Case Report

# Challenges in Managing Eyelid Basal Cell Carcinoma with Mohs Surgery and Close the Large Eyelid Defect after Tumor Removal

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**ABSTRACT**

***Background:*** *Basal cell carcinoma (BCC) is the most common skin cancer of the eyelid. The important things management we have to emphasize are to prevent recurrence rate and to get the good cosmetic result after reconstruct the large defect. The aim of this case report is to show the management of both eyelid basal cell carcinoma and large eyelid defect after tumor removal.*

***Methods:*** *Case Report. A Woman 40 years old was reffered from Dermatovenereology Department of Cipto Mangunkusumo Hospital with aggressive type BCC. The location of the tumor was at inferior left eyelid with size of 18 x 19 mm. They planned to perform Mohs' surgery and consulted to Plastic and Reconstruction division of Ophthalmology Department for eyelid reconstruction. After Mohs' surgery the horizontal length of the eyelid defect was more than 50% and vertical defect was more than 15 mm. The Mustarde cheek advancement flap were choosen.*

***Results:*** *Post operation necrotic tissue was noted do to ischemic problem. Oral corticosteroid was given with tapering dose for six days. One month later, the condition was improved and Dermatovenereology department performed diode laser for the necrotic tissue and further showing good result.*

***Conclusion:*** *The management for eyelid BCC were still challenging including the technique to reconstruct the large eyelid defect, the risk of recurrence after the tumor removal and post operative result. The dicision to choose the proper technique to reconstruct the eyelid defect and tight follow-up after surgery will give optimal functional and cosmetic result.*

***Keywords:*** *basal cell carcinoma; Mohs surgery; Mustarde cheek advancement flap; necrotizing flap; corticosteroid; diode laser*

Basal cell carcinoma is a malignant cutaneous neoplasm capable of extensive tissue destruction.[1] Although rarely metastasize, these tumors can be extremely destructive locally and can cause death if the invasion reach central nervous system.[1,2,3,4] Basal cell carcinoma is the most common skin cancer of the eyelid, accounting for 80-90% of cases.[1,3,5,6] Most BCCs arise on the lower eyelid or medial canthus. As many as 60% of these patients may have an unsuspected BCC located elsewhere on the face.[3] The eyelids and nose are the most common sites of BCC in young adults.[1]

In the past, 2-22% of patients with basal cell carcinoma of the eyelids ultimately required enucleation or exenteration, and the mortality from aggressive local involvement has been as high as 11 %.[3] Approximately 95% of all BCCs occur in people between 40 and 79 years of age.[1,2,7] The average age at diagnosis for BCC of the eyelid is nearly 60 y.o. However, BCC does occur in children and young adults. Many younger patients with BCC have an inherited predisposition to cutaneous neoplasia. Men are disproportionally afflicted, but this disparity has been diminishing.[1]

The important etiologies factors for this tumor are ultraviolet light exposure and hereditary.[1,4,5,7] The effects of sun exposure are cumulative, as reflected in the increasing incidence of the tumour with advancing age.[5] There are several managements of eyelid BCC, but the most important thing we have to emphasize is to prevent recurrence rate. Some studies reported the recurrence rates due to the underestimation of the subclinical extension of BCC are between 5% to 100%.[1] This varies according to the therapeutic method used.[5] The main treatment modality for BCC is surgical excision of the lesion with microscopic monitoring of its margins, Mohs microsurgery.[7,8]

The next challenge is how close of eyelid defect after the tumor removal. The goals of eyelid reconstruction are to provide structural and functional restoration with an acceptable aesthetic result.[9,10]. There are many considerations to close the defect, for instance likes the size or number of tissue loss, location, state of remaining ocular tissue, visual status of the fellow eye, age and general health condition, and the other structures involve of the eyelid defect.[5]

The purpose of this case report are to demonstrate the challenges how to managing inferior eyelid BCC, how to managing inferior eyelid defect after tumor removal and how to managing post surgery problems after close the defect.

## CASE ILLUSTRATION

Woman 40 years old came to DV Department of RSCM on August 22th, 2014, with chief complaint bleeding from her lower eyelid of the left eye. She had freckle on her medial lower eye lid of the left eye. Since 3 weeks before admission there was an ulcer revealed on that freckle, and sometime along with sensation of itchy, pain and bleed. There were no histories of allergy, same complaint before, diabetes, and hypertension. There was no history of family member whose suffer BCC.

The physical exam revealed the solitary ulcer, irregular shape, with diameter 1.5 cm, hyper-pigmentation and telangiectasia on the edge of ulcer located on the medial lower eye lid of the left eye. Patient diagnosed as suspicious basal cell carcinoma of the eyelid and plan to biopsy on the next week. The biopsy test result was nodular infiltrating type. Patient then plans to do the Mohs Surgery and consulted to Plastic and Reconstruction division of Eye Department to do eyelid reconstruction after Mohs Surgery.

On September 24th, 2014 she came to Plastic and Reconstruction Division of RSCM Kirana. The ophthalmological status of the left eye showed visual acuity was 6/6 with glasses, the Intra Ocular Pressure (IOP) was 13 mmgHg. The left inferior eyelid showed the black mass with multiple nodules size 18 x 19 mm with ulceration and neovascularization but no blood, pus nor exophthalmos (figure 1). Vertical eyelid fissure was 8 mm, horizontal eyelid fissure was 27 mm, and margin reflex distant was 1 mm. the other parts and the right eye were unremarkable. Patient was diagnosed with suspicious inferior eyelid Basal Cell Carcinoma of the left eye and plan to CT-Scan with and without contrast.

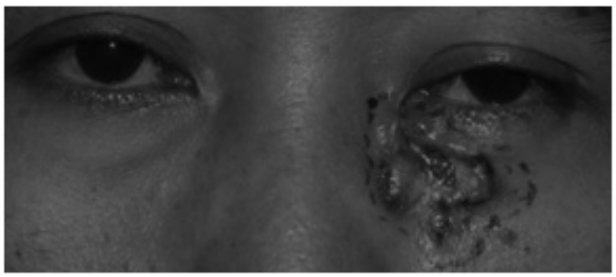

**Figure 1.** black mass with multiple nodules on the inferior eyelid of the left eye

Two weeks afterward, October 7th, 2014, patient came again to our division. The CT-Scan revealed mass infiltration until subcutaneous area on the inferior eyelid with deviation of nasal septum to the right side and concha hypertrophy but the bone was intact, than she was planned to do the surgery on October 27th, 2014.

On October 27th 2014 patient came to do the surgery. The surgeries were divided into two steps, the first step was incision the tumor with Mohs Surgery in local anesthesia and the second step was reconstruction the eyelid defect in general anesthesia. Before do the Mohs surgery, the tumor was marked with Dermatoscope (figure 2A) on the edge of the venectation, and then the location of incision was marked 2 mm peripherally from the previous mark (figure 2B & C). The local anesthesia was done by injection the mix solution of pehacaine and NaCl (1 : 3).

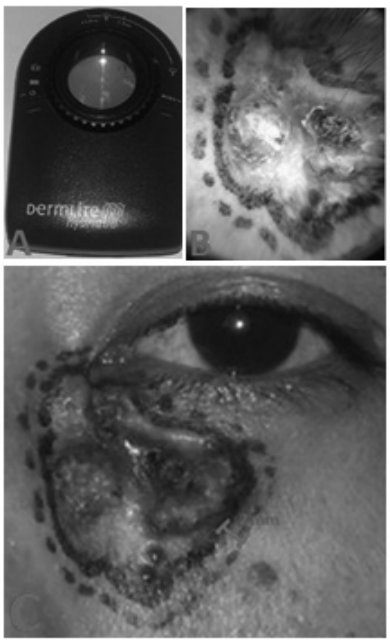

**Figure 2.** A) Dermatoscope; B) Edge of the lesion, which is viewed from dermatoscope; C) 2 mm length peripherally from the edge of the lesion venectation

The incision was started from the peripheral mark to take the entire lesion then continued with incision 2 mm peripherally with 2 mm depth (figure 3a & b) and make the mark incision (Figure 3c). After incision, the tumor was checked under microscope and the result was there were still remaining tumors on the edge of incision (figure 3d & e). The incision was continued on the part that still have remaining tumor with 2 mm in peripheral and depth orientation and checked again under microscope (figure 4a & b). After the second incision (figure 4c), the area of the lesion was totally clean from the tumor and continued to do the eyelid reconstruction surgery with cheek rotational flap procedure.

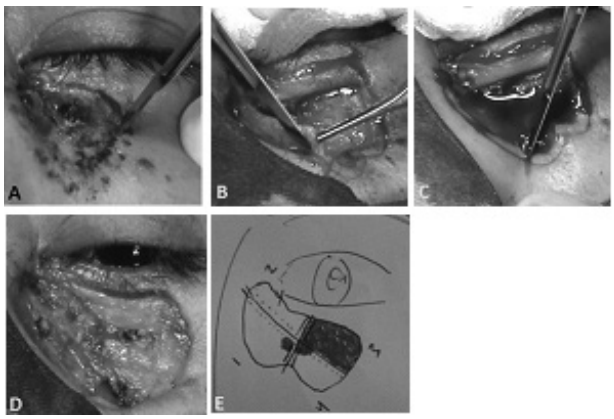

**Figure 3.** A) The incision on the edge of lession; B) Continue with incision 2 mm peripherally with 2 mm depth; C) Make the incision mark; D & E) After incision, the tumor was checked under microscope and the result was there were still remaining tumors on the edge of incision

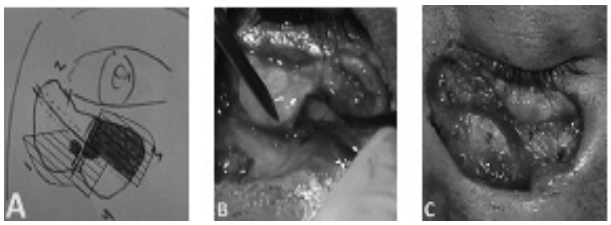

**Figure 4.** A) The location plan to re-incision; B) Second incision on the remaining tumor area; C) After 2nd incision appearance

Due to the widening of the incision until the medial canthus region from the second incision, the procedure was started from evaluation the lacrimal sac with Anel test from both punctums and the result was positive (figure 5A). The location of flap were marked from the upper

lateral side of the wound and pass through the lateral canthus then make a curvilinear line pass the ear tragus until ear lobe (figure 5B).

First incisions were made with subcutaneous depth from near the ear lobe follow the mark line till edge of the defect and then undermine as far as the same level of the ear lobe to make a flap (figure 5c). The next steps were rotated and pulled the flap to the nasal region to evaluate a good apposition between the flap to the defect side (figure 5d). All margin of the flap than sutured with simple interrupted sutures technique (figure 5e). The therapy after surgery were lincomycin 3 x 500 mg, cataflam 2 x 50 mg, kemicetine 3 x LE, Tranexamic acid 3 x 500 mg.

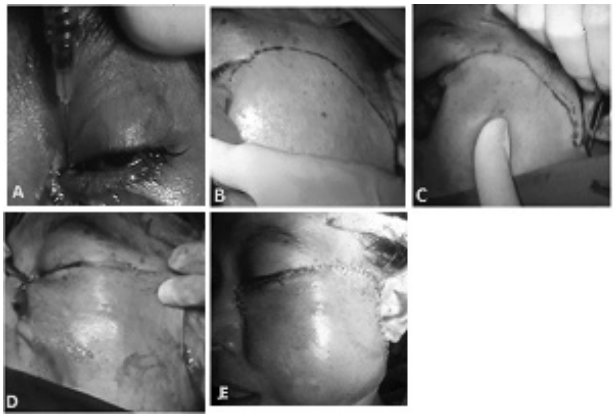


**Figure 5.** A) Anel test from both punctums; B) Marked the area to make a flap; C) First incisions were made with subcutaneous depth; D) Rotated and pulled the flap to the nasal region; E) Sutured all margin with simple interrupted sutures technique

One week after surgery, patient came to follow up. The physical examination showed the necrotic tissue on the margin of the flaps, edema and hematome but no pus or bleed (figure 6a). From DV department they planned to add oral corticosteroid 3 x 8 mg for two days and tappered 8 mg every two days. After 6 days, the edema and hematome were subsided , we planned to partial aff hecting and from the DV department planned to remove the crusta (figure 6b). The therapy was continued with a NaCl compress, oral methylcobalamin 1 x 1 tablet and Mederma® ointment 3 times.

One month after surgery, the condition got improve. For the therapy we just gave artificial tears and for the futher therapy was continued by DV department. From DV department they planned to NaCl compress within 15 minutes, wound dressing with Cutimed Sorbact Gel® and Cutimed Siltec®, Diode laser, and clindamycin 3 x 300 mg. Patient was planned to follow up every three days. Two weeks afterward, there were a yellow discharge revealed. They was changed antibiotic to amoxyclav 3 x 625 mg, continue Diode laser and fusidic acid 2 x daily and after two weeks therapy the condition show much improvement (figure 7).

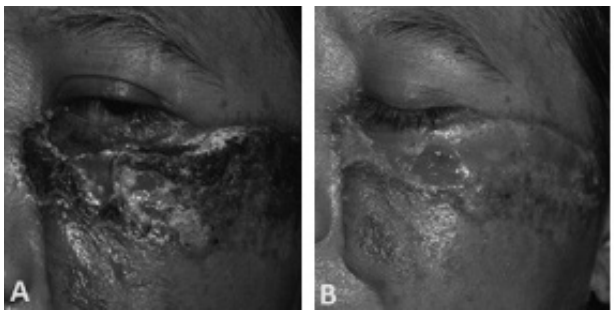


**Figure 6.** A) Necrotic tissue on the margin of the flap; B) Reddish appearance after remove the crusta and necrotic tissue

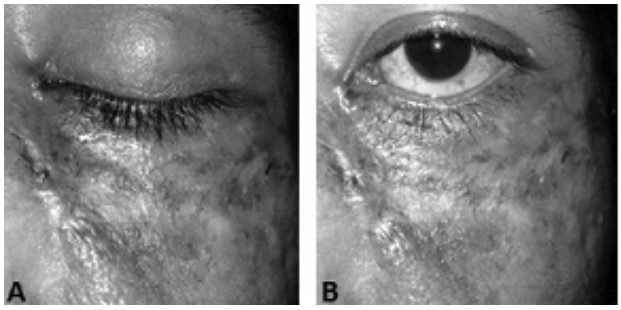


**Figure 7A & B.** The flap margin condition show much improvement with covered by healthy tissue

## DISCUSSION

Managing the eyelid tumors still became one of the challenging cases. Several consideration that surgeon have to thinks were how to diagnosed the type of tumor, how to remove it and how to close the tissue defect after tumor removal. Eyelid tumors can be benign or malignant but it is not always easy clinically to differentiate between them. As the consequence of failure to identify a malignant tumour in its early stages can be severe[5].

Our patient was diagnosed as eyelid BCC when she came for the first time to DV department. The diagnosis were based on the characteristic features of malignancy such as

ulceration, telangiectasias, pearly borders, and irregular margins and the tumor location and on the biopsy examination.[1,11] Although malignant eyelid disease is usually easy to diagnose on the basis of the history and clinical signs identified on careful examination differentiating between benign and malignant periocular skin lesions can be challenging because malignant lesions occasionally masquerade as benign pathology. Therefore, an eyelid biopsy is often required to make the correct diagnosis.[12]

Basal Cell Carcinoma which growth on the face will be appeared along embryonic fusion planes (figure 8). However, on the periocular area, it is most commonly found on the lower eyelid and medial canthus, and least often near the lateral canthus.[1,5,7,12] The differential diagnosis of BCC in clinical and histopathologic is broad, but in case the BCC was the most common malignant lesion of the periocular skin, we can suspected this tumor with BCC.[7] This clinical appearance were the same with our patient.

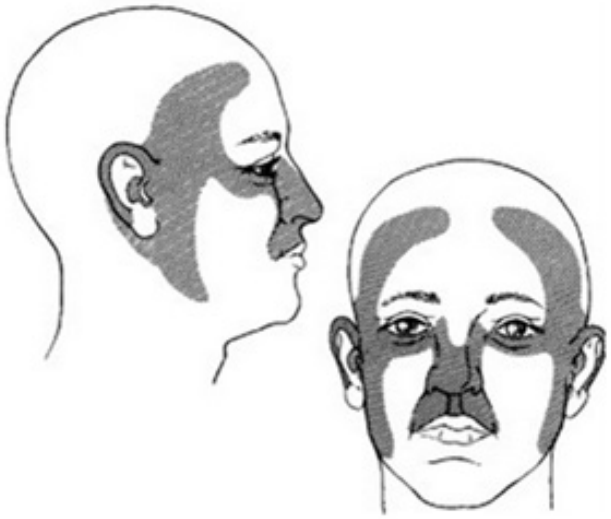

**Figure 8.** High-risk mask area of the face in basal cell carcinoma[13]

The important etiologies factors for this tumor are ultraviolet (UV) light exposure and hereditary.[1,4,5,7,12] The effects of sun exposure are cumulative, as reflected in the increasing incidence of the tumors with advancing age[5]. The mechanism how ultraviolet light induces BCC is not fully understood, although evidence suggests primary injury to DNA. In normal adults there is some decline in the ability to repair DNA with advancing age, but the clinical significance of this observation has not been established.[1]

The evidence supporting the role of UV radiation as a cause of BCC is based on six observations: 1) the tendency of BCC to arise on sun- exposed skin; 2) high rate of BCC among persons who work outdoors; 3) the predisposition of fair-skin individuals for BCC and the rarity of BCC among persons with dark-complexions; 4) the exceptionally high rate of BCC in people with UV-light sensitive diseases such as albinism and xeroderma pigmentosum; 5) the general decline in prevalence of BCC with increasing latitude; and 6) the ability of UV light to induce skin cancer in experimental animals.[1] The most mutagenic wavelengths are 290-320 nm or UVB.[1,14]

Based on the anamnesis, our patient has no history of chronic sun exposure. Actually, there are several observations that cannot be adequately explained by a theory based solely on UV light exposure. For instance, as many as a third of BCCs arise on skin surfaces that get relatively low sun exposure. BCC also is not common on some highly sun-exposed skin areas, such as the back of the hands and forearms.[1]

The other risk factors for BCC are focal trauma, ionizing radiation and arsenic exposure, tendency to freckle and smoking.[1,2,7] Freckle was one of the other risk factors that we can found in this patients, and from anamnesis she stated that the ulceration started from her freckle. Thermal burns trauma may be the most injury associated with BCC. The interval between primary injury and detectable carcinoma is variable, ranging from 2-75 years. There are also reported that BCC have been arise in the scar of chicken pox, small pox, hair transplant site and cutaneous lupus. Basal Cell Carcinoma can arise due to complication of radiation therapy on the site of radiation, but the latency between radiation and onset the tumor is long, in the range of decades. The threshold amount of radiation that induces skin cancers has not been established, but doses as low as 50 cGy have been associated with BCC of the scalp.[1]

There are several different clinical features of BCC, including nodular and nodulo-

ulcerative, pigmented, cystic, and infiltrating or morpheaform or sclerotic BCC.[1,2,6,7,12] The nodular BCC begins as a small papule and slowly enlarges to an irregular, dome-shaped tumor. The epithelial surface of the tumor is usually smooth, often described as pearly, with fine telangiectatic vessels beneath it. Ulceration may develop and is filled with a crusty exudate.The pigmented BCC is usually nodular or nodulo-ulcerative, ranging in color from light tan to deep brown.The cystic BCC may attain significant dimensions. The infiltrating BCC presents as an indurated, yellowish to tan patch or plaque with occasional focal ulceration and poorly defined margins.[1,7] On clinical feature our patient show solitary ulcer and with irregular shape, hyperpigmentation and venectation on the peripheral ulcer. From the appearance it looks like the infiltrating BCC and from the pathological examination also concluded that was a nodular infiltrative type.

There are several managements of eyelid BCC, but the most important thing we have to emphasize is to prevent recurrence rate.[1] Some studies reported the recurrence rates due to the underestimation of the subclinical extension of BCC are between 5% to 100%.[3] This varies according to the therapeutic method used. Tumors that recur tend to be more aggressive and difficult to manage.[7] The successful management of BCC requires an appreciation of its broad clinical expression and knowledge of its biologic behavior. Therapeutic outcome depends on many variables, the most important of which are the location, size, and growth pattern of the tumor, and the immune status of the patient. The goals of therapy for periocular BCC are threefold: 1) to completely eradicate the tumor; 2) to maintain the integrity and visual function of the eye; and 3) to achieve a good cosmetic result.[1]

The main treatment modality for BCC is surgical excision of the lesion with microscopic monitoring of its margins, for instance Mohs microsurgery. The other surgical and non-surgical modalities include standard frozen section, curettage and electrodessication, cryosurgery, radiotherapy, chemotherapy, photo-dynamic therapy, and immunotherapy.[4,7,8,15]

Selection of the appropriate therapy depends on the patient's age, anticipated life expectancy, cosmetic considerations, skill and the preference of the physicians, and the location, size, and pattern of growth characteristics of the tumor. [4,7,8] However, with no using surgical therapies or not include microscopic monitoring should be avoided for BCCs when they are not very small, when they are located in the medial canthus, or when the margins are clinically ill defined.[7] The morpheaform or sclerosing basal cell carcinoma is particularly resistant to nonsurgical forms of therapy.[6] The Randomized control trial study from Avril et all[8] reported that surgery proved to be superior to radiotherapy in treatment efficacy and cosmetic result. Thus, for untreated BCC of the face of less than 4 cm, surgery is recommended as first-line treatment.[8]

In order the medium-sized lesions, between 20-50 mm, and for those that are very extensive and difficult to resect, Jonathan stated that radiotherapy offers a good alternative to surgery, yielding a better cosmetic and functional result, with only a slightly higher failure rate.[3,14] For all recurrent tumors or high risk of recurrence tumor and deeply infiltrative tumors, regardless of size, Mohs' surgery with histologic control of margins is mandatory, and radiotherapy is not appropriate, unless the tumors are deemed unresectable.[3,4,6,7,14] Using standard frozen sections are not consider for our patient due to the high risk of recurrence because this technique is unable to examine the entire surfaces of excised tumor to prove that excision has been complete.[4,6]

Mohs Micrographic surgery (MMS) is a technique for the removal of complex or ill-defined skin cancer with histologic examination of 100% of the surgical margins.[7] This technique consideration based on Appropriate Use Criteria (AUC) document from the American Academy of Dermatology (AAD), American College of Mohs Surgery, American Society for Dermatologic Surgery Association, and American Society for Mohs Surgery reflects an ongoing effort to systematically review and categorize the appropriate use of MMS.[16] The AUC make the indication of MMS technique are based on the location and aggressiveness of the tumor and the patient characteristics (table 1).[16,17]

The locations were divided into three areas: 'H' area (''Mask areas'' of face, eyebrows, nose,

lips, chin, ear, genitalia, hands, feet, nail units, ankles, and nipples/areola), 'M' area (Cheeks, forehead, scalp, neck, jawline, pretibial surface) 'L' area (Trunk and extremities (excluding pretibial surface, hands, feet, nail units, and ankles).[16]

Type of aggressive features are morpheaform/fibrosing/sclerosing, infiltrating, perineural, metatypical/keratotic and micronodular BCC.[16] The patient characteristics should be consider about immune status, genetic syndrome, history of prior radiation and chronic infection.[16] From the AUC criteria our patient cancer was located on the 'H' area with nodular infiltrative (aggressive) feature and the size were almost 2 cm. This condition lead us to use the MMS for the treatment.

In five-year cure rate for primary BCC, MMS have the highest percentage in treatment modality, which is 99% compared with surgical excision, electro-desiccation and curettage, radiation, cryotherapy, and all non-Mohs modalities, which are 90%, 92%, 91%, 92%, 91% respectively.[15,17,18]

After the MMS had done, the next management is how close of eyelid defect. The goals of eyelid reconstruction are to provide structural and functional restoration with an acceptable aesthetic result.[9,10,12] Mohs' fresh tissue technique of tumor removal followed by eyelid reconstruction may be the most efficacious management of eyelid malignancies. The advantages of this approach are (1) a high cure rate, (2) conservation of tissue, (3) reduced operating room time, and (4) maximum utilization of specialty training.[6,17] For reconstructive purposes, the eyelid may be divided into two lamellae: the anterior lamella which consists of the skin and orbicularis oculi muscle, and the posterior lamella which consists of the tarsal plate and the conjunctiva. Both of these lamellae must be replaced in the repair of full-thickness defects to satisfy their functional requirements.[10]

The anterior lamella may be reconstructed with advancement or rotation myocutaneous flaps or full-thickness skin grafts. Reconstruction of the posterior lamella may be performed by tarsal transposition or rotation flaps, free autogenous tarsal grafts, sliding tarsoconjunctival flaps, or tarsal substitutes including preserved sclera, auricular cartilage, nasal septal chondromucosa, and hard palate mucosa. The lid reconstruction may be divided into three main groups; partial thickness lid defects not involving the lid margin, full thickness lid defects involving the lid margin and medial canthal defects. In full thickness lid defect requires three elements, which are outer layer of skin, inner layer mucosa, semi-rigid supporting structure (acting as the tarsal plate) interposed between them.[10]

There are many considerations due to this condition, for instance likes the size or number of tissue loss, location, state of remaining ocular tissue, visual status of the fellow eye, age and general health condition, and the other structures involve of the eyelid defect.[5] Study from Anderson and Ceilley reported[6] that in Mohs surgery the actual tumor mass excised was about 4 times larger than the area of the clinically apparent tumor. For lid reconstruction, the horizontally full-thickness marginal defects are classified into: small (<25% of the horizontal dimension of the lid margin), medium (25%-50%), and large (> 50%).[10] The other dimension that we have to consider in large defect is the vertical defect size. This is also classified into size 5 – 10 mm, 10 – 15 mm and more than 15 mm.[19]

**Table 1.** Indication table of Mohs Surgery based on Appropriate Use Criteria (AUC). Appropriate use scores and final ratings for 69 BCC indications. Appropriate indications (A; scores 7-9) are colored green; Uncertain indications (U; scores 4-6) are colored yellow; Inappropriate indications (I; scores 1-3) are colored red.[16]

| B. Primary aggressive BCC (healthy or immunocompromised patients) | | | | |
|---|---|---|---|---|
| | | Appropriate use score (1-9) | | |
| Indication | Size, cm | Area H | Area M | Area L |
| 4 | ≤0.5 | A (8) | A (8) | U (6) |
| 5 | 0.6-1 | A (9) | A (8) | A (7) |
| 6 | 1.1-2 | A (9) | A (9) | A (8) |
| 7 | >2 | A (9) | A (9) | A (8) |

There are a number of surgical procedures that can be used to reconstruct the lower eyelid defect, such as direct close with or without canthotomy and cantholysis, semicircular flap (Tenzel flap), the upper lid tarsoconjunctival pedicle flap (Hughes' flap), Free tarsoconjunctival graft, Periosteal flap, Mustarde cheek rotation flap.[5,20,19] In general, where the defect is small, direct closure of the eyelid is possible (figure 9a). Where the eyelid tissues are very lax, like in the older patient, direct closure may be possible for much larger defects occupying up to 50% of the eyelid.[5,9,10,12,19]

**Figure 9.** A) Illustration of the skin closure with the eyelid margin suture held in position away from the cornea; B) With simple lateral canthotomy and cantholysis; C) Tenzel semi-sircular flap[20,21]

If the defects is between 35 to 50% or medium size length of the eyelid and there was no laxity, direct closure on the wound is difficult, we can do a simple lateral canthotomy and cantholysis of the appropriate limb of the lateral canthal tendon, which is can effect a simple closure (Figure 9b).[5,9,19] But if this technique is not sufficient, it should be combined with a local periorbital skin and muscle flap or Tenzel semicircular flap.[19] (figure 9c).

In case the horizontal defect more than 50% up to 100% we should combine with a local periorbital skin and muscle flap. In this condition we have to divided into which lamella of the eyelid were loss. In case of posterior lamella loss, hughes' tarsoconjunctival flap, chondomucous grfat nasal septum and palatal mucoperiosteal flap can be used. With the vertical defect between 5-10 mm, hughes' tarsoconjunctival flap (figure 10a) or skin flap with lips mucous graft can be used for posterior lamella loss.[10,19,22]

In case of anterior lamella loss, Mustarde cheek advancement flap, Full thickness skin graft, Tripier or bipedilcle muscle flap, Nasolabial flap, Median forehead flap, Lateral temporal flap can be used. If the vertical defect more than 15 mm we can use Mustarde cheek advancement flap technique (figure 10b).[10,19,22] After tumor removal, the lower eyelid defect were not involving the margin, the posterior lamella and the inferior lacrimal punctum. However due to the defect size were more than 50% horizontally and more than 15 mm vertically, we decided to close with mustarde cheek advancement flap. This methode is a very useful flap for reconsruction in larger defect of the lower eyelid especially in long vertical defects.[22]

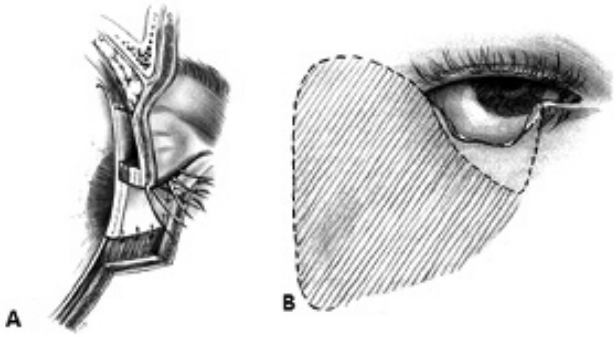


**Figure 10.** A) Hughes procedure, tarsus is incised 4 mm above margin and advanced inferiorly to close to the conjunctiva and retractors inferiorly, the anterior defect is closed with skin flap or graft; B) Mustarde cheek advanced flap, dotted line beneath lid is additional tissue to be removed[21]

This technique starts with marked lateral to defect extending to the lateral canthal area.[19,21,22] It should curve upward and carried down in front of the ear and then inferiorly over the jaw.[21,22] The entire flap is extensively undermined until the subcutaneous layer but not more than subcutaneous fat tissue to avoid the facial nerve.[19,22] Stiches the skin by interrupted suture technique with prolene 6-0 or silk 6-0 then pressed it with bandage.[19] We started the surgery with made a marked from the upper lateral side of the wound and pass through the lateral canthus then make a curvilinear line pass the ear tragus until ear lobe then made an subcutaneous undermined as far as the same level of the ear lobe to make a flap. After the flap had been made, we closed it with interrupted suture.

One week after surgery we found the nectrotic flap on the area of suturing. This condition can happen due to two factors, intrinsic and extrinsic factors.[23] The cause of intrinsic factors is ischemic condition, which is occurs mainly in the distal portion of free flaps.[23,24] The clinical problem the ischemic necrosis occurs in

5-10% of patients, even in experienced hands. The general consensus is that vasospasm and thrombosis due to surgical trauma and insufficient distal vascularity are the main pathogenic factors in flap failure.[24] Under physiological conditions, a balance of vascular effects between endothelium-derived relaxing factors (EDRFs) which can cause relaxation of vascular smooth muscle and endothelium-derived contracting factors (EDCFs) which can cause raise vascular tone maintains adequate tissue perfusion. However, an imbalance can occur as a result of surgical trauma.[23]

Specifically, traumatized sympathetic nerve endings release norepinephrine (NE), causing vasoconstriction especially in the small arteries in the distal portion of the flap where the perfusion pressure is low and the concentration of these vasoconstrictive substances is high due to the downstream effect. Hemoglobin from hemolyzed red blood cells (e.g., hematoma) is also a potent vasoconstrictor. Furthermore, the synthesis and release of EDRFs such as PGI2 and NO from the traumatized vascular endothelium are depressed. The end result is that there are high local levels of vasoconstrictive and prothrombotic neurohumoral substances in surgical trauma and these substances exacerbate vasospasm and promote thrombosis in flap surgery.[24]

Specifically, in sustained ischemia, mitochondrial ATP synthesis ceases and glycolysis ensues, resulting in a net breakdown of ATP and an accumulation of lactate and intracellular H+, causing intracellular acidosis. This build-up of intracellular H+ activates the Na+/H+ exchange isoform-1 (NHE-1) antiporter, resulting in extrusion of H+ and accumulation of intracellular Na+ to restore intracellular pH. There is a further increase in intracellular Na+ accumulation because Na+ extrusion is limited by inactivation of the energy-dependent Na+-K+-ATPase pump. Elevation of intracellular Na+ concentration causes an increase in intracellular Ca2+ by activation of the Na+/Ca2+ exchanger causing Ca 2+ influx. If these events continue, the cystolic Ca2+ will be overloaded, and significant uptake of Ca2+from the cytosol to the mitochondria will occur, resulting in mitochondrial Ca2+ overload which causes depolarization of mitochondria and impairs ATP synthesis, resulting in cell necrosis (figure 11).[24] When she came for follow up, in one week after surgery, she said that she not take a total bed rest but still worked as the housemaid since one day after surgery. This condition could influence the wound healing processes, due to the break down of more ATP.

The extrinsic factors are systemic hypotension, infection, and patient lifestyle. [23] From physical exam there were no history of systemic hypotension during follow up, and there were no history of diabetes mellitus. The infection should be considered in the pathogenesis of necrotic flap in this patient. Six weeks after surgery, we found the yellowish discharge like a pus from the flap. this condition could be sign of infection.

Smoking tobacco is associated with an increased chance of flap necrosis in facelift operations.[24,25,26] The mechanism by which tobacco or nicotine decreases flap survival is unknown but may involve direct endothelial damage, vasoconstriction caused by catecholamine release, or local concentrations of prostaglandins. These effects could be avoided if the nicotine was withdrawn 2 weeks before the flap was raised.[24] However, from anamnesis, there were no history of smoking tobacco.

Other causes include late evacuation of hematoma and wide undermining of the skin flaps with wound closure under tension. A tight flap can caused inadequate blood reaches the skin cells and cause the necrosis flap.[26] However, this cheek rotation flap procedure has disadvantage of producing an inferior vector of traction during healing and the possibility of facial nerve damage.[21] The tight flap might became the etiologic factor of the skin necrosis in this patient. After the reconstruction we found the flap looks tight if we compared with the contralateral face skin.

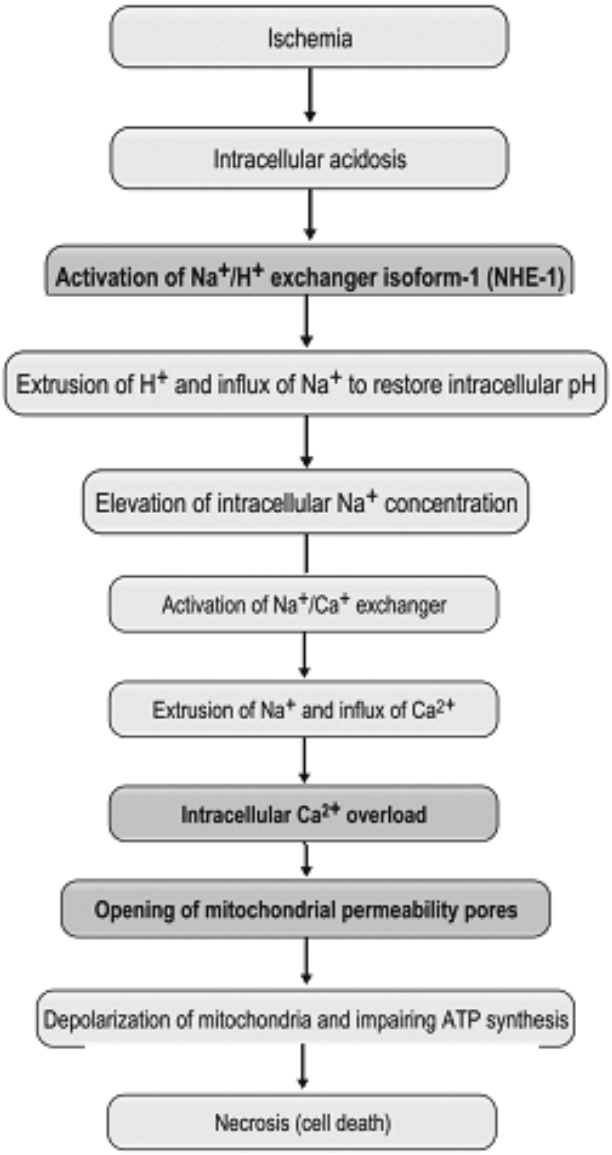


**Figure 11.** Sequence of necrosis tissue[24]

From DV department they used the diode laser or Low Level Laser Therapy (LLLT) to enhance wound healing process. Numerous case reports and clinical trials with humans have shown impressive wound healing outcomes using LLLT taken from no.27. The exact mechanism by which LLLT facilitates wound healing is largely unknown. However, several theories may help explain the enhanced wound contraction observed here. In vitro studies have shown an increase in fibroblast proliferation after irradiation, suggesting that LLLT therapy may facilitate fibroplasia during the repair phase of tissue healing.[27,28]

The basic principle mechanism of LLLT is using the electromagnetic radiation wave. There were two types of light that might be used, blue and red light. Both of this light have two functions, first is antibacterial agent and second is anti inflammation agent.[29] The red light is also can induce fibroblast activation and proliferation.[30] Diode laser that we use, has combination between blue and red light. It is proposed that synergistic effects of mixed light is due to synergy between the antibacterial and anti-inflammatory effect of blue and red light respectively.[31]

## CONCLUSION

The management for eyelid BCC were still challenging due to the risk of recurrence after the tumor removal. Mohs micrographic surgery offers the dual advantages of maximal cure rate and maximal preservation of normal tissue. With the preservering procedure during the surgery and follow up after the surgery, this condition can be managed succesfully. For this reason if there were a problem with the flap after surgery, the colaboration between the Ophthalmologist and the Dermatovenereologist were suggested.

## REFERENCES

1. Kersten RC, Ewing-Chow D, Kulwin DR, Gallon Margo CE, Waltz K. Basal Cell Carcinoma of the Eyelid and Periocular Skin. *Surv Ophthalmol* 1993; 38: 169-92.
2. Ormerod A, Rajpara S, Craig F. Basal cell carcinoma. *British Medical Journal* 2010; 4(1719): 1-34.
3. Jonathan J. Management Of Periocular Basal Cell Carcinoma.*Surv Ophthalmol* 1993; 38(2): 210-2.
4. NCCN Clinical Practice Guidelines in Oncology. *Basal Cell and Squamous Cell Skin Cancers*. National Comprehensive Cancer Network. Report number: 1, 2007.
5. Leatherbarrow B. Tumour management and repair after tumour excision. In: Collin R, Rose G (eds.)Fundamentals of Clinical Ophthalmology Plastic and Orbital Surgery. 1st ed. London: BMJ Books; 2001. p44-66.
6. Anderson RL, Ceilley RI. A Multispecialty Approach To The Excision And Reconstruction Of Eyelid Tumors. *Ophthalmology*1978; 85(11): 1150-63.
7. Rosner M. Basal cell carcinoma. In: Singh AD, Damato BE, Pe'er J, Murphree AL, Perry JD (eds.)Essentials of Ophthalmic Oncology. 1st ed. United State of America: Slack Incorporated; 2010. p76-80.
8. Avril MF, Auperin A, Margulis A, Gerbaulet A, Duvillard P, Benhamou E, et all. Basal cell carcinoma of the face: surgery or radiotherapy? Results of a randomized study. *British Journal of Cancer* 1997; 76(1): 100-6.
9. Ahmad J, Mathes DW, Itani KM. Reconstruction of the Eyelids after Mohs Surgery. *Semin Plast Surg* 2008; 22: 306-18.
10. Abdallah AM. Reconstruction of Eyelid Defects after Excision of Eyelid Tumors. *Saudi Journal of Ophthalmology* 2006; 20(3): 196-204.

11. M. Accuracy of Clinical Diagnosis of Cutaneous Eyelid Lesions. *Ophthalmology* 1997; 104: 479-84.
12. Rene C. Oculoplastic aspects of ocular oncology. *Eye* 2013; 27: 199-207.
13. Jahan-Tigh RR, Migden MR. *Incorporating Treatment Advances for Basal Cell Carcinoma Into Clinical Practice. http://www.medscape.org (accessed 12 December 2014).*
14. Cigna E, Tarallo M, Maruccia M, Sorvillo V, Pollastrini A, Scuderi N. Basal Cell Carcinoma: 10 Years of Experience. *Journal of Skin Cancer* 2011: 1-5.
15. Dhingra N, Gajdasty A, Neal JW, Mukherjee AN, Lane CM. Confident complete excision of lid-margin BCCs using a marginal strip: an alternative to Mohs' surgery. *Br J Ophthalmol* 2007; 91: 794-6.
16. Connolly SM, Baker DR, Coldiron BM, Fazio MJ, Storrs PA, Vidimos AT. AAD/ACMS/ASDSA/ASMS 2012 appropriate use criteria for Mohs micrographic surgery: A report of the American Academy of Dermatology, American College of Mohs Surgery, American Society for Dermatologic Surgery Association, and the American Society for Mohs Surgery. *J Am Acad Dermatol* 2012; 67(4): 531-50.
17. Jean L. Bolognia, MD, Joseph L. Jorizzo, MD and Julie V. Schaffer, MD. Mohs Surgery. In: Bolognia JL, Jorizzo JL, Schaffer JV (eds.)Dermatology. 3rd ed. England: elsevier; 2012. p2445-55.
18. Inkster C, Ashworth J, Murdoch JR, Montgomery P, Telfer NR, Leatherbarrow B. Oculoplastic reconstruction following Mohs surgery. *Eye* 1998; 12: 214-8.
19. Sub-bagian Plastik Mata dan Rekonstruksi. *Prosedur Diagnostik Bedah Plastik Mata dan Rekonstruksi*. Fakultas Kedokteran Universitas Indonesia. Report number: 1, 2010.
20. Long JA. Mohs reconstruction. In: Roy FH, Benjamin L (eds.)Surgical Techniques in Ophthalmology: Oculoplastic Surgery. 1st ed. England: Saunders Elsevier; 2009. p111-25.
21. Jackson IW, Pacheco EM. *Flaps, Grafts, and Alloplastic Materials in Lid Reconstruction and Repair.* Pennsylvania: Lippincott Williams & Wilkins; 2006. http://www.eyecalcs.com/ (accessed 29 January 2015).
22. Subramanian N. Reconstructions of eyelid defects. *Indian J Plast Surg* 2011; 44(1): 5-13.
23. Arnold MG, Chu EA, Odland RM. Skin Flap Physiology and Wound Healing. In: Flint PW, Haughey BH, Lund V, Niparko JK, Robbins KT, Thomas JR, et al (eds.)Cummings Otolaryngology. 6th ed. Philadelphia: Saunders Elsevier; 2015. p1124-36.
24. Pang CY, Neligan PC. Flap pathophysiology and pharmacology. In: Neligan PC (eds.)Plastic Surgery Six Volumes. 3rd ed. Canada: Elsevier; 2013. p573-86.
25. Weissman O, Farber N, Remer E, Tessone A, Trivizki O, Bank J, et al. Post-facelift flap necrosis treatment using charged polystyrene microspheres. *Can J Plast Surg* 2013; 21(1): 45-7.
26. Gladstone G, Myint S, Black EH, Brazzo BG, Nesi FA. Fundamentals of Facelift Surgery. *Ophthalmol Clin N Am* 2005; 18: 311-7.
27. Hopkins JT, McLoda TA, Seegmiller JG, Baxter D. Low-Level Laser Therapy Facilitates Superficial Wound Healing in Humans: A Triple-Blind, Sham-Controlled Study. *Journal of Athletic Training* 2004; 39(3): 223-9.
28. Inck EM, Cagnie BJ, Cornelissen MJ, Declercq HA, Cambier DC. Increased fibroblast proliferation induced by light emitting diode and low power laser irradiation. *Lasers Med Sci* 2003; 18: 95-9.
29. Shnitkind E, Yaping E, Geen s, Shalita AR, Lee WL. Anti-inflammatory properties of narrow-band blue light. *J Drugs Dermatol* 2006; 5: 605-10.
30. Barlow RJ, Hruza GJ. Laser and light tissue interaction. Goldberg DJ. In: Laser and Light. 1st ed. Philadelphia: Elsevier; 2005. p1-10.
31. Avci P, Gupta A, Sadasivam M, Vecchio D, Pam Z, Pam N, et al. Low-level laser (light) therapy (LLLT) in skin: stimulating, healing, restoring. *Semin Cutan Med Surg* 2013; 32(1): 41-52.