PATTERN OF OCCURRENCE, CLINICAL PRESENTATION AND MANAGEMENT OF ORAL AND MAXILLOFACIAL VASCULAR LESIONS AT MUHIMBILI NATIONAL HOSPITAL

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A dissertation submitted in (Partial) fulfillment of the requirement for the Degree of Master of Dentistry (Oral and Maxillofacial Surgery) of

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CERTIFICATION

The undersigned certify that they have read and hereby recommend for acceptance by Muhimbili University of Health and Allied Sciences a dissertation titled; **"*Occurrence and management of Oral and Maxillofacial vascular lesions at Muhimbili National Hospital*"** in partial fulfillment of the Requirement for the Degree of Master of Dentistry in Oral and Maxillofacial Surgery of Muhimbili University of Health and Allied Sciences.

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Date_____

DECLARATION AND COPYRIGHT

I **Dr. Abbas Mungia**,declare that this **dissertation** is my own original work and that it has not been presented and will not be presented to any other University for a similar or any other degree award.

Signature……..…… Date…….

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DEDICATION

I would like to dedicate this dissertation to:

My late parents who nurtured and guided me tirelessly

My best friend and lovely wife Fatema, for her endless love, dedication and support

My children Isihaka, Abra and Asra from whom I drew strength, confidence and happiness

ABSTRACT

Background: Vascular lesions are anomalies of vascular development and endothelial cell hyperplasia. They are best diagnosed through history and clinical examination. These lesions present with physical and psychological implications to the patients particularly if visibly disfiguring. Magnitude of these vascular lesions is not known in Tanzania likewise the demographic distribution. Treatment of these lesions is difficult due to lack of expertise and modern medical facilities, and this has led to an increased burden of untreated patients in Tanzania.

Overall objective: To determine pattern of occurrence, clinical presentation and management of oral and maxillofacial vascular lesions at Muhimbili National Hospital.

Methodology: This was a descriptive prospective cross-sectional hospital-based study that was conducted at the departments of Oral and Maxillofacial Surgery, Pediatrics, Oncology, and Ear Nose and Throat (ENT).All patients with oral and maxillofacial vascular lesions were interviewed using specially designed structured questionnaires and clinical examination forms. The interview enquired about socio-demographic information, site where vascular lesion was, when it was noticed, treatment before and current complaints. Later, the patients were clinically examined whereby the details of the examination included clinical presentation, type and site of the lesion. Radiological evaluation of some cases with extensive lesions included CT scan, CT angiography, MRI, MR angiography and Doppler ultrasound. Data collected were recorded in special clinical forms, checked for completeness and clarity, coded and later were entered into the computer for analysis using SPSS version 20. Descriptive analysis involved computation of percentages, frequency of occurrence, mean and cross tabulations of variables of interest. Inferential analysis included computation of Chi-Square test to compare proportion for possible association. A p-value of <0.05 was used as a cut off level for significance.

Results: A total of 102 patients with vascular lesions involving the oral and maxillofacial region were enrolled in this study. Forty nine (48.0%) were males while 53 (52.0%) were females with a male-to-female ratio of 0.9:1. The age of the patients ranged from 4 months to 101 years. Almost 65(63.7%) two thirds of these patients were aged 0-10 years.

Occurrence of the vascular lesions appeared almost two thirds 65(63.7%) at the age group 0-10 years whereby 33(50.8%) hemangioma and 30(46.2%) lymphangioma patients constituted significant proportion (p=0.003). Among patients with hemangioma there were more (58.6%) females compared to (41.4%) males with the ratio of 1.4:1.Fifty one (50%) patients had vascular lesions occurred at birth of which 29(56.2%) were lymphangioma. Majority 25(86.2%) of patients with Infantile hemangioma (IH) occurred in the 1st to 6th months of all lesions together after birth (p=0.000). The most frequently affected site in oral and maxillofacial regions by lymphangiomas were 18(72%) submandibular, 14(43.8%) tongue and 13(76.5%) cervical regions. Hemangiomas involved the lips 32(69.6%), 22(44%) cheeks, 22(56.4%) buccal mucosa and 15(65.2%) labial mucosa.

Sixty two (60.8%) patients had sclerotherapy, 21(20.6%) intervened surgically, 16(15.7%) sclerotherapy and surgery while 3(2.9%) were on observation. Forty two (41.2%) patients had their lesions resolved completely, 51(50%) patients had good response and were still going on with treatment. Nine (8.8%) had poor response and required advanced expertise (p= 0.000).

Conclusion: Vascular lesions were common in childhood particularly hemangioma which is also more common in females than males. Majority of lymphangiomas were found at birth. Swelling was the most presented clinical feature. Cheeks, lips, buccal mucosa, and tongue were most affected anatomical sites by all vascular lesions. Sclerotherapy by bleomycin found to be effective and efficient for vascular lesions particularly hemangioma and lymphangioma.

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LIST OF ABBREVIATIONS

AVM	:	Arteriovenous malformation
CH	:	Congenital Hemangioma
CT	:	Computed Tomography
CTA	:	Computed Tomography Angiography
DDS	:	Doctor of Dental Surgery
DNA	:	Deoxyribonucleic Acid
DIB	:	Difficult in breathing
ENT	:	Ear Nose and Throat
ESRK	:	Extracellular signal Regulated Kinase
FNAC	:	Fine Needle Aspiration Cytology
GLUT	:	Glucose Transporter
IH	:	Infantile Hemangioma
IBI	:	Intralesional Bleomycin Injection
MAPK	:	Mitogen-activated Protein Kinase
MDent	:	Master of Dentistry
MNH	:	Muhimbili National Hospital
MRA	:	Magnetic Resonance Angiography
MRI	:	Magnetic Resonance Imaging
MUHAS	:	Muhimbili University of Health and Allied Sciences
MVM	:	Multifocal Venous Malformations
OMFS	:	Oral and Maxillofacial Surgery
PKA	:	Protein Kinase A
SPSS	:	Statistical Package for Social Science
VEGF	:	Vascular endothelial growth factor

CHAPTER ONE

1.0 INTRODUCTION AND LITERATURE REVIEW

1.1 BACKGROUND

Vascular lesions comprise of vascular tumours and vascular malformations. Vascular malformations are congenital lesions that results from abnormal vascular development occurred during embryogenesis or morphogenesis. Vascular tumours are not true neoplasm and consist of hemangiomas which are caused by hyperplasia of vascular endothelial cells (Richter and Friedman, 2012).

Vascular malformations are classified according to types of blood vessels involved, thus can be lymphatic, capillary, venous and arteriovenous. In 1982, Mulliken and Glowack published a biological classification of vascular lesions based on endothelial characteristics, histology and clinical presentation (Duncan, 2004). Due to differences in biologic and radiographic behavior, malformations are further divided into slow-flow and fast-flow lesions. Low-flow malformations contain combinations of capillary, venous, and lymphatic component. High flow malformations contain arterial components in combination with other vascular structures (Lowe *et al.*, 2012).

Classification of vascular lesions

Vascular tumors	**Vascular malformations**
	Slow-flow
Infantile hemangioma	Capillary malformations
Congenital hemangioma	Venous malformations
Tufted angioma	Lymphatic malformations
Kaposiform hemangioendothelioma	**Fast-flow**
	Arteriovenous malformations

Modified from(Richter and Friedman, 2012).

Hemangiomas can either be infantile or congenital. Infantile Hemangiomas (IHs) are more common (70%) than congenital hemangioma (CH) 30% (Syed, 2016).

These lesions are complicated by ulceration mostly, hemorrhage, rapid growth when infected or in puberty and pregnancy, trauma, bleeding and pain (Zheng *et al.*, 2013). Other complications include airway obstruction, ophthalmic involvement, impact on speech, dysphagia, suffocation, congestive heart failure and death due to bleeding (Redondo, 2007). They may cause severe psychological distress or disfigurement and at that moment patients may warrant further discussion and consideration for treatment. The psychological impact should be considered at each stage of child's clinical management (Redondo, 2007, Zheng *et al.*, 2013, Mahady *et al.*, 2015).

Diagnosis of these vascular lesions is best made by clinical history and physical examination (Grasso *et al.*, 2008). In cases of unclear diagnosis or extensive lesions radiographic modalities are used including; MRI, CT scan, Doppler Ultrasound, CT or MRI angiograph (Greene and Orbach, 2011, Behravesh *et al.*, 2016). Fine needle aspiration cytology (FNAC) and open biopsy should not be requested for vascular lesions unless history and clinical examination doesn't confirm the diagnosis. This is reasonable because the specimen would mostly contain blood cells or lymphatic fluid (Mohamad *et al*., 2014). Glucose transporter 1 (GLUT 1) form a diagnostic marker of infantile hemangioma through immunostaining in contrast to congenital hemangioma which is histological GLUT1 negative (Buckmiller *et al*., 2010). These lesions can occur in any part of the body. Magnitude of these lesions is not known in our community likewise the demographic distribution. Prevalence of these lesions is important to know for future resource allocations and plan for their management. Multidisciplinary management approach has been advised for diagnostic accuracy and treatment plan (Bodra *et al*., 2016).

Treatments of these vascular lesions involve different methods such as observation or watchful neglect, use of steroids, chemotherapy, immunotherapy, pharmacotherapy, surgery, compression dressing, embolization, radiatiotherapy, cryotherapy, sclerosing agents and laser (Nthumba, 2013).

1.2 LITERATURE REVIEW

Pattern of occurrence of vascular lesions

Worldwide studies on the occurrence of infantile hemangioma have found less frequently in Japanese (0.8%), African-Americans (1.4%), and Asians (3%) (Pocock *et al*., 2006). A 10 years retrospective study was done in Brazil to estimate the prevalence of oral hemangioma and vascular malformation, a total of 2,419 clinical cases were evaluated and found that; oral hemangioma was diagnosed in 22 cases (0.9%) and oral vascular malformation in 31 cases (1.3%) (Corrêa *et al.,* 2007). In Jordan a retrospective study on oral and maxillofacial tumors among children and adolescents over 10 year found hemangioma was the most common benign tumor (Al-khateeb *et al.,* 2003*).*In Japan a retrospective study for 28 years showed hemangioma was the most common vascular lesion by 22% in children under the age of 15 years out of 2,747 patients (Sato *et al*., 1997).

A case control study was done in Dutch population to determine the prevalence of infantile hemangiomas, involved newborns at the age of 0-16 months among 2,204 cases, it showed that 219 (9.9%) of the newborns had hemangioma (Hoornweg *et al*., 2011). A Study in Nigeria on morphologic patterns of vascular tumors a 12 year retrospective review showed that hemangioma was the most frequently encountered tumor in the series by 71% of all vascular tumors in a total of 162 vascular tumors (Obaseki *et al.*, 2013). In Tanzania vascular lesions seen among 33 treated patients showed hemangioma was the commonest vascular lesion by 57.6% followed by lymphangioma in 36.4%. Cystic hygroma and syndromic vascular lesions were found less frequently (Moshy *et al.*, 2011).

Lymphatic malformation can be found at any age of life, approximately 50% are present at birth and 90% are diagnosed before or within 2 years of age (Zhou *et al.*, 2011). The incidence of lymphatic malformations is 1.2–2.8%.Their incidence is approximated to be 1 in 2000 to 4000 live births (Richter and Friedman, 2012).

A 6 years study on congenital vascular malformations among a total of 797 cases showed lymphatic malformations to be common at 39.5%(Lee *et al,* 2005). Worldwide capillary malformation(CM) occurs in 0.1-2% of the population (Orme *et al.,* 2013). It is more

common in whites than African Americans. The incidence of venous malformation is approximately 1:5,000-10,000; approximately 40% of them occur in the head and neck regions (Zheng *et al.*, 2013).

True prevalence of arteriovenous malformation (AVM) remains elusive and roughly estimated according to autopsy and population-based studies whereby approximately 1 per 100,000 person-years is an estimated prevalence with >50% involving the head and neck (Nassiri *et al.*, 2015). In the Netherlands Antilles, an annual incidence of symptomatic AVMs of 1.1 per 100,000 was reported between 1980 and 1990 (Jessurun *et al*., 1993).

Demographic distribution

Infantile hemangioma (IH) is the most common vascular tumor of childhood and is much more in females than males with a F:M ratio of = 3:1. Studies have shown that inheritance of infantile hemangioma can occur at least by two possible mechanisms; autosomal dominance and maternal transmission (Lowe *et al.*, 2012). When they occur, the most common location of hemangiomas is the head and neck which accounts for 60% of all cases (Syed, 2016). Majority (80%) of these lesions occur as single (focal) lesions, however, 20% of affected patients will have multiple (multifocal) lesions, and when they are > 5 suspicion of visceral involvement is to be thought, and complications may occur in about 20% of non-syndromic hemangiomas (Neville *et al.*, 2002). Segmental hemangiomas are more diffuse, extensive involving broad anatomic regions and may lead to significant morbidity. Patient with segmental hemangioma has to undergo investigations to rule out some syndromes including PHACES and Kasabach-Merrit. PHACE syndrome refers to posterior fossa brain abnormalities, hemangiomas, arterial malformations, coarctation of aorta and other cardiac defects, as well as eye abnormalities (Syed, 2016).

Conversely Kasabach-Merrit syndrome is a serious coagulopathy that has been associated with huge or extensive hemangiomas in infants, characterized by severe thrombocytopenia and hemorrhage because of platelet trapping within the tumor. The mortality rate is as high as 30% to 40 % (Neville *et al*., 2002).

An international observation cross-sectional study on infantile hemangiomas involving 693 individuals in five countries showed more girls than boys (66%-83% female) with 70% of cases with lesions affecting the head (Cazeau *et al.*, 2017). A recently register based study conducted in Minnesota estimated infantile hemangioma is more common in Caucasian at a frequency of 3.8% in infants aged 0 to 3 and male to female ratios vary from 1:1-3 (Castrén *et al*., 2016). Infantile hemangioma (IH) arises during the first 8 weeks of life with high proliferative phase for 6–12 months. This is followed by a gradual involution phase and a spontaneous regression by the age of 5–9 years (Lowe *et al.*, 2012). Oral mucosa and skin are most commonly affected followed by bone and muscles within the maxillofacial region (Mohan and Prasad, 2014). These appear as flat or raised deep blue hue or tint in color over the surface of the mucosa. Superficial lesions appear bright red and bosselated, firm, rubbery in consistency and blood cannot be evacuated on pressure application (Neville *et al.*, 2002).

Among infants with hemangioma multiple lesions may appear approximately 14% to 20%. They may involve other organ systems, such as the liver, gastrointestinal tract, and brain. Skeletal deformities are very rare with hemangiomas, infrequently a ‘‘mass effect’’ may occur on adjacent bone, or bony overgrowth, presumably due to local increased blood flow (Spring and Bentz, 2005).

Pathogenesis of infantile hemangioma is not known, it has been suggested by theories; intrinsic and extrinsic. The intrinsic theory suggests mutation in a critical gene in a precursor stem cell and that the clonal expansion of this single cell carrying a somatic mutation leads to hemangiomagenesis. Extrinsic theory suggests, growth factors, an abnormal hormonal milieu like increased estrogen levels, or tissue hypoxia with expression of hypoxia inducible factor (HIF1 α) and the stimulation of angiogenesis via vascular endothelial growth factor (VEGF), underlie hemangioma development (Mabeta and Pepper, 2011).

Lymphatic malformation affects males and females in equal numbers. However in some cases lymphatic malformation may not become apparent until adulthood. They are most common on the face particularly along the distribution of the trigeminal nerve, clinically macrocystic lesions occur in the neck with microcystic lesions in the tongue or cheek (Neville *et al*.,2002).

The most common site is the head and particularly the neck (90%).Other typical sites are the axilla, thorax, mediastinum, retroperitoneum, buttocks, and anogenital region (Redondo, 2007). Three theories have been proposed to explain the origin of this abnormality. The first suggests that a blockage or arrest of normal growth of the primitive lymph channels occurs during embryogenesis, the second that the primitive lymphatic sac does not reach the venous system, while the third advances the hypothesis that, during embryogenesis, lymphatic tissue lays in the wrong area (Grasso *et al.*, 2008).

Capillary malformations (CMs) are typically multifocal, <1–3 cm in diameter, round or oval pink macules and patches distributed on the face, trunk, and extremities, although vast majority are solitary, CMs of up to 15 cm in diameter, and with red, brown, or grey color may also be seen(Redondo, 2007). They may be present at birth, and more may develop over time. Depending on size and location, they may cause significant morbidity due to stigmatization or disfigurement and, rarely, herald the presence of an underlying syndrome. Capillary malformation is equated with port wine stain, as it is most commonly referred in literatures. Capillary malformation is also known as nevus flammeus (Maguiness and Liang, 2011). Although they are mostly solitary lesions, CM may exist as part of a syndrome including Sturge Weber Syndrome (SWS) which is the most common, Macrocephaly capillary malformation (M-CM), capillary malformation- arteriovenous malformation syndrome (CM-AVM), cutis marmorata telangiectatica congenita (CMTC), and overgrowth syndromes, such as Klippel-Trenaunay syndrome (KT) (Maguiness and Liang, 2011). Pathogenesis of capillary malformation is unknown, however a genome wide linkage analysis has identified mutation of a locus on chromosome 5q associated with familial disease (Orme *et al.*, 2013).

An autosomal dominant disorder, capillary malformation arteriovenous malformation syndrome (CM-AVM), associated with inactivating mutations of the RASA1 gene, which encodes p120RasGAP, a Ras-GTPase-activating protein involved in cellular proliferation, migration, and survival. There is significant phenotypic variability in this disorder, even within affected families, and associated fast-flow lesions can be cutaneous, subcutaneous, intramuscular, intraosseous, intracerebral, or intraspina (Weitz *et al.*, 2015). Basic

transforming growth factor (TGF-b) and CCN2 (connective tissue growth factor, a matricellular protein of the CCN family of extracellular matrix- associated heparin-binding proteins (Zhu *et al.*, 2017).

Venous malformations (VMs) are common and majority of these malformations are sporadic and more commonly occur in the mouth, airway tract and muscle. No predilection exists for either sex(Neville *et al.*, 2002). Previous studies have revealed that the pathogenesis of VM may be closely associated with TIE2 mutation, disorganized vascular structures as well as dysregulation of related molecules, including Tie2 (tyrosine kinase receptor for angiopoietin-1, angiopoietin-2, and angiopoietin-4) (Zheng *et al.*, 2013).They are compressible blue masses present at birth which continue to grow in proportion with the body (Amato *et al.*, 2015).

Arteriovenous malformations (AVMs) form connection between the arteries and veins; the area with abnormal vasculature and shunting is called the nidus (Greene and Orbach, 2011, Bodra *et al*, 2016, Pekkola *et al.*, 2013). Although present at birth, AVM may not become evident until childhood. The most common site of extracranial AVM is the head and neck, followed by the limbs, trunk, and viscera. Lesions have a pink-red cutaneous stain, are warm, have a palpable thrill or bruit, and may be initially mistaken for a capillary malformation or hemangioma (Greene and Orbach, 2011). Congenital arteriovenous malformation (AVMs) occur most often spontaneously and less commonly as part of a broader syndrome such as Parkes-Weber syndrome, phosphatase and tensin homolog hamartoma syndrome, Osler-Weber-Rendu (OWR) syndrome, and congenital lipomatous overgrowth, vascular malformations, epidermal nevi, and skeletal deformities (CLOVES)syndrome (Richter and Friedman, 2012). Most of these syndromes are secondary to sporadic gene mutations althougha few are inheritable. Recently, an association has been made between AVMs and a constitutively active form of NOTCH-4, a signaling protein involved in endothelial cell differentiation during vasculogenesis and angiogenesis (Nassiri *et al.*, 2015). Currently there is no study which has been done in Tanzania to determine the occurrence of the vascular lesions in oral and maxillofacial region.

Management of vascular lesions

Most hemangiomas undergo involution therefore management often involves watchful neglect. However, assurance is deemed to be necessary to the parent as a child grows to observe if involution will occur. Ten to twenty percent of hemangioma lesions need active intervention because of their tendency to bleed and ulcerate (Mohan and Prasad, 2014, Lowe *etal.,* 2012). Acceptable indications for intervention of hemangioma may include rapidly enlarging lesions, obstruction of the visual axis or breathing, bleeding, ulceration, significant induced astigmatism, and cosmetic concerns. The modalities available are intralesional and systemic steroid, bleomycin, interferon-alpha, vincristine, cyclophosphamide, topical timolol maleate, intralesional absolute alcohol (98% ethanol), oral propranolol, and surgical excision (Nigwekar *et al*., 2011). Complicated hemangiomas may require any combination of: compression therapy, intralesional steroids (some response in 50–90% of cases), interferon α-2a, chemotherapy, intralesional sclerosing agents (hypertonic saline, glucose), cryotherapy, arterial embolization, radiation therapy, and surgical intervention (Nthumba, 2013).

Systemic steroids have been used successfully for the treatment of hemangiomas. A review of 10 case series concluded that treatment of cutaneous hemangiomas with oral corticosteroids like prednisolone results in a decrease in size or cessation of growth in actively proliferating hemangiomas in 84% of patients(Spring and Bentz, 2005). The recommended initial dose is generally 2 to 3 mg/kg of prednisone daily for a 2-week trial. If there is evidence of regression or stabilization of the hemangioma, the treatment is continued for another 2 weeks and then tapered over several months, with completion by 1 year of age(Chu and DeVita Jr., 2008, Spring and Bentz, 2005). Intralesional steroids are used to stop progression and promote hemangioma involution. Various compounds, such as triamcinolone, betamethasone, and dexamethasone, can be injected into the lesion at 4- to 8-week intervals (Chu and DeVita Jr., 2008).

Sclerosants are best adjuncts to subsequent surgery, but this methods / treatment modality has the disadvantage of causing excessive scarring. Cryotherapy is also an effective treatment for small superficial lesions (McClean and Hanke, 2013). Bleomycin is a chemotherapeutic agent

(antiangiogenic, antimitotic antibiotic derivative) used for treatment of many types of tumors including lymphomas, testicular cancer, squamous cell carcinoma, warts, condyloma acuminata, keloid, hypertrophic scars and breast cancer (Kabel *et al*., 2017). Its cytotoxic effect may be attributed to its interaction with O_2 and Fe^{2+} leading to scission of DNA causing G_1 arrest and induces apoptosis of hemangioma. Its systemic application may be associated with serious pulmonary fibrosis (Clarke, 2009).

Intralesional bleomycin can be used in combination with triamcinolone and epinephrine to give synergistic effect. The bleomycin mean dose is 2.5-3 mg in major lesions or 0.25-0.3 mg/kg of body weight or alone to 0.5mg/kg/dose (not exceeding 15 mg/ dose). Triamcinolone amount is 2.5- 15 mg / day for adults and in children between 6 to 12 years old and is 2.5-8 mg / day for children under 6 years (Chu and DeVita Jr., 2008). Epinephrine dosage is 1/100000 of each injection mixture. Hemangiomas and arteriovenous malformations treated well with the combination while port-wine spots and high-flow lesions responded relatively poor (Masiha *et al.*, 2012, Regmi *et al.*, 2017). Interventional treatment option for vascular malformation has been done several years with intralesional bleomycin with significant results (Dabus and Benenati, 2013). Injection bleomycin intralesional or perilesional can be started at 2-3 weeks of life (Ionescu *et al*., 2015). Injection is done under the assistance of an ultrasound especially for cutaneous hemangiomas (Memon, 2016).

Intralesional use of bleomycin has also been suggested for the treatment of lymphatic lesions and it was first used in the treatment of cystic hygroma in children (Horbach *et al*., 2016). It is equally effective for lymphangioma and slow flow vascular malformation although vascular malformations require significantly higher numbers of injections than lymphangioma (Regmi *et al.*, 2017). Sclerotherapy using bleomycin has been tried as a first line of management on congenital lymphatic and vascular malformations and it produced encouraging results (Mathur *et al.*, 2005). A study on intralesional injection of bleomycin in 30 patients with cystic hygroma was done whereby 0.5 mg /kg of bleomycin diluted in 10-15 cc of distilled water and was injected in the cyst at multiple sites. The injection was repeated after every month depending on the response. Complete resolution of lesions was achieved in 2 patients after a

maximum of seven shots of intralesional bleomycin injections (IBI), while 18 (60%) resolved after a single dose. Twenty seven lesions (90%) resolved completely, 2(6.6%) lesions had good response and 1(3.3%) showed poor response. No recurrence were noted in a maximum 2 years follow-up (Rasool *et al.*, 2014).

Other methods of management of lymphangiomas include injection of ethibloc which varies with the size of the lesion and of an alcohol (60%) solution of zein (corn-protein) produces intra-vascular thrombosis, necrosis, and fibrotic reaction (Zhou *et al.*, 2011). Carbon dioxide laser and intalesional sodium morhuate debulking have also been used in the treatment of lymphangioma (Schwarcz *et al.*, 2006).

Currently, pulsed dye laser (PDL) is considered the gold standard for the treatment of capillary malformations. However, its use results in incomplete clearance despite multiple sessions and recurrences. Topical Imiquimod, Rapamycin and Axitinib have been also used for the management of CM (Cheon *et al.*, 2017). Capillary malformation (CMs) treatment by intralesional bleomycin injections is not possible due to the small diameter of the vessels. Electroporation—an electric field applied to the tissue could increase the permeability of endothelial cells, which could theoretically facilitate targeted localized bleomycin delivery (Horbach *et al.*, 2017).

Management of venous malformation (VMs) by sclerotherapy has become the current mainstream option. It can be used alone or combined with surgery and/or laser therapy. For large lesions, multiple treatments are necessary. Recurrence may possibly happen with sclerosing agents that incompletely treat the VMs being injected. The sclerosants commonly used are 5% sodium morrhuate, intralesional bleomycin, anhydrous ethanol and lauromacrogol (Zheng *et al.*, 2013,Duncan, 2004). Small cutaneous lesions may be treated with intralesional injection 1% sodium tetradecyl decanoate (Nthumba, 2013). When appropriately selected for surgical excision, most do not require subsequent treatment for disease control (Rosenberg *et al.*, 2014).

If an arteriovenous malformation (AVM) is small and asymptomatic, no treatment is required, especially in children. For a symptomatic AVM, complete excision with prior embolisation is the treatment of choice (Panda *et al.*, 1990, Manjunath *et al.*, 2015). Onyx- a copolymer for blood vessel occlusion is a liquid embolizing agent, use of which has become increasingly common in the past two decades, allows better implementation of focal embolization and less inflammatory response (Hsiao *et al.*, 2012).

Surgical ligation of proximal feeding vessels should be avoided as it not only aggravates the lesion by establishing new collaterals but also precludes later embolisation (Science *et al.*, 2013, Pompa *et al.*, 2011). Trans-arterial occlusion of multiple feeding arteries and radiotherapy frequently cause incomplete devascularisation and collateral vessel recruitment (Keshelava *et al.*, 2009, Thioub *et al.*, 2018). For superficial AVMs, patients should apply hydrated-petroleum to prevent desiccation and subsequent ulceration. Compressions garments for extremity lesions may reduce pain and swelling, but can also worsen symptoms. Since estrogen is proangiogenic and may stimulate AVM progression, progesterone is a recommended oral contraceptive (Greene and Orbach, 2011). Empirical evidence concerning whether ethanol sclerotherapy can offer more lasting long-term results than careful embolization with other liquid agents is lacking. Further, the complication risk due to its toxic nature warrants caution (Pekkola *et al.*, 2013). However there is lack of knowledge on the methods for the management of vascular lesions involving oral and maxillofacial region.

Outcome of the management of vascular lesions

In a study of 155 lesions treated by 3 to 6 monthly injections of systemic steroids triamcinolone acetonide, 60% to 80% of the hemangiomas showed more than 50% reduction in volume, without subsequent growth. Superficial hemangiomas treated by this method yielded the best resolution results (Spring and Bentz, 2005). Steroid therapy increases the mast cells number and cytokine concentration and decreases platelet-derived growth factor and interleukin-6. Immunotherapy drug bevacizumab binds to VEGF and prevents binding to VEGF receptors. Combination therapy of immunotherapy and steroids decreased the bleomycin dose to only 0.1–1.2 mg/session, which was substantially lower than the

conventional doses (1–10 mg/session), however sessions could be after every three month for a year (Chu and DeVita Jr., 2008).

A clinical evaluation of 82 maxillofacial hemangioma cases treated with a combination of bleomycin (8 mg, powder), 2% lidocaine (3 ml) and dexamethasone (1 ml, 5 mg), or a mixture concentration of bleomycin (2mg/ml) and prednisone (2-5mg/kg every other day) showed that all hemangiomas involuted completely after several intralesional injections with the appropriate dosage (Luo and Zhao, 2011).

Intralesional injection of bleomycin (IBI) alone was also used and gave significant results in a study that involved 32 patients with hemangiomas. These patients were treated with four to six visits of bleomycin injections. After a minimum follow-up of 6 years, the lesions regressed by 70 to 100% regression in 18 patients, 50 to 70% in 7 patients, and less than 50% reduction in 7 patients (Omidvari *et al.,* 2005).

Surgical excision of small vascular growths is possible without undue risk of hemorrhage, cosmetic concern or need for reconstruction compared to large, localized deforming vascular lesions. It requires expertise and clinical judgment, surgical management may also be combined with laser treatment (Nthumba, 2013, Ahčan *et al.,* 2004). Flash lamp pulsed dye lasers can be used to treat small superficial hemangiomas due to minimal depth of penetration. The neodymium: yttrium-aluminum-garnet (Nd: YAG) laser is the treatment of choice for deep hemangiomas with subcutaneous components. Other types of laser used include CO_2, Argon, Ruby and potassium titanyl phosphate (KTP) lasers (Ahčan *et al.,* 2004).

Treatment used when other methods have failed include vincristine or radiation therapy (Duncan, 2004), and becaplermin (recombinant human platelet–derived growth factor) that has been reported to be useful in ulcerated infantile hemangiomas, especially those in the deeper areas (Nthumba, 2013). Imiquimod 5% cream has been found to be useful in proliferating hemangiomas. Its action is mediated through activation of natural killer cells by Interferon gamma, which has antiangiogenic effect. Embolization is usually employed for hemangiomas complicated by congestive heart failure when medical management fails (Science *et al.*, 2013).

Intermittent pneumatic compression and continuous compression have been used to treat symptomatic hemangiomas, especially for lesions on the extremities, although the mechanism of action is unknown. Cryosurgery with the use of a contact probe cooled by liquid nitrogen to treat isolated, raised lesions has been reported to hasten involution though with risk of scarring (Mendiratta and Jabeen, 2010).

Pharmacological treatment with propranolol for infants have been studied and showed good outcome, before treatment a detailed medical history is taken, pediatric and cardiologic consultation has to be done. It is usually initiated in the 2^{nd}- 3^{rd} month of life, which is the hemangioma proliferation phase. Most often, the therapy starts with an oral dose of 0.5 mg/kg/day (in three portions) which is gradually increased to 2 mg/kg/day (sometimes 3 mg /kg/day) (Wójcicki andWójcicka, 2014). Propranolol competitively inhibits beta1- and beta 2-adrenoceptors. Its effect on hemangiomas occurs by causing vasoconstriction and inhibition of angiogenesis by down regulation of PKA. It also inhibit ESRK／MAPK cascade hence VEGF and basic fibroblast growth factor (bFGF) and matrix metalloproteinases (MMP-2 and MMP-9) impairment, and inducing apoptosis (Town, 2013, Buckmiller, Richter and Suen, 2010). Studies showed there was no definitive conclusion regarding the optimal time for terminating propranolol treatment for infantile hemangioma (IH) it primarily depended on the regression rate of the lesion after propranolol treatment (Chang *et al.*, 2017).

A study on intralesional bleomycin sclerotherapy (IBS) in 17 children with cervical and macrocystic lymphangioma showed that 50% of the lesions had good response, 35.7% had complete resolution and 14.3% had poor response. The average follow-up was 18.5 months (Erikçi *et al.*, 2013, Rozman *et al.*, 2011). Satisfactory lymphangioma regression after a combination of injection of intravenous antibiotics followed by intralesional injection of a steroid /bleomycin/bevacizumab has been reported. Such a combination injection maximized the synergy effect during treatment. Initial effective volume ratio of triamcinolone (40 mg/mL), bleomycin (1 mg/mL), and bevacizumab (25 mg/mL) was 1:2:1 or 1:3:1 (Hwang, 2017).

Case series on patients with recalcitrant lymphatic malformations of the tongue showed 5 patients received transmucosal bleomycin injection, with available per visit as 0.5 mg/kg and maximum of 15 mg per visit and followed over a 10 month period. Four patients had 1 visit while 1 required 2 visits. A total of 1 to 6mg was injected per visit. Overall reduction in size of the lymphatic malformation and improvement in all symptoms were observed in the patients by day 14. Average follow-up was 9 to 12 months (Cerrati *et al.*, 2015).

OK-432 (Picibanil) a lyophilized incubated chemotherapeutic agent comprised a mixture of group A Streptococcus Pyogenes of human origin. It has been advocated for the treatment of lymphangioma of the head and neck, and produces local inflammatory mediators particularly IL-1a, IL-4, IL-13, IL-5, IL-6, TGF-b1, bFGF, VEGF and Interferon gamma upon injection (Keski-nisula, 2003). Inflammatory mediators increases permeability of endothelial cells of lymphangioma vessels, thus accelerate lymph drainage and this increase in high flow will lead to shrinkage of the cystic spaces. Its application is 0.1 mg of OK-432 per single injection and it doesn't form fibrosis around the cyst (Mazlumoglu, 2017, Baskota *et al.*, 2007). In a study of 10 patients treated with OK-432 showed that, there was totals welling resolution in nine patients (90%) after the first dose. One patient also responded well after the second dose (Baskota *et al.*, 2007, Benzar, 2014, Keski-nisula, 2003).

Although the treatment of choice for lymphangiomasis complete surgical excision, their infiltrating nature and difficulty in distinguishing involved vital structures of head and neck from adjacent normal tissues makes complete surgical resection difficult (Bhalekar *et al.*, 2016).

Other sclerosing agents which have been used include; absolute ethanol, 5% Ethanolamine oleate and 3% polidocanol. New preparations and formulations of sclerotic agents are continually developed, including foam preparations like sodium tetradecyl sulphate and polidocanol. Ethanol is a widely available and commonly used sclerotic agent however, it is also associated with complications following injections, including skin ulceration, nerve injury, and systemic complications (Mohan *et al.*, 2015).

Doxycycline, a derivate of tetracycline, is a widely available and relatively inexpensive broad-spectrum antibiotic used in the treatment of postoperative lymphoceles with minimal side effects. It is useful as a sclerotic agent in lymphangioma therapy (Orlando *et al.*, 2014). Doxycycline which is available as a powder can be suspended in saline or contrast medium though is painful to inject, but effective and relatively nontoxic. Percutaneous phlebography, an imaging method of choice to observe the hemodynamics and angio architecture of the malformation, has been used prior to immediate percutaneous management (Sierre, 2016). Various agents like alcoholic solution of Zein (Ethibloc) and OK-432 have now been used for percutaneous sclerotherapy (Regmi *et al.*, 2017).

Sclerosing agent 3% sodium tetradecyl sulphate has been used for the management of venous malformation. In one study 1ml of 3% sodium tetradecyl sulphate was injected intralesionally at multiple sites into the mucosa, periphery first and then into the centre with dosage not exceeded 2ml. The injections were repeated every 2 weeks and a total of three sessions of injections were given. During the fifth week of treatment the lesion completely resolved by fibrosis (Singh and Kumar, 2017).

Low dose of ethanol sclerotherapy in head and neck venous malformation has been studied and gave promising results. In one study 51, patients were treated with percutaneous intralesional injection of alcohol every two weeks and followed up prospectively for a median period of 18 months whereby 7 sessions of sclerotherapy were completed. The resolution or improvement was observed in 48(94.1%) patients presented (Orlando *et al.*, 2014). Alcohol has been used as sclerosing agent in the treatment of arteriovenous malformations (AVMs) and has been found to be effective. It has been advised to use general anesthesia while giving injections due to possible local and systemic effects in doses above 1 ml/kg or if a volume greater than 60 ml is used (Marshalleck and Johnson, 2006). Conversely knowing the effective and efficient outcome of this method its use in managing vascular lesions involving oral and maxillofacial region has been upheld.

Hypertonic hot saline-sodium chloride 23.4% is on use also for vascular malformations as a sclerosing agent. It was nonspecific in cellular destruction and it caused dehydration to

endothelial cells and red blood cells (Eltohami *et al.*, 2016). Treatment of these lesions is difficult due to lack of management expertise and facilities as well as treatment methods. This has led to an increased burden of untreated patients in our community. Extensive lesions were being referred outside the country for further management. Subsequent studies revealed that intralesional bleomycin induced accelerated resolution in vascular anomalies (Mabeta and Pepper, 2011). There was a need for low income countries like Tanzania to try to use and make follow up of intralesional bleomycin injections in vascular lesions.

Findings on the management and outcome of the vascular lesions will assist in bringing treatment of these lesions to the community and increase awareness on the possible cure. It will also help to formulate policy of the country on the management of vascular lesions. Hence the aim of this study was to determine the pattern of occurrence, clinical presentation and management of oral and maxillofacial vascular lesions at Muhimbili National Hospital.

1.3 CONCEPTUAL FRAMEWORK

The occurrence of vascular lesions has been associated with a number of factors like demographic and genetic of familial factors. Occurrence of these lesions has also been shown to appear as syndromic manifestations. Genetic or familial factors have also been associated with vascular lesions, as most vascular lesions are single and when they occur multiple often are familial. This study intended to describe the relationship of these factors with vascular lesions. The diagram below illustrates the relationship of the independent variables (factors), with the dependent variable (vascular lesions).

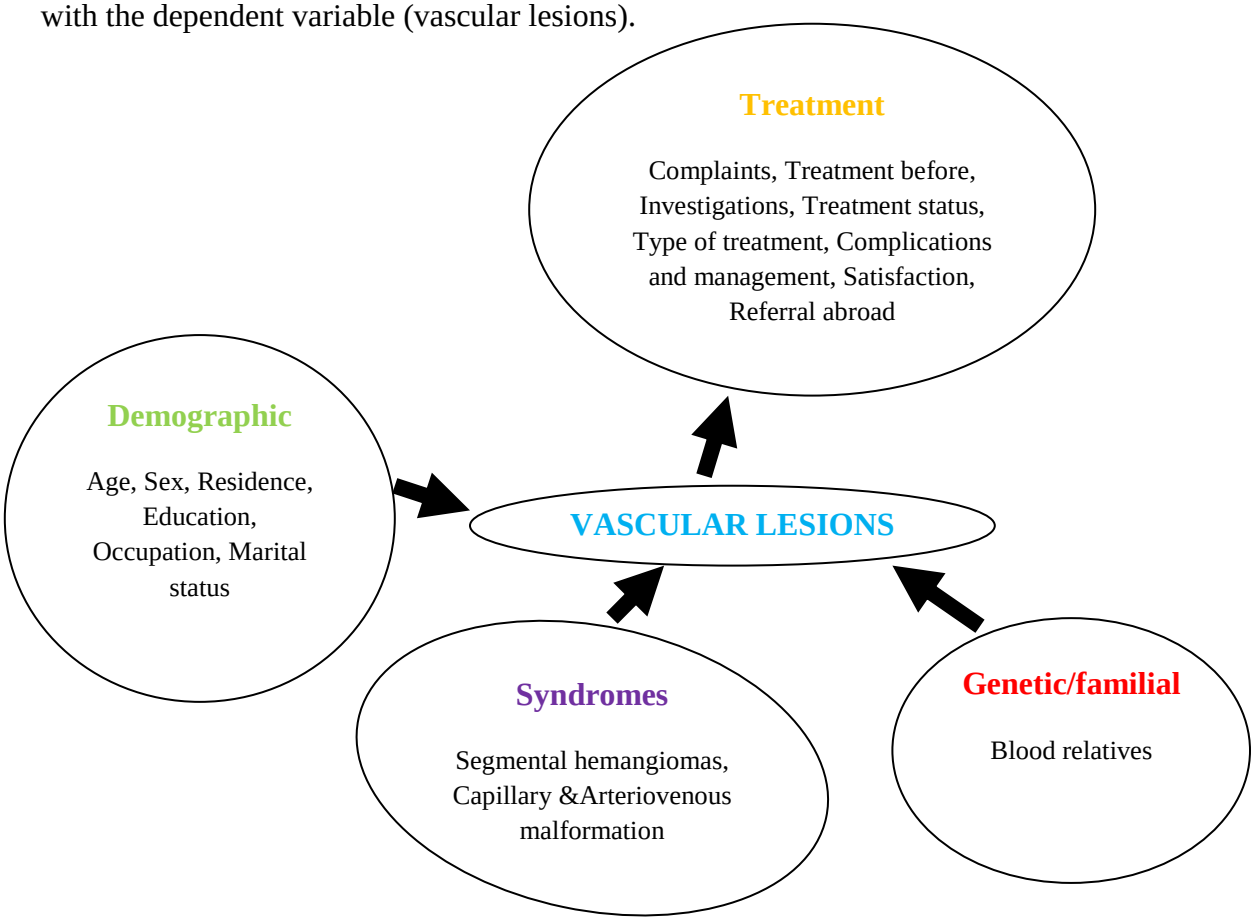


Figure 1.Conceptual framework for the occurrence and management of vascular lesions (Abbas M, 2018)

1.4 PROBLEM STATEMENT

Vascular lesions have physical and psychological distress to the patients, family and doctors particularly if visibly disfiguring maxillofacial region. These occur when they are huge, vastly destructive and untreated. Extensive lesions may also lead to significant morbidity and mortality.

Magnitude of these lesions is not known likewise demographic distribution for resource allocations and plan for their management. Treatment of these lesions is difficult due to the lack of management knowledge and facilities as well as treatment methods like embolization, sclerotherapy, sclerotherapy and surgery or surgical intervention; this has led to an increased burden of untreated patients. Previously extensive lesions were referred outside the country for further management owing lack of some expertise. There is no documented study to determine appropriate treatment methods of these vascular lesions and their outcome.

1.5 RATIONALE OF THE STUDY

The results of this study will shed light and increase expertise on different modalities of treatment of vascular lesions. Gaps identified like methods of treatments will help to improve management through inclusive multidisciplinary approach. This will decrease the number of patients who are usually referred outside the country for treatment.

Prevalence of these lesions is important to certainly know for future resource allocations and plan for their management. Patients awareness will also be increased which in turn motivating the health service seeking behavior.

The results can be used for setting the treatment protocol of vascular lesions at Muhimbili National Hospital and the country as a whole. Also these results can be used by the Ministry of Health to allocate resources for supplying the medicine and appropriate equipment. Furthermore, the results can help the country to save resources for these patients instead of referring them abroad. Lastly this research was part of the production of the dissertation as a partial fulfillment of the Master of Dentistry in Oral and Maxillofacial Surgery course.

1.6 RESEARCH QUESTION

This study was guided by the following research questions:

1. What is the pattern of occurrence of oral and maxillofacial vascular lesions at Muhimbili National Hospital?
2. Which methods of management were used for oral and maxillofacial vascular lesions at Muhimbili National Hospital?
3. What is the management outcome of the oral and maxillofacial vascular lesions at Muhimbili National Hospital?

1.7 OBJECTIVES

1.7.1 Broad objective

To determine pattern of occurrence, clinical presentation and management of oral and maxillofacial vascular lesions at Muhimbili National Hospital

1.7.2 Specific objectives

1. To determine the pattern of occurrence of oral and maxillofacial vascular lesions at Muhimbili National Hospital.
2. To determine the treatment modalities used for oral and maxillofacial vascular lesions at Muhimbili National Hospital.
3. To determine the outcome of the management of oral and maxillofacial vascular lesions at Muhimbili National Hospital.

CHAPTER TWO

2.0MATERIALS AND METHODS

2.1 Study setting

The study was conducted at the Oral and Maxillofacial Surgery (OMFS) clinic and in OMFS wards 23 and 24.It also involved Pediatric, Oncology, Ear Nose and Throat (ENT) departments of Muhimbili National Hospital.

2.2 Study design

It was a prospective descriptive cross section hospital based study.

2.3 Study duration

This study was conducted for nine (9) months from (1st June 2018 to 28th February 2019).

2.4 Study population

The study population included all patients with vascular lesions attended the departments of Oral and Maxillofacial Surgery (OMFS), Pediatrics, Oncology, Ear Nose and Throat (ENT) and those who were admitted in wards 23 and 24at Muhimbili National Hospital

2.5 Inclusion criteria

All patients who were diagnosed with Oral and Maxillofacial vascular lesions attending at Muhimbili National Hospital during the study period and had signed consent were included.

2.6 Exclusion criteria

Patients who were treated before, those on follow up and also who did not give consent.

2.7 Sampling procedure

A convenient sampling method was used whereby all patients with vascular lesions who presented at the departments of Oral and Maxillofacial Surgery, Oncology, Ear Nose and Throat, and Pediatrics, and those admitted in OMFS wards 23 and 24. All patients with vascular lesions received interviewed, examined and investigated, and those diagnosed or confirmed to have vascular lesion were included after consent or assent.

2.8 Sample size estimation

Sample size of this study was estimated based on a previous study by (Schwarcz *et al.*, 2006)

2.9 Sample size

The sample size was calculated from the formula:

$$n = \frac{Z^2 P(1-P)}{\varepsilon^2}$$

Where:

n = Sample size

Z = 95% confidence interval (1.96)

ε= Error/margin (5%)

P = previous prevalence (Schwarcz *et al.*, 2006)

$$n = \frac{(1.96)^2 x 0.07 x (1-0.07)}{(0.05)^2}$$

n = 100

Adding 10% of non-respondent n= 110

2.10 Data collection

All patients aged 18 years and above were interviewed using a specially designed questionnaire (Appendix V) to obtain demographic data, patients presenting complaints, anatomical site affected by vascular lesion, duration of lesion and treatment given. For the children<7 years guardians or parents were interviewed to obtain required relevant information. Patients were clinically examined, radiologically investigated and the findings were recorded in specially designed clinical form. Satisfaction level of the patients was enquired at the end of the individual treatment and at the end of the duration of the study.

2.11 Patients interview

Patients were interviewed through face to face by the investigator using structured questionnaire. Patients were introduced by the investigator in the study and questionnaires were given to them to read so that they could understand the context of the questions. For the patients above 18 years consent form was used while those with 7-17 years used assent form. For children < 7 years guardian/parents interviewed to get necessary information. The investigator then verbally asked the questions and appropriate answers were marked in the questionnaires.

2.11.1 Clinical examination

A thorough patient clinical examination was carried out by the principal investigator in the wards (23 and 24) in the side room with the patients in the examination bed using natural and artificial light. At the Oral and Maxillofacial Surgery clinic examination was done while the patient was seated on a dental chair using artificial light, the details of the vascular lesion examination included clinical presentation (swelling, ulceration, bleeding, disfigurement, skin color, characteristics of the lesion, difficult in breathing, type of vascular lesion, and site of occurrence like in the neck, submandibular region, periorbital, midface, lips, buccal mucosa, tongue and other parts of the body. Examination of an ulcer involved mode of onset, duration, any associated pain, any discharge, number, location, edges (undermine, punched out, sloping, rolled out or everted, floor, margins, base, relation to deeper structures, and pulsation. Examination of the swelling entailed location, size, shape, consistency, overlying color and temperature, mobility, fluctuation, pulsation, depth, edge, trans illumination, tenderness and surface. Re-examination was performed to assess new findings after treatment.

2.11.2 Investigations

All patients had series of routine and specific investigations which included routine hematological (full blood pictures, electrolytes, sickling test), cardiac and radiological investigations. Radiological investigations were for selective extensive vascular lesions and included CT scan, CT angiography, MRI, MR angiography and Doppler ultrasound. Records of the pictures / photos were also done. The interpretations of radiological investigations were

done by the principal investigator with assistance from an experienced radiologist. Cardiac investigations involved those aged patients with clinically risk of cardiovascular diseases which included electrocardiograph (ECG) and echocardiogram (ECHO). Histopathology involved fine needle aspiration cytology (FNAC) to confirm diagnosis scientifically before treatment. The findings of the clinical and radiological evaluations were recorded in the special clinical forms (Appendix VII). The data were checked for completeness and clarity and then the data were entered into the computer for analysis. Radiological records involved dimensions of the lesions before and after treatment to assess progress during sclerotherapy, intralesional injections were done under ultrasound guidance from the first to the six visits and involved almost all patients who undergone intralesional injections. Radiological investigations were done again after treatment. Those whose lesions resolved completely were regarded as cured while those with reduction in swelling were regarded as having good response and those with no response as poor response.

2.11.3 Validity and Reliability of data

2.11.3.1 Validity and Reliability of questionnaire data

Validity of the questionnaire was ascertained during its development by giving questions to experts to see if they had enough face and content validity. Further the questionnaire was administered to a group of vascular lesion patients who were asked to respond to the questions and state whether the questions were clear to them or not. The reliability of the questionnaire was tested during the main data collection, whereby 10% of the subjects were asked to fill in the same questionnaire during the main study and after two weeks. The paired data were analyzed to ascertain the degree of correlation.

2.11.4 Validity and reliability of clinical data

The validity of the instruments/supplies which involved the Dental chair, Gloves, Syringes, sclerotherapy drugs, IV or Oral drugs, CT scans, MRI,FNAC and Doppler ultrasound were used to collect the clinical data, and are used internationally, and have been shown to be of acceptable validity.

2.9 Data handling and analysis

Data were entered into a computer and analyzed using Statistical Package for Social Sciences (SPSS) programme version 20.0. Collected data were coded, cleaned and transformed by recording and grouping**.** Descriptive analysis included computation of percentages, frequency of occurrence, mean and cross tabulation of variables of interest such as age, sex, marital status, occupation and residence. Age groups were categorized from 0-10, 11-20, 21-30, 31-40, 41years and above according to the occurrence and involution of some of these vascular lesions particularly hemangioma and lymphangioma.

Inferential analysis included computation of the Chi-square or Fisher's exact test to compare proportions of possible associations. Significance was considered at the level when P-value was< 0.05. Dependent variable was occurrence of vascular lesion ie hemangioma, lymphangioma, capillary, venous or arteriovenous malformation as summarized in table appendix V. Chi-square test was used to test for differences in independent socio-demographic variables which included age, sex, residence, education level and occupation. Other variables included familial, sites of vascular lesion, number of lesions, and extent of the vascular lesions.

2.12 Ethical clearance

Scientific and ethical approval of the study was obtained from the MUHAS Senate Research & Publication Committee (Appendix VIII). Permission to conduct the study was obtained from the Executive Director of MNH-Research and Publication Committee. Following detailed explanation concerning the nature and purpose of the study, informed consent (Appendix I and II) was sought from the participants in writing before being enrolled in the study. For the minor/children assent was sought from the parents or guardians. Participant's confidentiality was accorded and also their right to participate or withdraw without any conditions. Those patients who were found to have vascular lesions were provided with available treatment as per MNH protocol.

2.13 Dissemination of results

This study was conducted with the primary aim of writing an academic dissertation as part of the requirement for the award of Master of Dentistry (Oral and Maxillofacial Surgery) degree of MUHAS. In addition, the abstract was presented at the MUHAS scientific conference and will be published in scientific journal and made available at MUHAS repository.

CHAPTER THREE

3.0 RESULTS

3.1 Socio-demographic characteristics

A total of 102patientswith vascular lesions involving oral and maxillofacial region were enrolled in this study. Forty nine (48.0%) were males while 53 (52.0%) were females with a male-to-female ratio of 0.9:1.

The age of the patients ranged from 4 months to 101 year and mean age of 28.1± 6.1 years. Nearly two thirds65(63.7%) of these patients were aged 0-10 years followed by those aged11-20 years. Almost all participants (96%) were aged between 40 years and below and only 4(3.9%) patients were above 40 years. Likewise, more than a half 61(59.8%) patients were from rural areas (Table 1).

Table1. Distribution of the patients with vascular lesions according to age, sex, and residence (N=102)

	Age in years	**n**	**Percentage (%)**
Age category	0-10	65	63.7
	11-20	16	15.7
	21-30	14	13.7
	31-40	3	2.9
	41+	4	3.9
Sex	Male	49	48
	Female	53	52
Residence	Urban	41	40.2
	Rural	61	59.8

3.2 Pattern of occurrence of vascular lesions

Overall occurrence of the vascular lesions at the age group 0-10 years showed that, patients with hemangiomas 33(50.8%) and lymphangiomas 30(46.2%) had significant proportion-formed almost two third. Four patients (3.9%) aged 41years and above had hemangiomas. One (6.3%) patient with mixed vascular lesion (Hemangiolymphangioma) was found at the age group of 11-20 years. This study showed that; there were more 58(56.9%) patients with

hemangiomas than those with 34(33.3%) lymphangiomas. Among patients with hemangioma, there were more females nearly (58.6%) compared to males (41.4%)(Table 2)

Table 2. Distribution of patients according to age group, sex and types of vascular lesions

	Age groups												
Age(years)	**0-10**		**11-20**		**21-30**		**31-40**		**41+**		**Total**		**Grand total**
Sex	**M**	**F**	**M**	**F**	**M**	**F**	**M**	**F**	**M**	**F**	**M**	**F**	**M & F**
HM*	14	19	5	8	3	5	0	1	2	2	24	34	58(56.9%)
LGM*	21	9	1	1	0	1	0	1	0	0	22	12	34(33.3%)
CM	0	1	0	1	0	1	1	0	1	0	2	3	5(4.9%)
VM	0	0	0	0	0	2	0	0	0	0	0	2	2(2%)
AVM	0	1	0	0	0	1	0	0	0	0	0	2	2(2%)
HLA	0	0	0	0	1	0	0	0	0	0	1	0	1(1%)
Grand total	**65(63.7%**		**16(15.7%)**		**14(13.7%)**		**3(2.9%)**		**4(3.9%)**		**49(48%)**	**53(52%)**	**102(100%)**

P value <0.05

Abbreviations:HM, Hemangioma; LGM, Lymphangioma; CM, Capillary Malformation; VM, Venous Malformation; HLA, Hemangiolymphangioma, M: Male, F: Female

In this study, 51(50%) vascular lesions occurred at birth, predominated by 29(56.9%) congenital lymphangiomas followed by 15(29.4%) congenital hemangiomas(CH).Majority 25(86.2%) of patients with Infantile hemangioma (IH) occurred mostly in the 1 to 6 months after birth, followed by those 18(81.8%) which occurred after birth. The least occurrence either at birth or after birth was seen in other vascular lesions though somehow they showed leniency at birth (Table 3).

Table3. Distribution of vascular lesions according to time of occurrence

Occurrence	Types of vascular lesions						Total
	HM*	LGM*	CM	VM	AVM	HLA	
At birth	15(29.4%)	29(56.9%)	3(5.9%)	1(2%)	2(3.9%)	1(2%)	51(50%)
1st to 6th months	25(86.2%)	3(10.3%)	1(3.4%)	0(0%)	0(0%)	0(0%)	29(28.4%)
> 6 months	18(81.8%)	2(9.1%)	1(4.5%)	1(4.5%)	0(0%)	0(0%)	22(21.6%)
Total	**58(56.9%)**	**34(33.3%)**	**5(4.9%)**	**2(2%)**	**2(2%)**	**1(1%)**	**102(100%)**

P value < 0.05

Abbreviations: HM, Hemangioma; LGM, Lymphangioma; CM, Capillary Malformation; VM, Venous Malformation; AVM, Arteriovenous Malformation, HLA, Hemangiolymphangioma

Hemangioma occurred in almost all anatomical sites in the oromaxillofacial region with the majority of the lesions 32(67.6%) of the lesions involved the lips followed by the 22(44%) cheeks, 22(56.4%) buccal mucosa and 15(65.2%) labial mucosa. Lymphangioma also involved all sites except the nose and majority of the lesions involved the 18(72%) submandibular region followed by the 14(43.8%) tongue and cervical region 13(76.5%). Capillary malformation occurred fairly distributed in almost all sites except the nose were in did not feature. The 50(49.0%)cheeks were the most affected by almost all vascular lesions except arteriovenous malformation followed by the lips affected by 46(45.1%) lesions, the buccal mucosa affected by 39(38.2%) lesions and the tongue with 14(43.8%) lesions. The forehead and the nose were the least affected sites. The other lesions (VM, AVM, and HLA) occurred distributed in a very few sites in the maxillofacial region. Ten (9.8%) patients had single type of lesion involving more than one site in the oral and maxillofacial region (Table 5). Almost all 98(96.1%) vascular lesions were single (focal) whereby 55(56.1%) hemangiomas and 34(34.7%) lymphangioma comprised greater proportion. Three (75%) patients who had hemangiomas had their lesions occurred in multiple sites beyond maxillofacial region and one (25%) patient with capillary malformation. Sites involved included; chest, upper arm, flank, thigh, orbit and calf.

Table 4. Distribution of vascular lesions according to anatomical sites in oral and maxillofacial region

Max fac region	Hemangioma	Lymphangioma	Capillary Malformation	Venous malformation	Arteriovenous malformation	Hemangio-lymphangioma	Total	P-value
Cervical	2(11.8%)	13(76.5%)	2(11.8%)	0(0%)	0(0%)	0(0%)	17(16.7%)	0.001
Cheeks	22(44%)	21(42%)	5(10%)	1(2%)	0(0%)	1(2%)	50(49.0%)	0.021
Lips	32(69.6%)	4(8.7%)	5(10.9%)	2(4.3%)	2(4.3%)	1(2.2%)	46(45.1%)	0.000
Submandibular	2(8%)	18(72%)	3(12%)	0(0%)	1(4%)	1(4%)	25(24.5%)	0.000
Forehead	1(33.3%)	0(0%)	2(66.7%)	0(0%)	0(0%)	0(0%)	3(2.9%)	0.000
Pre-auricular	3(42.9%)	2(28.6%)	2(28.6%)	0(0%)	0(0%)	0(0%)	7(6.9%)	0.099
Periorbital	5(50%)	3(30%)	2(20%)	0(0%)	0(0%)	0(0%)	10(9.8%)	0.323
Tongue	12(37.5%)	14(43.8%)	3(9.4%)	0(0%)	2(6.3%)	1(3.1%)	32(31.4%)	0.016
Buccal mucosa	22(56.4%)	10(25.6%)	3(7.7%)	2(5.1%)	1(2.6%)	1(2.6%)	39(38.2%)	0.214
Floor of the mouth	2(16.7%)	5(41.7%)	2(16.7%)	0(0%)	2(16.7%)	1(8.3%)	12(11.8%)	0.000
Oropharynx	1(20%)	1(20%)	1(20%)	0(0%)	1(20%)	1(20%)	5(4.9%)	0.000
Labial mucosa	15(65.2%)	2(8.7%)	4(17.4%)	1(4.3%)	1(4.3%)	0(0%)	23(22.5%)	0.004
Nose	2(66.7%)	0(0%)	0(0%)	0(0%)	1(33.3%)	0(0%)	3(2.9%)	0.005
Single type of lesion with multiple sites	3(30%)	5(50%)	1(10%)	0(0%)	1(1%)	0(0%)	10(9.8%)	0.228

Abbreviation: Max fac, Maxillofacial

3.3Clinical presentation of vascular lesions

Majority 98(96.1%) of the patients presented with swelling in the OMF region followed by pain in 48(47.1%) patients, ulceration in 24.5% of the patients. Other features in a descending order included; 23(22.5%) bleeding, 18(17.6%) dysphagia, 12(11.8%) difficult chewing, 7(6.9%) difficult in breathing and 7(6.9%) difficult vision (Figure 2)

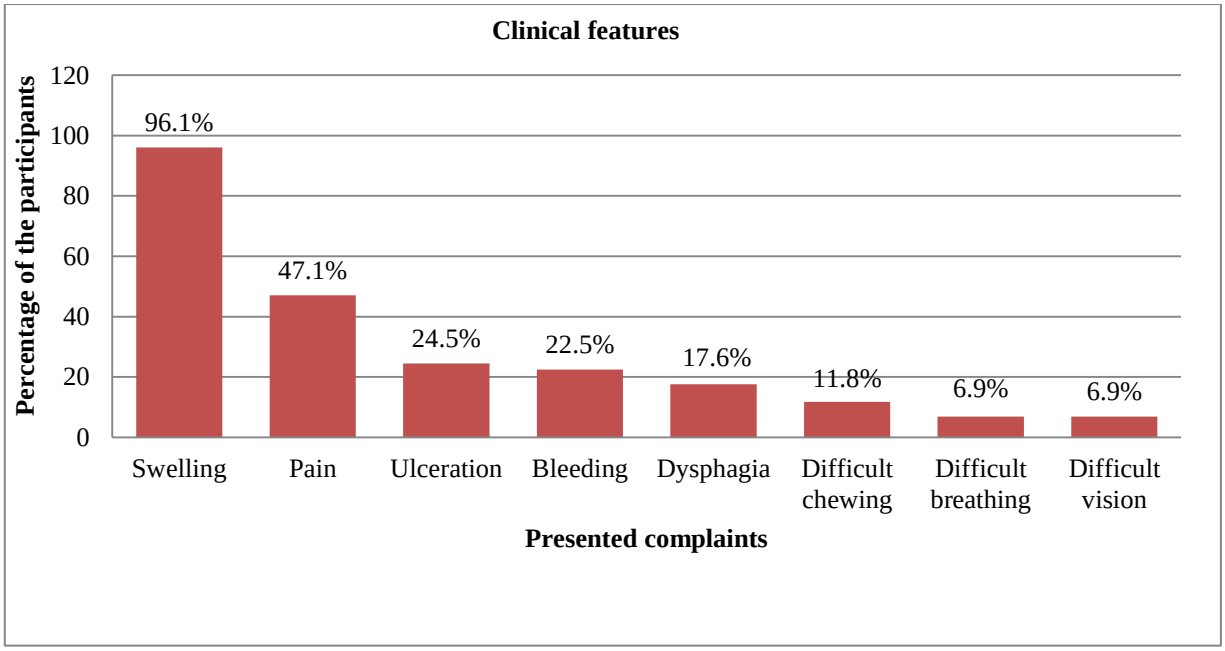


Figure2. Distribution of the participant's clinical presentations of vascular lesions

3.4 Management of patients with vascular lesions

In this study majority 62 (60.8%)patients were treated by intralesional bleomycin injection (IBI), 21(20.6%) patients who underwent underwent surgical intervention, 15.7% patients who received intralesional injection and underwent surgery and 2.9% patients who were kept on observation (Figure 3).

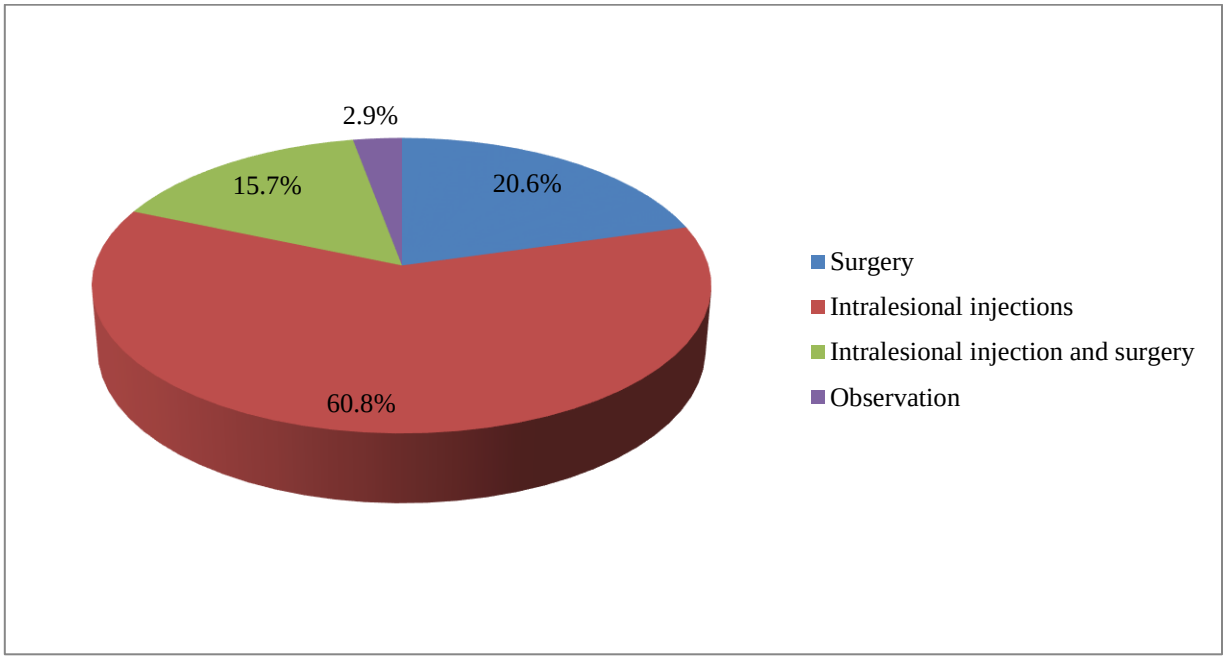


Figure 3.Distribution of patients with vascular lesions according to the methods of treatment used

Patients underwent sclerotherapy by intralesional bleomycin injections (IBI) from first to six visits after every 1 1/2 months intervals, few patients reached six visits (3.4%). Study showed almost three quarter 74(73.1%) underwent bleomycin injection for hemangioma and lymphangioma at different visits. There were few other vascular lesions patients 7(6.8%) who underwent sclerotherapy (Table 5). Minimum injection was 1ml and maximum was 15mls with an average of 12mls, almost all 91.2% patients underwent sclerotherapy under general anaesthesia

Table 5.Distribution of patients who underwent sclerotherapy according to type of vascular lesions and frequency of visits to sclerotherapy

Types of vascular lesions	1st visit	2nd visit	3rd visit	4th visit	5th visit	6th visit	Total
Hemangioma	51(87.9%)	40(68.9%)	25(43.1%)	18(31%)	5(8.6%)	2(3.4%)	58(56.9%)
Lymphangioma	23(67.6%)	19(55.9%)	8(23.5%)	5(14.7%)	0(0%)	0(0%)	34(33.3%)
Capillary malformation	3(60%)	3(60%)	2(40%)	0(0%)	0(0%)	0(0%)	5(4.9%)
Venous malformation	2(100%)	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)	2(2%)
Arteriovenous malformation	1(50%)	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)	2(2%)
Hemangio-lymphangioma	1(100%)	1(100%)	0(0%)	0(0%)	0(0%)	0(0%)	1(1%)
Total	**81(79.4%)**	**63(61.8%)**	**35(34.3)**	**23(22.5%)**	**5(4.9%)**	**2(2%)**	**102(100%)**

Majority 70(68.6%) patients were subjected to CT scan to get dimensions of the lesions before and after treatment. The study showed those 22 (56.4%) patients with hemangioma and 18(81.8%) patients with lymphangioma had their swellings reduced in size by 70-100%. However there was relatively proportion of 11(28.2%) patients with hemangioma and 3(13.6%) patients with lymphangioma who had the lesions reduced by 50-70%. There was poor reduction (<50%) in all types of vascular lesions but 100% in capillary malformation (Table 6).

Table 6. Distribution of response to sclerotherapy according to vascular lesions and reduction rates in percentage

	Reduction			
Types of vascular lesions	**70-100%**	**50-70%**	**< 50%**	**Total**
Hemangioma*	22(56.4%)	11(28.2%)	6(15.4%)	39(55.7%)
Lymphangioma*	18(81.8%)	3(13.6%)	1(4.5%)	22(31.4)
Capillary malformation	0(0%)	0(0%)	5(100%)	5(7.1%)
Venous malformation	0(0%)	0(0%)	1(100%)	1(1.4%)
Arteriovenous malformation	0(0%)	0(0%)	2(100%)	2(2.9%)
Hemangio-lymphangioma	0(0%)	0(0%)	1(100%)	1(1.4%)
Total	**40(57.1%)**	**14(20%)**	**16(22.9%)**	**70(100%)**

P value < 0.05

3.4 Treatment outcome of vascular lesions

Forty two (41.2%) patients were regarded as completed / cured or with excellent outcome. Among these patients 23(39.7%) who had hemangioma and 19 (55.9%) with lymphangioma formed greater proportion. Those hemangioma completed patients involved; 10 patients who were under sclerotherapy only, 6 sclerotherapy and surgery and 7 surgery only. Patients with lymphangioma who completed involved; 8 patients who were under sclerotherapy and surgery, 7 underwent surgery and 4 on sclerotherapy only. Half of the patients 51(50%)had good response after 4 visits and continued with treatmentwhich involved visits of sclerotherapyand, sclerotherapy together with surgical intervention. Nine (8.8%) patients had no response and required advanced expertise for treatment. It included 1(1.7%) of hemangioma and 1(2.9%) lymphangioma patient. Others were all patients of capillary and arteriovenous malformations (Table 7).

Table 7. Distribution of patients response according to vascular lesion and type of treatment used

Type of treatment	Sclerotherapy alone			Sclerotherapy and Surgery			Surgery alone			
Type of vascular lesions	**Cured**	**Good response**	**No response**	**Cured**	**Good response**	**No response**	**Cured**	**Good response**	**No response**	**Total**
HM*	10	19	1	6	11	0	7	4	0	58(56.9%)
LGM	4	3	1	8	10	0	7	1	0	34(33.3%)
CM	0	0	5	0	0	0	0	0	0	5(4.9%)
VM	0	2	0	0	0	0	0	0	0	2(2%)
AVM	0	0	2	0	0	0	0	0	0	2(2%)
HLA	0	1	0	0	0	0	0	0	0	1(1%)
Total	**14(13.7%)**	**25(24.5%)**	**9(8.8%)**	**14(13.7%)**	**21(20.6%)**	**0(0%)**	**14(13.7%)**	**5(4.9%)**	**0(0%)**	**102(100%)**

P-value <0.05

Abbreviations:HM, Hemangioma;LGM, Lymphangioma;CM, Capillary Malformatiom;VM,Venous Malformation;AVM, Arteriovenous Malformation;HLA,Hemangiolymphangioma

More than a half 58(56.9%) patients with vascular lesions comprised by 36(35.3%) patients with hemangioma and 22(21.6%) patients with lymphangioma were satisfied with treatment. Twenty (19.6%) patients were fairly satisfied while 24(23.5%) patients were not satisfied(Table 8).

Table 8.Distribution of patients according to satisfaction to treatments and types of vascular lesions

Patient Satisfaction	Type of vascular lesion						
	HM	**LGM**	**CM**	**VM**	**AVM**	**HLA**	**Total**
Satisfied	36(35.3%)	22(21.6%)	0(0%)	0(0%)	0(0%)	0(0%)	58(56.9%)
Fairly satisfied	11(10.8%)	5(4.9%)	2(2%)	1(1%)	0(0%)	1(1%)	20(19.6%)
Not satisfied	11(10.8%)	7(6.9%)	3(2.9%)	1(1%)	2(2%)	0(0%)	24(23.5%)
Total	**58(56.9%)**	**34(33.3%)**	**5(4.9%)**	**2(2%)**	**2(2%)**	**1(1%)**	**102(100%)**

Abbreviations:HM,Hemangioma;LGM,Lymphangioma; CM,CapillaryMalformation; VM,VenousMalformation; AVM,Arteriovenous malformation; HLA, Hemangiolymphangioma

Majority 87(85.3%) patients with vascular lesions had no immediate complications following treatment, however 9(8.8%) patients had difficult in breathing and 2(2%) patients had facial nerve paralysis. The remaining 4(3.9%) had mild pain, fever and recurrence (Figure 4).

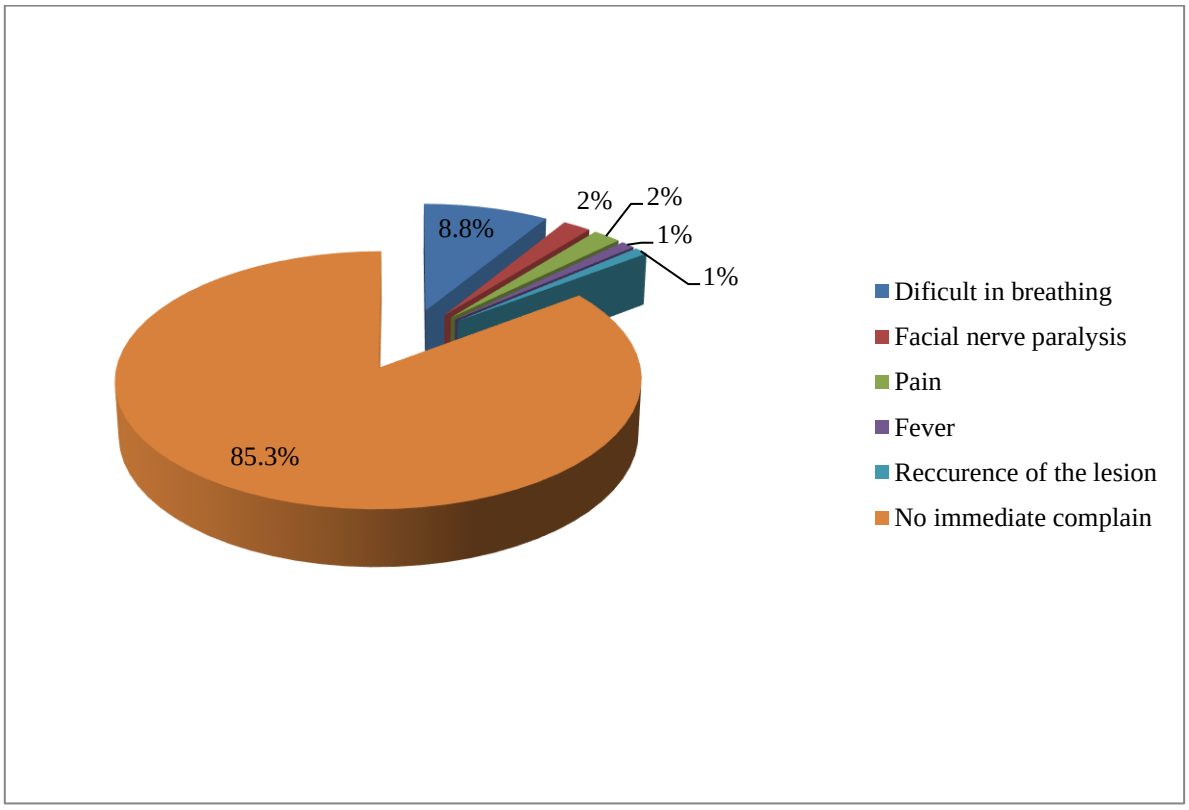


Figure 4. Distribution of patients according to the complications after treatment

Figures of patients pictures and images showing responses of vascular lesions according to treatment in oral&maxillofacial region

Figure 5(a) & 5(b) A 5 years old child with hemangioma of the right face with disfigurement and displaced eye, figure 5c the same child after 4 visits of sclerotherapy followed by surgery

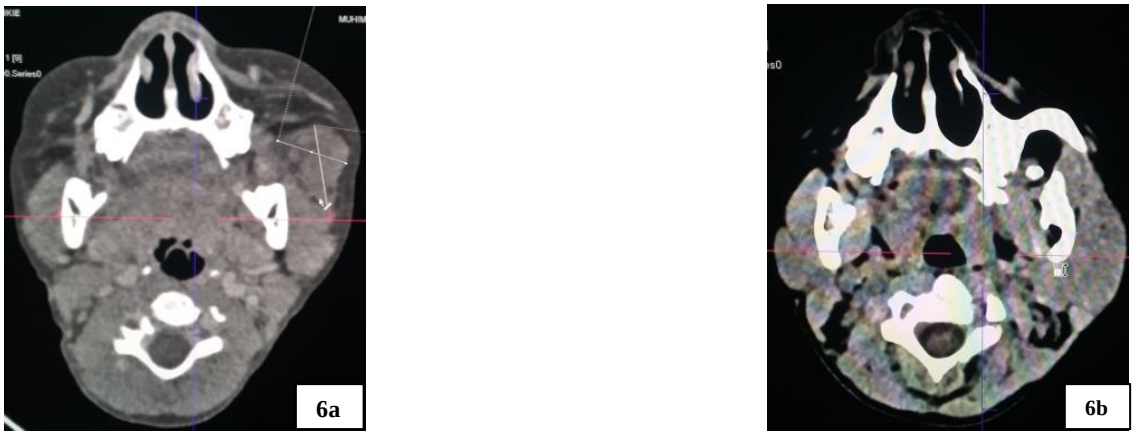


Figure 6(a) Hemangioma on 8 year old child in the left cheek measured 3.10x2.74 cmby CT scan before sclerotherapy

Figure 6(b)The same child in figure 6a showing resolution of the lesion after five visits ofsclerotherapy

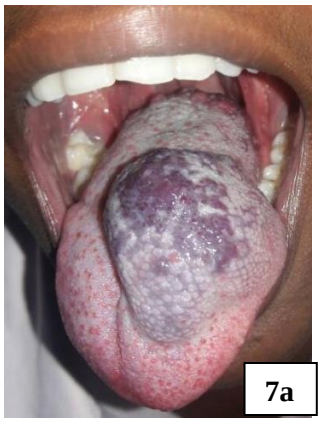
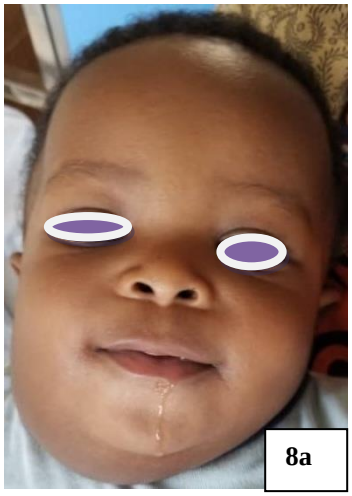


Figure 7(a) A 16 years old girl with hemangioma of the tongue before sclerotherapy. Figure 7(b) The same girl in figure 7a showing resolution of the lesion after 5 visits of sclerotherapy



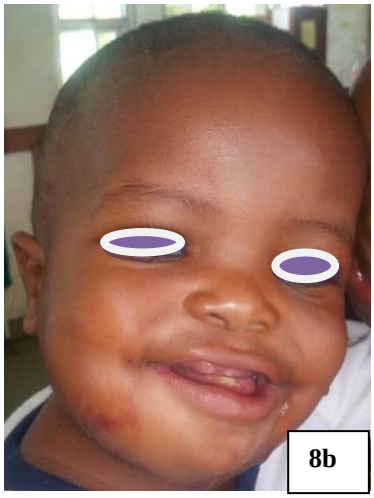
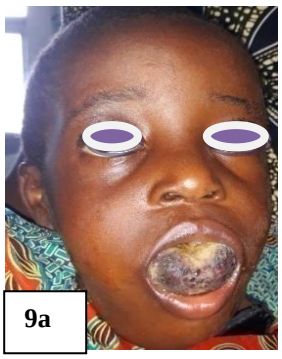


Figure 8(a) A 1 year old child with cystic hygroma in the cervical region right. Figure 7(b) The same patient in figure 8a after surgery with cervical approach



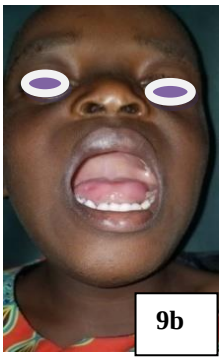


Figure 9(a) Tongue hemangioma to a 5 years old child before sclerotherapy

Figure 9(b) The same patient in figure 9a showing resolution of the lesion after four visits of sclerotherapy

CHAPTER FOUR

4.0 DISCUSSION

The current study showed that there were more patients with haemangiomas as compared to lymphangiomas. Similar findings were reported in another study (Gill *et al.,* 2012). The probable reasons for this difference in occurrence are that, clinically lymphangioma may occasionally appear similar to haemangioma when it has a significant capillary component. Also many arterio-venous malformations (AVM) are misdiagnosed as haemangiomas during infancy only later to cause progressive soft tissue destruction, ulceration, bleeding and disfigurement (Greene and Orbach, 2011, Darrow *et al.*, 2015).

This study revealed that, the occurrence of vascular lesions was higher in the age group of 0-10 years which comprised almost two thirds (63.75%), followed by 11-20 years age group. This observation was in congruent with other studies (Al-khateeb *et al.,* 2003, Sato *et al*., 1997). There were variations in the occurrence of hemangioma whether at birth as congenital hemangioma (CH) or few weeks or months after birth as infantile hemangioma (IH) which appears later in childhood or with an adulthood onset. Conversely patients with infantile hemangioma (IH) appeared with significant proportion after birth as compared to congenital hemangioma (CH).This was also similar to other studies (Hoornweg *et al*., 2011).

In this study the age of onset of vascular lesions after birth was found to vary from 1st to 6th months and after 6 months. This has been explained by other studies reflecting the fact that infantile hemangioma (IH) arises during the first 8 weeks of life with biological behavior of high proliferative phase for 6–12 months than the rate of infant growth, then followed by a gradual involution phase with a spontaneous regression by the age of 5–9 years (Lowe *et al.*, 2012).

Majority of lymphangiomas in this study occurred at birth and few after birth. This finding was somehow contrary to other studies which reported that lymphangiomas could be found at any age of life, approximately 50% being present at birth and 90% being diagnosed before or

within 2 years of age (Aydin *et al.,* 2015). The probable reason for the differences could be due to the study design and geographical variation in the pattern of occurrence of vascular lesions in different parts of the world (Moshy *et al.*, 2011). The finding of the present study showed that most patients with haemangiomas presented after birth while vascular malformations presented at birth concurred with other studies (Abidullah *et al.*, 2014).

Other vascular malformations (capillary, venous, arteriovenous and hemangiolymphangioma) showed no age predilection and could occur at any age withan adulthood onset. This finding was also reported by other studies (Jayapalan *et al.*, 2014). Hemangiolymphangioma (HLA) was the least observed lesion(only one case) in this study ,this rarity of the mixed lesion has also been reported in other studies (Murphy *et al*, 2017, Shetty *et al.*, 2010).

The results of this study showed that hemangiomas had female sex predilection than male at a ratio (1.4:1). Similar finding has been reported in other studies where there was female predilection (ratio = 1-3:1) (Cazeau *et al.*, 2017, Darrow *et al.*, 2015). The study also found that, lymphangiomas appeared more in males than females at a ratio of 1.8:1. This is contrary to other studies which reported that there was no race or sex predominance (Richter and Friedman, 2012). This slight difference may be attributed by the small sample size, geographical differences and study setting.The other vascular malformations; capillary, venous, arteriovenous and hemangiolymphangioma showed no sex predilection similar to what was in other study (Murphy *et al*, 2017).

Submandibular, tongue and cervical region were the most affected by lymphangioma. This could be due to abundant lymphatic channels which manifest early during embryogenesis and along the distribution of the trigeminal nerve (Grasso *et al.*, 2008). Hemangiomas were seen involving the lips, cheeks, buccal mucosa and labial mucosa; this can be explained by the presence of high deep vascularity and thick mucosa. These frequently involved sites were also been reported in other studies (Mohan and Prasad, 2014). Least affected areas by all vascular lesions were forehead, floor of the mouth, oropharynx and nose. The cheeks, lips (upper and lower) and the buccal mucosa of both sides were the most affected by almost all types of vascular lesions as reported also by other study (Senapathi and Pradeep, 2014).

Almost all (96.1%) vascular lesions occurred as focal (single) lesions with hemangiomas and lymphangioma forming a greater proportion. Appearance or occurrence of vascular lesions in several anatomical sites (in an individual patient) was observed in three patients with hemangiomas and another with capillary malformation who had the chest, upper arm, flank, thigh, orbit and calf being affected. There is variation in the sites involved in multiple occurrences as reported also by other studies (Spring and Bentz, 2005).

One patient with infantile multiple hemangiomas had 5 anatomical sites affected involved the left cheek, left upper arm, the back at the level of the waist, left flank and left calf region. Clinical and thorough investigations including imaging studies for visceral involvement were done; patient found to have no syndrome related vascular lesions. Other studies reported that;80% of hemangiomas occur as single (focal) lesions, but 20% of affected patients will have multiple (multifocal) lesions > 5 in which suspicion of visceral involvement is to be thought and complications occur in about 20% of non-syndromic hemangiomas (Neville *et al.*, 2002).

The most common reasons or complaints that made patients to seek care according to this study were swelling which led to disfigurement, pain, ulceration and bleeding. Others were difficult in swallowing, difficult in chewing, difficult in breathing and difficult in vision. Swelling which led to disfigurement as a significant complaint has been reported by other studies (Lee and Chung 2018). However contrary to our findings other studies reported that ulceration and bleeding as the main complaints (Redondo, 2007, Zheng *et al.*, 2013, Mahady *et al.*, 2015). The difference may be due to lack of early management which allowed the lesions to become extensive to involve other vital structures through compression or constant trauma especially in the oral cavity occasioned by ulcerations and bleeding.

Management used in this study included surgical intervention, sclerotherapy by intralesional bleomycin injections (IBI), sclerotherapy and surgery, and observation. Surgical intervention for definitive management involved patients with small operable lesions of hemangiomas and lymphangioma through cervical, perilesional and lateral rhinotomy incisions. These small lesions were those clinically found to have no complications like bleeding or need for

advanced know how, unlike lymphangioma were surgery is the gold standard of treatment. Some patients with extensive lesions underwent debulking to reduce the size necessarily to avoid bleomycin side effects when large (400mls) volume is used for sclerotherapy. Debulking involved lymphangiomas of the tongue, face and submandibular region, and then followed by sclerotherapy later after they had healed. One patient underwent medial maxillectomy due to hemangioma in the left maxillary sinus. Surgical intervention as a choice has been reported previously by other studies (Alster and Tan, 2016). Follow-up was also eminent with the mean observation after surgery for 3 months.

Intralesional bleomycin injection was a choice instead of perilesional injection. In this study bleomycin injection was the main mode of treatment generally in hemangiomas, lymphangiomas, venous malformation and as a trial therapy in capillary and arteriovenous malformations, and hemangiolymphangioma (HLA). Bleomycin was used in combination with water for injection or normal saline 10mls and 5mls of 2% lignocaine to make a constitution of 15mls, maximum injection was 15mls and the minimum was 1ml per lesion with an average of 12mls per session, the amount injected in small lesions was 7.5mls and in extensive lesions was 15mls. This was congruent with other studies (Luo and Zhao, 2011).

Almost all 91.2% patients underwent sclerotherapy under general anaesthesia with few through local anaesthesia, this was necessary because majority of the patients were children who had phobia with injections. Furthermore intralesional sclerotherapy was also done under the guidance a hand held Doppler ultrasound by interventional radiologist, this reduced the risk of injecting bleomycin in vessels instead of lesion hence patient had to be in sedation. Patients were subjected to a range of one to six visits at an interval of 1½ months between visit, and follow-up of 2 months for those had completed treatment.

Forty two patients had excellent outcome and their lesions disappeared completely hence regarded as cured included; hemangiomas and lymphangiomas patients. The lymphangioma patients were those with small lesions that were considered amenable for sclerotherapy regardless main stay of treatment being surgery. This explains also the fact that after 4^{th} visits

14 patients had their lesions disappeared and 14 underwent surgical intervention after sclerotherapy while 14 were intervened by surgery alone.

These cases were clinically examined and radiologically investigated to certainly be sure there was substantial reduction. Swelling reduction did not necessarily correspond to the number of visits that the patient made though in 39(38.2%) patients had their lesions reductions corresponded with 4 visits of sclerotherapy made. This finding is similar to other studies (Omidvari *et al.*, 2005).

Owing the short study period, it was not possible for all patients to complete management hence there were 51(50%) patients comprised of hemangiomas, lymphangiomas, venous and hemangiolymphangioma malformations who had to continue with treatments and were regarded to have good response.

Nine (8.8%) patientshad no response at all hence regarded as poor responders and needed advanced treatment with appropriate equipments for therapy like pulse dye laser (PDL) for capillary malformation, embolization and surgery for arteriovenous malformation. This has also been reported by other studies (Garrahan *et al.*, 2016). Extensive lesionsneed advanced expertise for surgery and could need soft tissue reconstruction.

Fifty eight patients comprised of hemangioma and lymphangioma were satisfied with the outcome of the treatment following reduction or disappearance of the swelling.Twenty patients were fairly satisfied and 24 patients were not satisfied at all. This may reflect the self-esteem, reduction in the psychological burden, cosmetic appealing and other discomforts, this has been reported in several studies (Redondo 2007, Zheng *et al.*, 2013, Mahady *et al.*, 2015).

Immediate complaints were encountered in nine patients who had mild difficult in breathing post sclerotherapy. These were managed by intravenous steroids, antibiotics and analgesics followed by close observation for 1 to 3 days. Tracheostomy was necessary in one patient of arteriovenous malformation who had massive edema that involved the floor of the mouth, tongue and the oropharynx following sclerotherapy, and kept on observation for 7 days. Two patients had severed facial nerve occurred immediately after surgical intervention of cystic

hygroma with cervical incisions hence had facial nerve paralysis. On the other hand no patient found to have pulmonary fibrosis due to low safety dose of bleomycin (15mg) used, risk would occur if the maximum dose of 400mg of bleomycin would be used. This had been reported also by other study (Regmi *et al.*, 2017). Pain and fever were other complaints by few patients following sclerotherapy due to massive inflammatory mediators particularly prostaglandin 2, tumor necrotic factor alpha, monocyte chemo attractant protein-1, cyclooxigenase-2 and IL-10. These were managed by intravenous or oral analgesics paracetamol then close observation. Such findings have also been reported by other studies (Regmi *et al.*, 2017, Redkar *et al.*,2017).

Recurrence of the lesion occurred in one patient who had previously undergone surgery due to cystic hygroma. There occurred continuous accumulation of lymphatic fluid after surgery that prompted re-operation after compression dressing and drainage for 1 month has failed. Following repeated surgery the lesion disappeared after 2 months observation. Such complications in lymphangioma have been reported in numerous studies (Dogan *et al.*, 2010, Philemon *et al.*,2009, Suni *et al.*, 2012).Because of the non-encapsulated and "infiltrating" nature of the lymphangioma, complete removal is difficult, and hence recurrence is common tissues (Zheng *et al.*, 2013).For this reason it warranted different approach of treatments.

Management was somehow compromised due to lack of appropriate instruments or equipments and advanced expertise especially when dealing with extensive lesions, and financial constraints as a factor in underdeveloped countries. Lack of appropriate equipments and technical knowhow like laser therapy and embolization led to compromised treatment of some patients who could have otherwise benefited. Non compliance ie lost to follow-up and refusal of some patients thus loss of flow of management hence increases a chance for recurrencelater. Some patients had permanent complications like facial nerve paralysis after surgery hence had negative experience and refused treatment, hence jeopardized further interventions. This explains also the fact that there was drastic fall of patients from first visit to the fourth visit.

Management of these lesions should be prioritized with necessary equipments eg laser therapy and embolization through interventional radiology. The most common treatment was intralesional injection of bleomycin under interventional radiology.

Vascular lesions were common in childhood particularly hemangioma which is also more common in females than males. Majority of lymphangioma were found at birth. As a result of clinical examination it showed that swelling was the most presented feature furthermore cheeks, lips, buccal mucosa, and tongue were most affected by all types of vascular lesions. Sclerotherapy by bleomycin found to be effective and efficient for vascular lesions particularly hemangioma and lynphangioma. Therefore the study is calling for the use of bleomycin for the treatment of vascular lesions which has been shown to be effective especially in resource limited set ups like ours.

4.1 CONCLUSION

In conclusion the study was expressed by the aim of assessing the pattern of occurrence, clinical presentation and management of vascular lesions. The study came out with the overall findings that, vascular lesions were common in childhood particularly hemangioma which is also more common in females than males. Majority of lymphangioma were found at birth. As a result of clinical examination it showed that swelling was the most presented feature. Cheeks, lips, buccal mucosa, and tongue were most affected anatomical sites by all types of vascular lesions. Thus management involved sclerotherapy by bleomycin, slerotherapy and surgery, and surgery. Sclerotherapy by bleomycin found to be effective and efficient for vascular lesions particularly hemangioma and lymphangioma. Therefore the study is calling for the use of bleomycin for the treatment of vascular lesions which has been shown to be effective especially in resource limited set ups like ours.

4.2 RECOMMENDATIONS

This study has brought forward the following recommendations:

a. Management of these lesions can be improved by modern medical facilities, eg laser therapy in capillary malformation, embolization through interventional radiology in arteriovenous malformation. This will shorten cycle of management instead of trials with no good outcome.
b. Awareness should be conducted national wide in order to enlighten people on the possible intervention due to available management.
c. To conduct a study with a large sample size in the country in order to get unknown burden of vascular lesions and plan of their management among patients in Tanzania.
d. Efforts should be put forward to train our own experts on advanced management of these vascular lesions and establishing our own vascular lesion centre
e. Treatment should be part and parcel to alleviate psychological circumstances of the patients due to disfigurements

4.3 STUDY LIMITATION AND MITIGATION

a) Sclerosing agent ie bleomycin formed the basis of treatment for sclerotherapy; this may lead to bias on the effectiveness in comparison to other sclerosing agents.
b) The cost for vials of bleomycin sclerosing agent was not affordable by many patients, this led to untimely management until waiver is obtained except for health insured patients
c) Lost to follow-ups due to some reasons eg lack of satisfaction through cosmetic appealing while on treatment, side effects after treatment like difficulty in breathing, financial constraints and others led to inability to reach the required sample size timely, loss of flow of management hence recurrences might be a consequence later.
d) Time bound of nine months, is a short period to provide complete management to all patients and explore more findings for future studies

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APPENDENCES

Appendix I: Informed consent form-English version

MUHIMBILI UNIVERSITY OF HEALTH AND ALLIED SCIENCES (MUHAS)

DIRECTORATE OF RESEARCH AND PUBLICATIONS

ID-NOHD/MUH/T.233/2016 | | | | | | | | | | | |

Consent to participate in study on occurrence and management of vascular lesions among patients attending at Muhimbili National Hospital, Tanzania

Greetings! My name is Dr. Abbas Mungia, currently a final year postgraduate student pursuing master of dentistry in Oral and Maxillofacial surgery at Muhimbili University of Health and Allied Sciences. I am working on this research study with the objective of investigating the occurrence and management of vascular lesions at Muhimbili National Hospital, Tanzania.

Purpose of the Study: We are enrolling inpatients and outpatients to participate in this study. In doing this study we will be able to document the common vascular lesions intra oral and extra oral among Tanzanians, including yourself, until the research is finished.

What Participation Involves: If you agree to join the study, you will be required to be examined your intra oral and extra oral vascular lesions.

Confidentiality: All information we collect on forms will be entered into computers with only the study identification numbers.

Risks: We do not expect that any harm will happen to you because of joining this study.

Rights to withdraw and Alternatives: Taking part in this study is completely your choice. If you choose not to participate in the study or if you decide to stop participating in the study you will continue to receive all services that you would normally get from this hospital. You can

stop participating in this study at any time, even if you have already given your consent. If you miss this session today for any reason but wish to come back into the study, we will be ready to accept your participation in the study. Refusal to participate or withdrawal from the study will not involve penalty or loss of any benefits to which you are otherwise entitled.

Benefits: If you agree to take part in this study, like all participants, you will benefit from knowing your vascular lesion and kind of management as well as receiving free advice on proper care. We hope that the information we learn from this study will benefit others.

In Case of Injury: We do not anticipate that any harm will occur to you as a result of participation in this study. However, if any physical injury resulting from participation in this research should occur, we will provide you with medical treatment according to the current standard of care in Tanzania. There will be no additional compensations to you.

Who to Contact: If you ever have questions about this study, you should contact the Principal Investigator Dr. Abbas Mungia (0712150785), Muhimbili University of Health and Allied Sciences, P.O.Box 65001, Dar es Salaam, or through the email address abbasmusa71@yahoo.com. If you ever have questions about your rights as a participant, you may call Dr Bruno Sunguya, Chairman of the Senate Research and Publications Committee P.O Box 65001 Dar es Salaam; phone +255222152489 Dar es Salaam; as well as supervisor of the study Dr Jeremiah Moshy who can be contacted through phone number 0754293242.

Signature:

Do you agree?

Participant agrees …… Participant does NOT agree ……..

I, _____ have read the contents in this form. My questions have been answered. I agree to participate in this study/ I allow my child to participate in the study (for participants >18 years).

Signature of a parent/guardian _____

Signature of a researcher _____

Date of signed consent _____

Appendix II: Informed consent form –Swahili version

CHUO KIKUU CHA SAYANSI ZA AFYA MUHIMBILI (MUHAS)

KURUGENZI YA TAFITI NA CHAPISHAJI FOMU ZA RIDHAA

Namba ya utambulisho……

Ridhaa ya kushiriki kwenye utafiti unaohusu vivimbe vinavyotokana na mishipa ya damu/maji pamoja na matibabu yake kwa wagonjwa wanaohudhuria Hospitali ya Taifa Muhimbili

Salamu! Naitwa Dr. Abbas Musa Mungia, nashughulika kwenye utafiti huu wenye lengo la kutathimini uvimbe katika kinywa, uso na shingo kwa wagonjwa katika Hospitali ya Taifa Muhimbili, Tanzania.

Umuhimu wa utafiti: Utafiti huu unafanyika katika kutimiza sehemu ya matakwa ya shahada ya uzamili ya upasuaji kinywa na meno ya Chuo Kikuu cha Afya na Sayansi za Tiba Muhimbili. Utafiti unalenga kuchunguza sababu, ainana tiba ya kuumia katika sehemu za kinywa na uso unaombwa kushiriki katika utafiti kutokana na upeo na ufahamu ulio nao ambavyo ni muhimu kwa utafiti huu. Tafadhali kuwa mkweli na muwazi kwa vile matokeo ya utafiti huu yanaweza yakatoa maamuzi na mapendekezo ya baadae.

Jinsi ya kushiriki: Ukikubali kushiriki katika utafiti huu, utasailiwaili kuweza kujibu maswali toka kwenye dodoso lililo andaliwa kwa ajili ua utafiti huu na kisha utafanyiwa uchunguzi ambao utahusisha kuangalia maeneo ulioumia, pia utafanyiwa vipimo mbalimbali kama vile vipimo vya damu kuangalia wingi wa seli mbalimbali za damu na picha za x-ray itakapolazimu.

Usiri: Taarifa zote zitakazo kusanywa zitaingizwa kwenye ngamizi kwa kutumia namba za utambulisho kutakuwa na usiri na hakuna mtu yeyote asiyehusika atakayepata taarifa zilizokusanywa.

Hatari: Hatutegemei madhara yeyote kutokea kwa kushiriki kwako katika utafiti huu.

Faida: Kama utakubali kushiriki katika utafiti huu taarifa utakazotoa zitakuwezesha kujua ukubwa wa tatizo ambao ni muhimu katika uamuzi wa kuzuia au kupunguza tatizo.

Athari na kukitokea madhara: Haitegemewi kupata madhara yoyote kutokea kutokana na ushiriki wako katika utafiti huu.Baadhi ya maswali yanaweza yasikupendeze, unaweza kukataa kujibu swali lolote la aina hiyo na unaweza kuamua kusimamishaa udahili wakati wowote.

Uhuru wa kushiriki na haki ya kujitoa: Kushiriki kwenye utafiti huu ni hiari. Unaweza kujitoa kwenye kujitoa kwenye utafiti huu wakati wowote hatakama umeshajaza fomu ya ridhaa ya kushiriki utafiti huu.Kukataa kushiriki au kujitoa kwenye utafiti huu hakutaambatana na masharti yoyote.

Nani wa kuwasiliana naye: Kama una maswali kuhusiana na utafiti huu wasialiana na mtafiti mkuu wa utafiti huu,Dr abbas Musa Mungia (0712150785) wa chuo kikuu cha afya na sayansi za tiba Muhimbili,S.L.P 65001,Dar es Salaam,au barua pepe abbasmusa71@yahoo.com. Kama una swali kuhusu stahili zako kama mshiriki unaweza kumpigia Dr. Bruno Sunguya, Mwenyekiti wa Kamati ya Utafiti na Machapisho, chuo Kikuu cha Afya na Sayansi za Tiba Muhimbili,S.LP 65001 Dar es salaam,simu : +255222152489 Dar es Salaam au msimamizi wa utafiti Dr Jeremiah Moshy (0754293242).

Je umekubali?

Mshiriki amekubali……..mshiriki hajakubali……

Mimi……..nimesoma na kuelewa maelezo ya fomu hii. Maswali yangu yamejibiwa .Nakubali kushiriki katika utafiti huu /Naruhusu mtoto wangu ashiriki kwenye utafiti (Kwa washiriki zaidi ya miaka 18).

Sahihi ya mzazi/mlezi …….……

Sahihi ya mtafiti mkuu……

Tarehe ya kutia sahihi ya idhini ya kushiriki……..

Appendix III: Informed assent form-English version

MUHIMBILI UNIVERSITY OF HEALTH AND ALLIED SCIENCES (MUHAS)

DIRECTORATE OF RESEARCH AND PUBLICATIONS

ID-NOHD/MUH/T.233/2016

Consent to participate in study on occurrence and management of vascular lesions among patients attending at Muhimbili National Hospital, Tanzania

Greetings! My name is Dr. Abbas Mungia, currently a final year postgraduate student pursuing master of dentistry in Oral and Maxillofacial surgery at Muhimbili University of Health and Allied Sciences. I am working on this research study with the objective of investigating the occurrence and management of vascular lesions at Muhimbili National Hospital, Tanzania.

Purpose of the Study: We are enrolling inpatients and outpatients to participate in this study. In doing this study we will be able to document the common vascular lesions intra oral and extra oral among Tanzanians, including yourself, until the research is finished.

What Participation Involves: If you agree to join the study, you will be required to be examined your intra oral and extra oral vascular lesions.

Confidentiality: All information we collect on forms will be entered into computers with only the study identification numbers.

Risks: We do not expect that any harm will happen to you because of joining this study.

Rights to withdraw and Alternatives: Taking part in this study is completely your choice. If you choose not to participate in the study or if you decide to stop participating in the study you will continue to receive all services that you would normally get from this hospital. You can stop participating in this study at any time, even if you have already given your consent. If you miss this session today for any reason but wish to come back into the study, we will be ready to accept your participation in the study. Refusal to participate or withdrawal from the study will not involve penalty or loss of any benefits to which you are otherwise entitled.

Benefits: If you agree to take part in this study, like all participants, you will benefit from knowing your vascular lesion and kind of management as well as receiving free advice on proper care. We hope that the information we learn from this study will benefit others.

In Case of Injury: We do not anticipate that any harm will occur to you as a result of participation in this study. However, if any physical injury resulting from participation in this research should occur, we will provide you with medical treatment according to the current standard of care in Tanzania. There will be no additional compensations to you.

Who to Contact: If you ever have questions about this study, you should contact the Principal Investigator Dr. Abbas Mungia (0712150785), Muhimbili University of Health and Allied Sciences, P.O.Box 65001, Dar es Salaam, or through the email address abbasmusa71@yahoo.com . If you ever have questions about your rights as a participant, you may call Dr Bruno Sunguya, Chairman of the Senate Research and Publications Committee P.O Box 65001 Dar es Salaam; phone +255222152489 Dar es Salaam; as well as supervisor of the study Dr Jeremiah Moshy who can be contacted through phone number 0754293242.

Signature:

Do you agree?

Participant agrees …… Participant does NOT agree ……..

I, _____ have read the contents in this form. My questions have been answered. I agree to participate in this study **(for participants <18 years).**

Signature of a participant _____

Signature of a researcher _____

Date of signed consent _____

Appendix IV: Informed assent form –Swahili version

CHUO KIKUU CHA SAYANSI ZA AFYA MUHIMBILI (MUHAS)

KURUGENZI YA TAFITI NA CHAPISHAJI FOMU ZA RIDHAA

Namba ya utambulisho……

Ridhaa ya kushiriki kwenye utafiti unaohusu vivimbe vinavyotokana na mishipa ya damu/maji pamoja na matibabu yake kwa wagonjwa wanaohudhuria Hospitali ya Taifa Muhimbili

Salamu! Naitwa Dr. Abbas Musa Mungia, nashughulika kwenye utafiti huu wenye lengo la kutathimini uvimbe katika kinywa, uso na shingo kwa wagonjwa katika Hospitali ya Taifa Muhimbili, Tanzania.

Umuhimu wa utafiti: Utafiti huu unafanyika katika kutimiza sehemu ya matakwa ya shahada ya uzamili ya upasuaji kinywa na meno ya Chuo Kikuu cha Afya na Sayansi za Tiba Muhimbili. Utafiti unalenga kuchunguza sababu, ainana tiba ya kuumia katika sehemu za kinywa na uso unaombwa kushiriki katika utafiti kutokana na upeo na ufahamu ulio nao ambavyo ni muhimu kwa utafiti huu. Tafadhali kuwa mkweli na muwazi kwa vile matokeo ya utafiti huu yanaweza yakatoa maamuzi na mapendekezo ya baadae.

Jinsi ya kushiriki: Ukikubali kushiriki katika utafiti huu, utasailiwa ili kuweza kujibu maswali toka kwenye dodoso lililo andaliwa kwa ajili ua utafiti huu na kisha utafanyiwa uchunguzi ambao utahusisha kuangalia maeneo ulioumia, pia utafanyiwa vipimo mbalimbali kama vile vipimo vya damu kuangalia wingi wa seli mbalimbali za damu na picha za x-ray itakapolazimu.

Usiri: Taarifa zote zitakazo kusanywa zitaingizwa kwenye ngamizi kwa kutumia namba za utambulisho kutakuwa na usiri na hakuna mtu yeyote asiyehusika atakayepata taarifa zilizokusanywa.

Hatari: Hatutegemei madhara yeyote kutokea kwa kushiriki kwako katika utafiti huu.

Faida: Kama utakubali kushiriki katika utafiti huu taarifa utakazotoa zitakuwezesha kujua ukubwa wa tatizo ambao ni muhimu katika uamuzi wa kuzuia au kupunguza tatizo.

Athari na kukitokea madhara: Haitegemewi kupata madhara yoyote kutokea kutokana na ushiriki wako katika utafiti huu.Baadhi ya maswali yanaweza yasikupendeze, unaweza kukataa kujibu swali lolote la aina hiyo na unaweza kuamua kusimamishaa udahili wakati wowote.

Uhuru wa kushiriki na haki ya kujitoa: Kushiriki kwenye utafiti huu ni hiari. Unaweza kujitoa kwenye kujitoa kwenye utafiti huu wakati wowote hata kama umeshajaza fomu ya ridhaa ya kushiriki utafiti huu. Kukataa kushiriki au kujitoa kwenye utafiti huu hakutaambatana na masharti yoyote.

Nani wa kuwasiliana naye: Kama una maswali kuhusiana na utafiti huu wasialiana na mtafiti mkuu wa utafiti huu,Dr abbas Musa Mungia (0712150785) wa chuo kikuu cha afya na sayansi za tiba Muhimbili,S.L.P 65001,Dar es Salaam,au barua pepe abbasmusa71@yahoo.com . Kama una swali kuhusu stahili zako kama mshiriki unaweza kumpigia Dr. Bruno Sunguya,Mwenyekiti wa Kamati ya Utafiti na Machapisho, chuo Kikuu cha Afya na Sayansi za Tiba Muhimbili,S.LP 65001 Dar es salaam, simu: +255222152489 Dar es Salaam au msimamizi wa utafiti Dr Jeremiah Moshy (0754293242).

Je umekubali?

Mshiriki amekubali……..mshiriki hajakubali……

Mimi……..nimesoma na kuelewa maelezo ya fomu hii. Maswali yangu yamejibiwa.Nakubali kushiriki katika utafiti huu **(Kwa washiriki chini ya miaka 18).**

Sahihi ya mshiriki……

Sahihi ya mtafiti mkuu……

Tarehe ya kutia sahihi ya idhini ya kushiriki……..

Appendix V: Questionaire –English version

MUHIMBILI UNIVERSITY OF HEALTH AND ALLIED SCIENCES

Demographic data

1. 1. Serial No.....2. Date…….. 3. Hospital Reg. No……..
2. Residence;
   1.ward/division…….2.District……....3.Region……..……..Telephone no…….
3. Category 1. In patient 2. Out patient
4. If minor information is given by parent/guardian Sex 1.Male 2.Female 3.Not minor
5. Sex (patient)
   1 Male 2. Female
6. Age
   1 ……years and …….months
7. Marital status (parent or guardian if minor)
   1 Single
   2 Married
   3 Widow
   4 Widower
   5 Cohabiting
   6 Divorced
8. Education level (parent or guardian if minor)
   1 No formal education
   2 Primary education
   3 Secondary education
   4 Tertiary education
9. Occupation (parent or guardian if minor)
   1 Peasant
   2 Petty trader
   3 Business
   4 Employed
   5 Un employed
10. Department in which patient is attending
    1 Oral and Maxillofacial Surgery
    2 Pediatrics
    3 Oncology
    4 Ear Nose and Throat

Occurrence

11 When did your complain occurred?
   1. Since birth
   2. …….. weeks/months/year or years after birth

12 Why did you decide to come to the hospital?
   1. Because of pain
   2. Disfigurement
   3. Difficult breathing
   4. Difficult in swallowing
   5. Bleeding
   6. Difficult vision
   7. Ulceration
   8. Difficult chewing

13 Is there a family member / members (blood relative) with the same complain as yours?
   1. Yes
   2. No

14 If yes in question 12 above, what is your relationship?
   1 Father 2 Mother 3 Sister 4 Brother

15 Site where vascular lesion is
   1 Face
   2 Cheek
   3 Neck
   4 Tongue
   5 Head
   6 Others mention……..

16 Number of lesion/lesions
   1 Multiple (mention)…..
   2 Single

Treatment

17 Have you ever been treated before because of your swelling?
   1 Yes
   2 No

18 If yes in question 16 above where did you get treatment?
   1 At the regional hospital
   2 At Zonal hospital
   2 National hospital
   3 Outside the country

19 In which phase of treatment are you?

1. Starting
2. On oral tabs Propranolol for ……weeks /……month/s
3. ……weeks /months after surgery
4. 1^{st} / 2^{nd} / 3^{rd} / 4^{th} / 5^{th} / 6^{th} / 7^{th} visits of Intralesional injections
5. Follow up ……weeks/months/year after treatments of tabs Propranolol /injections

20. Which investigation/s did you do before treatment?

1. Doppler Ultrasound / Ultrasound
2. CT Angiograph
3. MR Angiograph
4. MRI
5. CT scan
6. Others eg FNAC……..
7. Didn't do any investigation

21. Which type of treatment did you get?

1. Oral medications ie Propranolol tabs
2. Surgery
3. Lesion injections
4. Lesion injections and surgery
5. Watchful neglect

Complications after treatment

22. Did you get any problem after treatment? 1. Yes 2. No

23. If yes in question 22 above which problems did you get?

1. Pain
2. Fever
3. Respiratory problems
4. Skin problems
5. Necrosis of the tissues
6. Others (mention)……

24. Were you given any other medications /application after lesion treatment?

1. Antibiotics
2. Pain killer/s
3. Drugs against swelling (Anti-inflammatory drugs)
4. Topical anaesthesia

25. If injections were given for treatment, applied under which condition?
    1. Local anaesthesia
    2. Sedation
    3. General anaesthesia
    4. Analgesics before injection
    5. Not given any medication before

Post treatment

26. Were you satisfied with the outcome of your treatment?
    1. Very well satisfied
    2. Fairly satisfied
    3. Somehow satisfied
    4. Not satisfied
    5. Not satisfied at all

Appendix VI: Questionnaire –Swahili version

CHUO KIKUU CHA SAYANSI ZA AFYA MUHIMBILI

Dodoso kuhusu taarifa binafsi

1 1. Namba….....2. Tarehe …….. 3. Namba ya Hospitali ……..

2 Makazi 1.Kata…….2.Wilaya……..3.Mkoa……4.Namba ya simu……..

3 Aina ya Mgonjwa 1. Mgonjwa aliyelazwa 2. Mgonjwa wa nje

4 Taarifa za mgonjwa zinatolewa na mzazi/mlezi (kama miaka <18) ,Jinsi 1.Mke 2.Mme 3.Si mtoto

5 Jinsi (mgonjwa)

1. Mme 2. Mke

6 Umri

1. Miaka …….na miezi……

7 Hali ya ndoa(mzazi au mlezi kama ni mtoto miaka <18)

1. Sijaolewa /Sijaoa
2. Nimeolewa /nimeoa
3. Mtaliki
4. Mtalika
5. Tunaishi bila ndoa
6. Tumeachana

8 Kiwango cha elimu(mzazi au mlezi kama ni mtoto miaka <18)

1. Sikusoma
2. Elimu ya msingi
3. Elimu ya sekondari
4. Elimu ya juu

9 Kazi (mzazi au mlezi kama ni mtoto miaka <18)

1. Mkulima
2. Mfanya biashara ndogondogo
3. Biashara
4. Muajiriwa
5. Sina kazi

10 Idara ambayo mgonjwa anatibiwa

1. Kinywa na meno
2. Watoto
3. Magonjwa ya saratani
4. Masikio Pua na Koo

11 Tatizo lako lilianza lini?
1. Tangu kuzaliwa
2. Wiki/miezi/miaka…….. baada ya kuzaliwa

12 Nini kilikusababisha kuja hospitali?
1. Sababu ya maumivu
2. Kuharibika sehemu yangu ya mwili
3. Kushindwa kupumua
4. Kushindwa kumeza
5. Kutokwa na damu
6. Kushindwa kuona
7. Kupata kidonda
8. Kushindwa kutafuna

13 Je una ndugu (wa kuzaliwa nae) yeyote katika familia yako anatatizo kama hili lako?
1. Ndio 2. Hapana

14 Kama ndio katika swali la 12 hapo juu,ni upi uhusiano wako?
1. Baba 2. Mama3.Dada4.Kaka

15 Mahali ulipo uvimbe
1. Usoni
2. Kwenye shavu
3. Kwenye shingo
4. Kwenye ulimi
5. Kichwani
6. Sehemu nyingine zitaje ……..……..

16 Idadi ya kivimbe/vivimbe
1. Vingi (orodhesha)……, 2.Kimoja

17. Ulishawahi kupata matibabu kabla kutokana na tatizo lako?
1. Ndio
2. Hapana

18 Kama jibu ni ndio katika swali la 16, je ulipata wapi matibabu?
1. Hospitali ya Mkoa
2 Hospitali ya Kanda
3. Hospitali ya Taifa
4. Nje ya nchi

19 Upo katika hatua gani ya matibabu?
1. Ndio ninaanzamatibabu
2. Nipo katika dawa za kumeza wiki /miezi…….
3. Wiki/miezi/miaka…….baada ya upasuaji
4. Hatua ya 1/2/3/4/5/6/7 ya kuchoma sindano
5. Nipo katika ufuatiliaji wiki/miezi/miaka…… baada ya dawa za kumeza/sindano za kuchoma

20 Aina gani ya kipimo/vipimo ulifanya kabla ya matibabu?

1. Doppler Ultrasound / Ultrasound
2. CT Angiograph
3. MR Angiography
4. MRI
5. CT scan
6. Vinginenyo (mf FNAC) …….
7. Sikufanya kipimo chochote

21 Aina gani ya matibabu ulipata?

1. Dawa za kumeza ie. Propranolol
2. Upasuaji
3. Sindano za kuchoma kwenye uvimbe
4. Sindano za kuchoma na upasuaji
5. Kuangalia uvimbe kwa muda

22 Je ulipata matatizo yeyote baada ya matibabu? 1. Ndiyo 2.Hapana

23 Kama ndio katika swali la 22 hapo juu, matatizo gani ulipata?

1. Maumivu
2. Joto kupanda
3. Matatizo ya kifua
4. Matatizo ya ngozi
5. Kuharibika mahala palipochomwa

24 Je ulipewa matibabu mengine baada ya matibabu ya uvimbe?

1. Dawa za kuzuia vijidudu (Antibiotics)
2. Dawa za kuondoa maumivu
3. Dawa ya kuzuia uvimbe (Antiinflamatory drugs)
4. Dawa ya kupaka sehemu ya kuchoma

25 Kama ulichomwa sindano kwa ajili ya matibabu ya uvimbe, ulipewa katika hali gani?

1. Sindano ya kuchoma ya kuondoa maumivu (local anaesthesia)
2. Nilipewa dawa ya kusinzia (sedation)
3. Nilipewa nusu kaputi
4. Nilipewa vidonge vya kuondoa maumivu kabla ya kuchomwa sindano
5. Sikupewa aina yeyote ya dawa kabla

26 Je uliridhika na matokeo ya matibabu yako baada ya kutibiwa?

1. Niliridhika kabisa
2. Niliridhika
3. Niliridhika kiasi tu
4. Sikuridhika
5. Sikuridhika kabisa

Appendix VII: Clinical examination form

Extra oral Examination (R for right and L for left)

Neck (cervical region) (R/L)	1 Yes	2 No
Cheek (R/L)	1 Yes	2 No
Lower lip	1 Yes	2 No
Upper lip	1 Yes	2 No
Submandibular (R/L)	1 Yes	2 No
Forehead	1 Yes	2 No
Scalp	1 Yes	2 No
Preauricular (R/L)	1 Yes	2 No
Periorbital (R/L)	1 Yes	2 No
Parietal region (R/L)	1 Yes	2 No

Others (mention)…….

Intra oral Examination

Tongue	1 Yes	2 No
Buccal mucosa (R/L)	1 Yes	2 No
Labial mucosa	1 Yes	2 No
Floor of the mouth	1 Yes	2 No
Oropharynx	1 Yes	2 No

Characteristics of the vascular lesion

Flat or raised deep blue in color over surface of mucosa	1 Yes	2 No
Deep lesions with bluish tint or hue	1 Yes	2 No
Bright red bosselated	1 Yes	2 No
Firm rubbery in consistency	1 Yes	2 No
Blood cannot be evacuated on pressure application	1 Yes	2 No
Soft consistency	1 Yes	2 No
Multiple cysts in consistency	1 Yes	2 No
Round or oval pink macules	1 Yes	2 No
Red brown or grey	1 Yes	2 No
Pink red cutaneous stains	1 Yes	2 No
Warm	1 Yes	2 No
Palpable thrill or bruits	1 Yes	2 No

Type of vascular lesion

Hemangioma	1 Yes	2 No
Lymphangioma	1 Yes	2 No
Capillarymalformation	1 Yes	2 No
Venous malformation	1 Yes	2 No
Arteriovenous malformation	1 Yes	2 No

Associated complications

Ulceration	1 Yes	2 No
Bleeding	1 Yes	2 No
Pain	1 Yes	2 No
Difficult breathing	1 Yes	2 No
Difficult in swallowing	1 Yes	2 No
Difficult in talking	1 Yes	2 No
Difficult with vision	1 Yes	2 No
Disfigurement	1 Yes	2 No

Treatments needs

Antibiotics and analgesics	1 Yes	2 No
Inj sclerosing agent alone (eg bleomycin, other…)	1 Yes	2 No
Inj sclerosing agent combined (Bleomycin, Lignocaine, N/S, Dexamethasone etc)	1 Yes	2 No
Embolization	1 Yes	2 No
Surgical intervention	1 Yes	2 No
Sclerosing agent and Surgical intervention	1 Yes	2 No
Oral medications (eg propranolol)	1 Yes	2 No
Watchful neglect	1 Yes	2 No
Blood transfusion	1 Yes	2 No
I/V fluids	1 Yes	2 No

Others (mention)……

Phase/s of treatment

S/N	Type of treatment	Duration	Treatment received (ml or mg if inj)	Follow-up duration	Remark
1	Starting, Weight….kg				
2	Oral medications				
3	Intralesional injections	Number of visits			
	Date. …….	1st visit after ……weeks/months			
	Date …….	2nd visit after ……weeks/months			
	Date. …….	3rd visit after ……weeks/months			
	Date ……..	4th visit after ……weeks/months			
	Date. …….	5th visit after ……weeks/months			
	Date …….	6th visit after ……weeks/months			
	Date ……...	7th visit after ……weeks/months			
4	Surgery		Type of surgery and incision;		
5	Injections and surgery		Type of surgery and incision;		

Outcome of treatment

Size of the lesion before treatment (if applicable) 1…….Cm by clinical examination 2…….Cm by CT scan 3……Cm by MRI 4…….Cm by Doppler Ultrasound 5.By photos/pictures

No of Visits (**Mark)**	Size of the lesion in Cm after starting treatment by Clinical exam/CT Scan/MRI/Doppler Ultrasound /Photos or Pictures (**circle appropriate**)	Percentage of reduction
1st		
2nd		
3rd		
4th		
5th		
6th		
7th		

Further treatment

Referred out of the country because of;

1. Extensive lesion
2. Lack of necessary expertise
3. Lack of equipments
4. Lack of supplies for treatment
5. Others (mention)……

Appendix VIII: Letter of ethical clearance

MUHIMBILI NATIONAL HOSPITAL

Cables: "MUHIMBILI"
Telephones: +255-22-2151367-9
FAX: +255-22-2150534
Web: www.mnh.or.tz

Postal Address:
P.O. Box 65000
DAR ES SALAAM
Tanzania

In reply please quote:

MNH/TRC/Permission/2019 12th September, 2018

To: Head,
Dental Services
Muhimbili National hospital

RE: PERMISSION TO COLLECT DATA AT MNH: 2018

Name of Student	Dr. Abbas M. Mungia
Title	**"OCCURANCE AND MANAGEMENT OF ORAL AND MAXILLOFACIAL VASCULAR LESIONS AMONG PATIENTS ATTENDING AT MUHIMBILI NATIONAL HOSPITAL".**
Institution	Muhimbili University of Health and Allied Sciences
Supervisor	Dr. Jeremiah R. Moshy
Period	10/09/2018 to 10/03/2019 (6 months)

You have been permitted to collect data in respect to the undertaking of the above mentioned study.

Please ensure that you abide to the ethical principle and other conditions of yours approval.

Sincerely,

Dr. Joan Rugemalila
Ag. Head of Teaching, Research and Consultancy Coordination Unit

Cc: DSS
Cc: Dr. Abbas M. Mungia

HEAD, TEACHING RESEARCH & CONSULTANCY UNIT
MUHIMBILI NATIONAL HOSPITAL
P. O. BOX 65000
DAR ES SALAAM

MUHIMBILI UNIVERSITY OF HEALTH AND ALLIED SCIENCES

OFFICE OF THE DIRECTOR OF POSTGRADUATE STUDIES

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DAR ES SALAAM
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Tel G/Line: +255-22-2150302/6 Ext. 1015
Direct Line: +255-22-2151378
Telefax: +255-22-2150465
E-mail: dpgs@muhas.ac.tz

Ref. No. DA.287/298/01A/

3rd September, 2018

Dr. Abbas M. Mungia
M.Dent. Oral and Maxillofacial Surgery
MUHAS.

RE: APPROVAL OF ETHICAL CLEARANCE FOR A STUDY TITLED: "OCCURRENCE AND MANAGEMENT OF ORAL AND MAXILLOFACIAL VASCULAR LESIONS AMONG PATIENTS ATTENDING AT MUHIMBILI NATIONAL HOSPITAL DAR ES SALAAM, TANZANIA"

Reference is made to the above heading.

I am pleased to inform you that, the Chairman has, on behalf of the Senate, approved ethical clearance for the above-mentioned study. Hence you may proceed with the planned study.

The ethical clearance is valid for one year only, from 3rd September, 2018 to 2nd September, 2019. In case you do not complete data analysis and dissertation report writing by 2nd September, 2019, you will have to apply for renewal of ethical clearance prior to the expiry date.

Dr. Emmanuel Balandya
ACTING: DIRECTOR OF POSTGRADUATE STUDIES

cc: Director of Research and Publications
cc: Dean, School of Dentistry, MUHAS

MUHIMBILI UNIVERSITY OF HEALTH AND ALLIED SCIENCES

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Ref. No. HD/MUH/T.233//2016

4th September, 2018

Executive Director
Muhimbili National Hospital
P.O. Box 65000
DAR ES SALAAM.

Re: INTRODUCTION LETTER

The bearer of this letter Dr. Abass M. Mungia is a student at Muhimbili University of Health and Allied Sciences (MUHAS) pursuing M.Dent. Oral and Maxillofacial Surgery.

As part of his studies he intends to do a study titled: ***"Occurrence and Management of Oral and Maxillofacial vascular lesions among patients attending at Muhimbili National Hospital Dar es Salaam, Tanzania".***

The research has been approved by the Chairman of University Senate.

Kindly provide him the necessary assistance to facilitate the conduct of his research.

We thank you for your cooperation.

Ms. L.C. Kapama
For: DIRECTOR, POSTGRADUATE STUDIES

cc: Dean, School of Dentistry
cc: Dr. Abbas M. Mungia