, if there is a significant conductive hearing loss, it indicates ossicular chain involvement and surgery is indicated. After removing tympanosclerosis plaques and involved ossicles, ossiculoplasty with or without tympanic membrane reconstruction is performed.[16] On the other hand, if the stapes is fixed, a staged operation is necessary in which a first stage myringoplasty and management of the malleus and the incus is followed by a second stage surgery after a few months' interval.[16]

Several studies were conducted regarding tympanosclerosis. Most of the studies were focussing on the incidence, histopathology, distribution of plaques and degree of hearing loss.

Pal I and Sengupta A did a study on the distribution of tympanosclerosis, their clinical presentation, and the possible surgical treatment, the results of those surgeries and the histopathological nature of tympanosclerosis plaques. They found that TS occurs equally in both sexes and the commonest site being tympanic membrane affecting any quadrant of pars tensa sparing pars flaccida, associated commonly with conductive hearing loss and very rarely sensorineural hearing loss (SNHL). Histopathology revealed dense bundles of collagen with hyaline degeneration and scattered areas of calcification.[1]

Kaur K, Sonkhya N, Bapna as did another study to identify the incidence of tympanosclerosis amongst patients with chronic suppurative otitis media and also to study the correlation between the degree of hearing loss and the site of tympanosclerosis. Audiometric and operative findings of 200 patients of chronic suppurative otitis media were analysed. The incidence of tympanosclerosis was found to be 19% (out of 200 patients). The hearing loss associated

with tympanosclerosis was of the conductive type in the majority of cases. Ossicular mobility was found to be normal in 71.1% of the cases.[3]

In our study, males have slightly more incidence of TS patches (M: F - 24:23). Also, TM was the most common site for tympanosclerotic patches with majority seen in posterior superior quadrant of the TM (mild CHL), followed by handle of malleus (moderate CHL). Regarding histopathology of TS patches, fibrotic changes were more evident with fragments of keratin material. Two cases with cholesteatoma had refractory foreign material with nonspecific granulation tissue surrounded with chronic inflammatory infiltrate. In our study, 40 (77%) had intact and mobile ossicular chain. Seven cases (13%) had partially eroded malleus, three cases (6%) were having absent malleus, one case (2%)–both malleus and incus were absent and other one case (2%) where all the ossicles were absent except foot plate of stapes.

Asiri S, Hasham A, Anazy F A, Zakzouk S, Banjar A conducted a study to estimate the incidence of tympanosclerosis among patients with chronic suppurative otitis media (COM), its association with cholesteatoma and also the type of hearing loss as well as its relation to the degree and site of tympanosclerosis. They concluded that incidence of tympanosclerosis was found to be 11.6 per cent (90 patients out of 775 COM cases) and also found that association of cholesteatoma and tympanosclerosis may be regarded as uncommon (2.2 percent).[17]

In our study, out of total 160 cases operated for chronic otitis media, 52 (32.5 %) has histopathological evidence of tympanosclerotic plaques. Ten percent (5 cases) also showed association with squamosal disease, which was significantly higher compared to previous studies. All of the 5 cases who were associated with squamosal disease were males.

Yetiser S, Hidir Y, Karatas E, Karapinar U conducted a study to review the previous reports and to analyze the long-term surgical outcome of 30 patients who have been operated for tympanosclerosis. They concluded that the success of the surgery was dictated by the location and the extent of tympanosclerotic involvement.[18]

In our study, out of 52 cases, 29 underwent tympanoplasty. 16 patients underwent cortical mastoidectomy and tympanoplasty. Modified radical mastoidectomy with tympanoplasty done in 4 cases and other 3 underwent intact canal wall mastoidectomy with atticotomy and tympanoplasty.

Summary and Conclusion

The purpose of this study was to evaluate the patients with TS patches in chronic otitis media of both mucosal and squamosal type. Total of 160 cases were operated for chronic otitis media, 52 (32.5%) had histopathological evidence of tympanosclerotic plaques. Males were showing slightly more incidence. Association of squamosal disease (cholesteatoma) with TS patches was seen significantly higher compared to previous studies. Previously not more than 5 percent were seen; however, our study showed a 10 percent association. Interestingly all the subjects with cholesteatoma were males. The TS patches were seen more in the posterosuperior quadrant of TM where the hearing loss is only mild, followed by handle of malleus with moderate CHL.

On histopathology, Cholesteatoma associated cases showed granulation tissue, dense collagen, scattered fibroblasts and inflammatory cells. Fibrotic changes were more evident with fragments of keratin material. Only 13 cases showed extensive hyalinization and calcification, of which 2 cases were associated with cholesteatoma.

TS is commonly associated clinically and pathologically with COM more with mucosal type. It causes significant hearing loss compared to similar cases without TS. TS patches over critical areas like stapes foot plate, facial nerve canal and semi-circular canals may be difficult to manage because of their strategic location. The present study starred a significant association of TS with COM. The associated clinical manifestations, hearing loss and management of these cases as done in this study, add to the existing information in the literature.

More studies are needed to unfold the mystery of TS patches in future. The surgeon has the key role depending on expertise to remove the plaques with less morbidity.

Abbreviations

TS – Tympanosclerosis

COM – Chronic Otitis media

TM – Tympanic membrane

CHL – Conductive hearing loss

HRCT – High Resolution Computed Tomography

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Original Article

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Role of Non-Cultured Keratinocyte Cell Grafting in Take of Full Thickness Skin Graft in Post Burn Contracture

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Abstract

All deeper burns i.e. second degree deep dermal and full thickness heals by scarring that causes restrictions in the movements and aesthetics issues for patients. Burn reconstructive surgery requires that the defects after release should be replaced with donor tissues which have matching texture and colour like autologous skin grafting or flap surgeries. Here we are using this method to look for role in take of FTSG post burn contracture. Full-thickness skin grafts include full thickness of the epidermis and dermis whereas split-thickness skin grafts (STSG) include the entire epidermis and only partial dermis. The main complication of this procedure is risk of graft failure. Keratinocyte cells suspension is claimed to hasten the wound healing. In this article, we share our experience of using non-cultured keratinocyte grafting (NCKG) in improving the take of full thickness skin graft (FTSG).

Keywords: NCKG; FTSG; Post burn contracture.

Introduction

Burn trauma constitutes the second most common cause of trauma related deaths after vehicular accidents, in both developing and developed country. An extensive burn is the most devastating injury that human being had to suffer. After immediate concern for survival in victim, restoration to pre-injury status, and return to daily activities becomes important for victim and treating team.[1] A healed burn patient may be left with contractures and scars with varying degrees of functional issues and cause social stigma among victims. The healed contractures need to be revised and raw areas covered with FTSG or STSG which may be associated with delayed wound healing of the skin grafts due to aberrant vascularity of the graft bed.

Materials and Methods

This study was conducted in Plastic surgery department in a tertiary care center in the month of November-December 2021. The patient is male child a case of Post burns recurrent band like

constriction of Right index and middle fingers with restriction of daily activities with USS:12/13 (figure 1). Release of post burns contracture with FTSG with K-wire fixation was done. (Figure 2) post operatively there was graft loss (figure 3) for which we have used non cultured keratinocyte epidermal graft (NCKEG) to manage the graft loss. Under all aseptic precautions, a 3cm x 1cm area of groin region was marked (Figure 4) Local anaesthesia (2% xylocaine) was given.

The donor area was derma braded (Figure 4) after the application of mupirocin ointment. The paste, containing dermabraded cells, was collected, homogenized, and was applied on the wound.

A non-adherent dressing was placed on it followed by gauze dressing. The wound was inspected on the 7th day and thereafter weekly. Remnant raw area was calculated on each dressing.

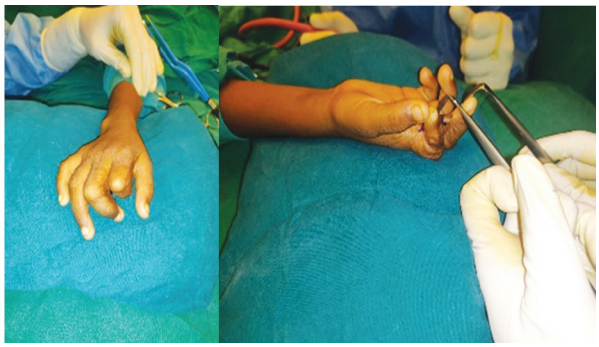

Fig. 1: Post burns contracture in right index and middle finger.

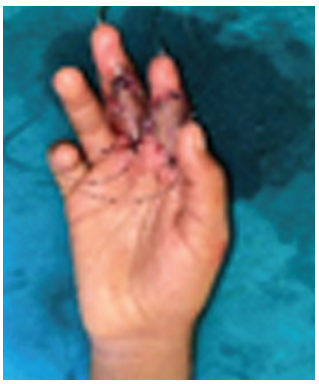

Fig. 2: PBC release with FTSG and K WIRE fixation done.

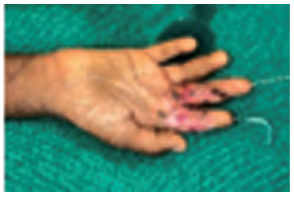

Fig. 3: FTSG raw area.

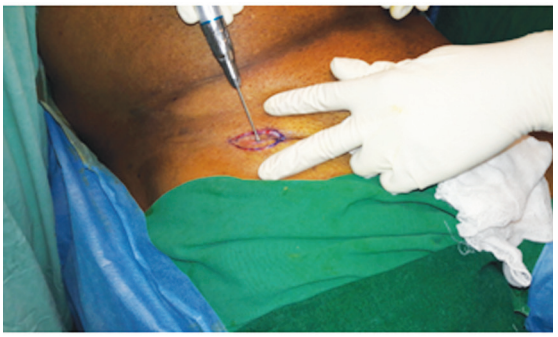

Fig. 4: NCKG harvested.

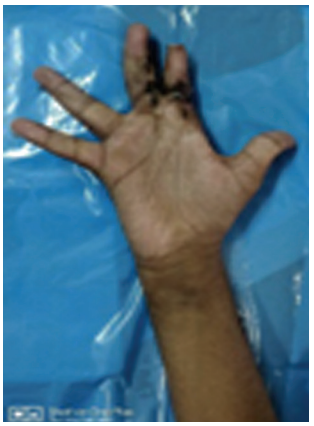

Fig. 5: Healed FTSG.

Results

NCKG treated wound showed accelerated wound healing (figure 5). Though the grafted cells did not survive but rapid epithelialization started from the periphery of the wound.

Discussion

Wound healing is a complex process. It involves three phases-inflammation proliferation and maturation.[1] The chronic wounds are characterized by a prolonged and persistent proliferative phase due to altered local and systemic factors. The spectrum of modalities available to manage these types of wounds is very wide.

Conveniently it can be grouped into four categories - conventional therapy, novel therapy, reconstructive therapy, and cell-based therapy. Conventional therapies include–conventional dressings with or without topical application of antimicrobial agents, growth factors; various biological dressings such as silver and alginate; hyperbaric oxygen, etc. Novel therapies include the use of platelet-rich plasma, negative pressure wound therapy (NPWT), and skin substitutes. These are minimally invasive with much better

healing efficacy than conventional therapies. Reconstructive therapy, such as skin and flap grafting, are invasive and damage the normal tissue also. Cell-based therapy is also emerging as a part of wound management.[2,3]

Application of cultured keratinocytes appears to promote healthy granulation tissue formation within the wound bed. The graft, when applied as a sheet, act as an occlusive dressing, preventing wound dehydration and maintaining a moist environment. The majority of evidence suggests that cultured epidermal allografts do not survive indefinitely after transplantation.[4] Their brief contact with the wound, however, seems sufficient to stimulate reepithelialisation, particularly when dermal tissue is present in the wound bed. This may be due to the release of growth factors by keratinocytes which may favourably influence wound healing. In addition to this, there is a release of several growth factors by keratinocytes that promote wound healing. It is known that cultured keratinocytes release various factors that enhance the growth of other cells in vitro including keratinocytes, fibroblasts, and melanocytes. Identified factors include interleukin-1, other interleukins, and transforming growth factor-alpha.

These keratinocytes may be autologous or allogenic in origin. These cells are separated from skin graft by using trypsin or other methods. After separation, these are cultured in appropriate media to form a sheet. These sheets are used as graft to cover the wound. In our case, we have used autologous non-cultured, non trypsinised keratinocytes cells to promote the healing. We observed favourable result in terms of formation of healthy granulation tissue and rapid epithelialization of the wound from the margins.

Conclusion

In this study we found that non-cultured keratinocyte grafting has role in healing of the wound and the wound heals at faster rate. But since it is a single case study, definite conclusion cannot be made. Large randomized control trials are required to confirm the efficacy of NCKG in wound healing.

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The second page should carry the full title of the manuscript and an abstract (of no more than 150 words for case reports, brief reports and 250 words for original articles). The abstract should be structured and state the Context (Background), Aims, Settings and Design, Methods and Materials, Statistical analysis used, Results and Conclusions. Below the abstract should provide 3 to 10 keywords.

Introduction

State the background of the study and purpose of the study and summarize the rationale for the study or observation.

Methods

The methods section should include only information that was available at the time the plan or protocol for the study was written such as study approach, design, type of sample, sample size, sampling technique, setting of the study, description of data collection tools and methods; all information obtained during the conduct of the study belongs in the Results section.

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Present your results in logical sequence in the text, tables, and illustrations, giving the main or most important findings first. Do not repeat in the text all the data in the tables or illustrations; emphasize or summarize only important observations. Extra or supplementary materials and technical details can be placed in an appendix where it will be accessible but will not interrupt the flow of the text; alternatively, it can be published only in the electronic version of the journal.

Discussion

Include summary of key findings (primary outcome measures, secondary outcome measures, results as they relate to a prior hypothesis); Strengths and limitations of the study (study question, study design, data collection, analysis and interpretation); Interpretation and implications in the context of the totality of evidence (is there a systematic review to refer to, if not, could one be reasonably done here and now?, What this study adds to the available evidence, effects on patient care and health policy, possible mechanisms)? Controversies raised by this study; and Future research directions (for this particular research collaboration, underlying mechanisms, clinical research). Do not repeat in detail data or other material given in the Introduction or the Results section.

References

List references in alphabetical order. Each listed reference should be cited in text (not in alphabetic order), and each text citation should be listed in the References section. Identify references in text, tables, and legends by Arabic numerals in square bracket (e.g. [10]). Please refer to ICMJE Guidelines (http://www.nlm.nih.gov/bsd/uniform_ requirements.html) for more examples.

Standard journal article

[1] Flink H, Tegelberg Å, Thörn M, Lagerlöf F. Effect of oral iron supplementation on unstimulated salivary flow rate: A randomized, double-blind, placebo-controlled trial. J Oral Pathol Med 2006; 35: 540-7.

[2] Twetman S, Axelsson S, Dahlgren H, Holm AK, Källestål C, Lagerlöf F, *et al*. Caries-preventive effect of fluoride toothpaste: A systematic review. Acta Odontol Scand 2003; 61: 347-55.

Article in supplement or special issue

[3] Fleischer W, Reimer K. Povidone iodine antisepsis. State of the art. Dermatology 1997; 195 Suppl 2: 3-9.

Corporate (collective) author

[4] American Academy of Periodontology. Sonic and ultrasonic scalers in periodontics. J Periodontol 2000; 71: 1792-801.

Unpublished article

[5] Garoushi S, Lassila LV, Tezvergil A, Vallittu PK. Static and fatigue compression test for particulate filler composite resin with fiber-reinforced composite substructure. Dent Mater 2006.

Personal author(s)

[6] Hosmer D, Lemeshow S. Applied logistic regression, 2nd edn. New York: Wiley-Interscience; 2000.

Chapter in book

[7] Nauntofte B, Tenovuo J, Lagerlöf F. Secretion and composition of saliva. In: Fejerskov O,

Kidd EAM, editors. Dental caries: The disease and its clinical management. Oxford: Blackwell Munksgaard; 2003. p. 7-27.

No author given

[8] World Health Organization. Oral health surveys - basic methods, 4th edn. Geneva: World Health Organization; 1997.

Reference from electronic media

[9] National Statistics Online—Trends in suicide by method in England and Wales, 1979-2001. www.statistics.gov.uk/downloads/theme_health/HSQ 20.pdf (accessed Jan 24, 2005): 7-18. Only verified references against the original documents should be cited. Authors are responsible for the accuracy and completeness of their references and for correct text citation. The number of reference should be kept limited to 20 in case of major communications and 10 for short communications.

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