International Journal of Recent Advances in Multidisciplinary Topics
Volume 5, Issue 5, May 2024
https://www.ijramt.com | ISSN (Online): 2582-7839


# Pregnancy Tumor: A Controversy

P. Sarita[1], Anandamay Goswami[2*], Santosh Kumar Subudhi[3], Stithaprajna Lenka[4], Someswar Chakraborty[5], Karan Baruah[6], Avijit Garai[7], Rupali Sahu[8]

[1]*Assistant Professor, Department of Gynaecology and Obstetrics, IMS and SUM Hospital, Bhubaneswar, India*
[2,5,6,7,,8]*Post Graduate Trainee, Department of Oral & Maxillofacial Surgery, Institute of Dental Sciences, Bhubaneswar, India*
[3,4]*Professor, Department of Oral & Maxillofacial Surgery, Institute of Dental Sciences, Bhubaneswar, India*

***Abstract*: Pyogenic granuloma is one kind of inflammatory hyperplasia that can occur in the oral cavity. This statement is deceptive because the lesion is not caused by an infection but rather by a number of stimuli, such as hormonal changes, traumatic injuries, low-grade local irritation, or microtrauma from dental cleaning. It is not noticeable in nonpregnant males or females and typically manifests in the gingiva during the second and third trimesters of pregnancy in pregnant patients. Pyrogenic granulomas associated with pregnancy are referred to as pregnancy tumors. In essence, a pyogenic granuloma is a limited, ecstatic granulation tissue formation triggered by irritation. It might manifest intraorally as a raised mass or as a sessile lesion, among other forms. Their normal hue ranges from deep crimson to reddish purple, and they are soft, painless, and between 0.5 and 1 cm in size. They are more common in females and are usually observed on keratinized tissue. Even though excisional surgery is still the preferred course of treatment, additional protocols have been proposed, such as the use of ND: YAG laser, flash lamp pulsed dye laser, cryosurgery, intralesional injection of ethanol or corticosteroids, and sodium tetradecyl sulfate sclerotherapy.**

***Keywords*: Pyogenic granuloma, Pregnancy, Tumour, Gingiva, Inflammation.**

## 1. Introduction

The name "pyogenic granuloma" or "granuloma pyogenicum" was first used by Hartzell in 1904, although HULLIHEN recorded the first instance of PG in English literature in 1844.According to histology, there are two forms of PG: non-LCH type and lobular capillary hemangioma (LCH TYPE) [2]. Progesterone and estrogen levels in the blood rose from a 100:1 ratio in the early stages of pregnancy to about a 1:1 ratio at term. It is well recognized that these hormones have an impact on the immune system during pregnancy [5]. Pyrogenous granulomas, another name for pregnancy tumors, have been shown to arise in 0.2 to 9.6% of cases, depending on gravidity. After the first trimester, they typically appear, grow swiftly, and then typically disappear after birth. On the other hand, if a pregnancy tumor is excessively big, causes pain, bleeds regularly, or causes occlusion issues, it may need to be removed during the second trimester [6]. Pyrogenic granuloma is one kind of inflammatory hyperplasia that occurs. Inflammatory hyperplasia is the collective term for a wide range of nodular growths of the oral mucosa that histologically resemble inflammatory fibrous and granulation tissues. Its genesis is thought to be non-neoplastic. Though it is a common skin illness, it is extremely rare in the gastrointestinal tract, except than the mouth cavity, where keratinization is often observed [7]. The most common area in the oral cavity is the gingiva, and granulomas are most commonly detected anteriorly on the labial section of the gingiva in the maxilla [9]. Hartzel referred to the lesion as a "pyogenic granuloma" or "granuloma pyogenicum," although this term is deceptive because the lesion is not filled with pus [10]. The main histological feature of pyrogenic granulomas is the proliferation of endothelial cells, resulting in capillary lobules that are separated by connective tissue septa [11]. Pyogenic granuloma is a rather frequent condition that mimics a tumor. It is brought on by granulation tissue swelling in reaction to generalized irritation or an infection. Pyogenic granulomas are more common in women, with a 2:1 ratio. Even though pyogenic granulomas can occur at any age [3]. Pyogenic granuloma is a frequent lesion that looks like a tumor. It is brought on by the granulation tissue expanding in reaction to an irritant or non-specific infection. Pyogenic granulomas are more common in females by a ratio of about 2:1. Regardless of the age at which pyogenic granulomas can occur.

The lesion was first identified by Poncet and Dor in 1897. Hartzell later named it "pyogenic granuloma" or "granuloma pyogenicum" in 1904. However, the scientific community considers the term "granuloma" deceptive because it is unrelated to pus and has a different histological appearance. Seventy-five percent of the oral type is found in the lips, tongue, buccal mucosa, palate, and gingiva; the remaining extra gingival locations are the skin, eyes, and other body parts. Lobular capillary hemangiomas is the classification given to these tumors. Compared to the maxilla, the mandible is less involved in the oral cavity. These days, a patient's diagnosis is straightforward and depends on both new methods and a biopsy. Even though the therapy is a challenging issue, it is achievable as medical knowledge grows. PG can grow quickly and often bleed abundantly with little or no injury. PG mostly impacts young women who are in their second decade of life. because of the way that changes in hormones affect the arteries. The pathophysiology of it is generally thought to be greatly impacted by female sex hormones. When the lesion is small, painless, and bleeding-free, oral prophylaxis and the

*Corresponding author: goswamianandamay@gmail.com

elimination of irritating materials are advised. An excised lesion during pregnancy may have a higher recurrence ratio. It is possible to remove PG using cryosurgery, scalpels, and lasers. Laser surgery is the best method for minimal bleeding or suturing, fast surgical recovery, good patient comfort, and ease of use. Based on reported success stories with Er: Yag (Erbium-Doped Yttrium Aluminium Garnet) and diode lasers, it looks like using a laser will reduce the rate of lesion recurrence. Histologically, two areas were found in LCH PG: a lobular area and a superficial, frequently ulcerative area, in accordance with Toida et al.'s criteria. The former was characterized by the lobular proliferation of vascular components, whereas the later was represented by inflammatory granulation tissue beneath the ulcers. Non-LCH PG was shown to have two sections that matched the topography of LCH type: a core area composed of vascular granulation tissue with various degrees of inflammatory cell infiltration, and a surface ulcerative area similar to that observed in LCH PG [10].

## 2. Epidemiology

Some researchers see PG as a benign neoplasm [10], despite the fact that PG is thought to be a reactive tumor-like lesion that develops in response to a range of stimuli, such as chronic low-grade local irritation [3], [11], traumatic damage, hormonal factors [12], or particular types of drugs [13]. It's now believed to be unrelated to infection, even though pyogenic organisms were originally thought to be the cause [2], [3]. Of them, twenty-eight (65.1%) were found on the marginal gingiva, and eleven (11.6%) were found on the alveolar gingiva. a higher propensity for the vestibular gingiva, where 23 individuals (64.7%) were discovered. Of the granulomata on the gingiva, only 12% (four patients) had positive information about trauma; nevertheless, the bulk (70%) of those at the extra gingival regions seemed to have been traumatized [8]. The term "pyogenic granuloma" is misleading because the lesion is not a granuloma and does not contain pus [1], [3], [11], [14]. It should be noted that some authors have proposed that infectious agents, such as Bartonella henselaea (peliosis hepatis), B. henselae and B. quintana (bacillary angiomatosis), and human herpes virus type 8 (Kaposi’s sarcoma and Angio lymphoid hyperplasia), could be a factor in recurrent PG. However, there is no evidence that pathogenic microorganisms are present in larger PG groups [15].

Since trauma is responsible for approximately one-third of lesions [16], the presence of past trauma is not rare, especially in the case of extra gingival PGs [3], [17]. Poor dental hygiene could have been a significant factor for a number of these people [1], [3], [11]. While Milano et al. [19] reported a case of post-gingival recession (PG) associated with aberrant tooth development, Aguilo (2018) confirmed the occurrence of PG subsequent to damage to a primary tooth. Angiogenesis and the rapid growth of PG are known to be facilitated by a number of substances, including basic fibroblast growth factor (Basic fibroblast growth factor [22]), vascular endothelial growth factor [21], and connective tissue growth factor [23]. Among other medications, cyclosporine plays a major role in the development of PG. Four cases of oral PG were described by Bachmeyer et al. [24] and Lee et al. in patients receiving cyclosporine for chronic graft-versus-host disease. Furthermore, several iatrogenic stimulations utilized in dentistry have the potential to cause PG. Fowler et al. [8] reported the first case of PG being linked to controlled tissue regeneration. An allograft of demineralized, freeze-dried bone yielded the lesion when osseous lesions were patched with expanded polytetrafluoroethylene membranes. During bone marrow transplantation (BMT), oral issues might emerge for two reasons: infections and regimen-related toxicity. Although bone marrow transplantation (BMT) seldom results in oral granulomatous lesions, Kanda et al. described a patient who developed parodontal glands (PGs) at an early stage after allogenic BMT for multiple myeloma. Following the discovery of inclusion bodies in fibroblasts in 1980, Davies et al. [27] deduced that there was a disruption in the metabolism of proteins. It was suggested that PG is a lesion brought on by an ape-like tissue organizer as a result of papillary fibroblasts' gene suppression, maybe as a result of a C-type virus infection. It is important to note that a tumor's growth rate is influenced by both the rate of cell death and the proliferative activity of the tumor cells. While oral granulomatous lesions following bone marrow transplantation (BMT) are uncommon, Kanda et al. reported a patient who experienced early development of parodontal glands (PGs) following allogenic BMT for multiple myeloma. In 1980, Davies et al. discovered inclusion bodies in fibroblasts and concluded that this indicated a disturbance in protein metabolism. One idea links PG lesions to ape-like tissue organizers and papillary fibroblast gene suppression, which may be caused by an infection with a C-type virus. It is significant to remember that a tumor's growth rate is determined by both the proliferative activity and the rate of tumor cell demise. According to Epivatianos et al. [9], etiological variables were present in 16% of cases and there was a prevalence in women (1:1.5). In contrast, non-LCH PG was more frequently (86%) linked to etiological factors. 75% of instances of oral PG, the most common gingival tumor in reality, occur in the gingiva, most likely as a result of calculus or foreign debris caught in the gingival fissure. The lips, tongue, and buccal mucosa are the next most commonly encountered sites [1], [3], [11]. The maxillary gingiva has slightly more lesions than the mandibular gingiva; anterior sections have more lesions than posterior regions. Furthermore, these lesions considerably more commonly damage the facial gingiva than the lingual gingiva; in fact, some of these lesions affect both tissues by spanning the space between teeth [3].

## 3. Clinical Features

PG affects all age groups. There is no glaring majority for any one gender. Reddish-colored, smooth- or lobulated, exophytic vascular nodules, or PG, can grow rapidly. Larger lesions sometimes lobulate and develop into pediculated, mushroom-shaped tumors. In PGs, there is frequently bloodshed. Based on their respective rates of proliferation and vascularity, the two forms of PG—the lobular capillary hemangioma (LCH) type and the non-LCH type—have different histologies and clinical symptom. Reports for LCH

types are more frequently sessile, but most reports for non-LCH kinds are pedunculated. Lobular arrangements of proliferating sprouting capillaries and blood arteries characterize the LCH type. Although the non-LCH form has a vascular central core that resembles granulation tissue, the LCH type does not have any fibrous maturation. The perivascular mesenchymal cells in the center region of the non-LCH pyogenic granuloma do not show alpha smooth muscle actin (SMA) binding, in contrast to the lobular area of the LCH type. Therefore, it seems that the pathophysiology of LCH and the non-LCH type takes different paths. Under a microscope, it can be difficult to distinguish between an inflammatory hemangioma and a pyogenic granuloma. Pyogenic granuloma and inflammatory hemangioma can be distinguished from one another with the aid of GLUT-1 stain. The glucose transport type protein GLUT-1 staining positively is seen in an inflammatory hemangioma, whereas it is lacking in a pyogenic granuloma. The main histological features of pyogenic granulomas are capillary lobules separated by connective tissue septa as a result of active endothelial cell development [11]. Guidelines for the treatment of granulomas associated to pregnancy have been published. Delaying intervention until the postpartum period is often preferred since lesions may heal when hormones calm. Given the high recurrence of pyrogenic granulomas associated with port wine stains, complete surgical excision is usually advised. We believe that part of the reason for the recurrence may have been the failure to remove many vascular anastomoses in the interproximal gingiva between the teeth. With these teeth extracted, better access, a more complete excision, and curettage were made possible [6]. Fully grown pyogenic granuloma lesions exhibited a polypoid morphology, consisting of numerous lobules of mature capillaries and venules with plump endothelial cells separated by septa of different thicknesses of connective tissue. These subsequent lesions had less strong inflammatory infiltration, and often the edges of the lesions were partially surrounded by an adnexal epithelial collarette.The two types of PG exhibited different clinical features in [11], as reported by Epivatinos and al. Their results showed that while LCH PG presented as sessile lesions more frequently (66%), non-LCH PG usually developed as pedunculated lesions (77%). The average size of PG is 2.5 cm, and it takes weeks or months for it to grow to its maximum size, after which it will stay in place indefinitely. The lesion develops clinically gradually, painlessly, and without any symptoms. According to Goodman-Topper and Bimstein's research [7], PG rarely causes significant bone loss.

## 4. Etiology

A number of etiologic factors, including physical trauma, persistent low-grade trauma, hormones, microorganisms, and some drugs, can result in post-traumatic growth (PG) [2]. Calculus and bad dental hygiene are common triggers for many people. Nine The second and fifth decades of life exhibit the highest occurrence, with a predilection for women. More often than not, the maxilla rather than the mandible and the front region of the buccal face are affected. It presents clinically as a well-defined, smooth, elevated, exophytic, sessile, or pedunculated growth that is covered in hemorrhagic and erythematous papules that vary in size from a few millimeters to several centimeters. The growth is painless. From a statistical perspective, $p<0.005$ suggests that 2:15% of expecting mothers had pregnancy tumors. The only lesion types that had increases in frequency associated with pregnancy were pyrogenic granuloma and benign migrating glossitis [5].

As evidenced by the fact that only 15% of the tumors are on the alveolar area, the majority of pyogenic granulomas are found on the gingiva's periphery. Given that most gingival granulomata are located on the labial gingiva in the anterior region of the oral vestibule, one may be tempted to hypothesize that regular toothbrushing, which is mainly restricted to vestibular tooth surfaces, plays a significant role in gingival microtrauma and gingiva irritation (Ainamo, 1971). In the current study, two lesions related to gingival recession caused by toothbrush trauma were discovered. Another local irritant that may be important in the development of the granuloma is gingival irritation. In response, bacterial plaque induces this type of inflammation. Pregnancy-related progesterone excess and the use of oral contraceptives can exacerbate it. Therefore, a limited tissue reaction to a non-specific irritant, such as trauma, inflammation, microtrauma, or local irritation, could be used to characterize pyogenic granuloma. The tissue organizer that is triggered by this non-specific irritation might be a C-type virus [9]. The highest diameter of the lesion was 7.7 +/ 3.5 mm, and the most common types of lesions were gingival (34.8%), ulcerated (88.4%), and pedunculated (79%). This information was reported in a recent paper. These results align with the clinical manifestations of LCH PG.10] LCH PG generally manifested as sessile growth in contrast to non-LCH PG. Additionally, non-LCH PG had significant etiological factors more often than LCH PG. The current study established a relationship between non-LCH and LCH PG, with a significantly higher frequency of etiological factors.

It makes plausible to suggest that non-LCH PG could be a reactive hyperplastic lesion given known local etiological variables. One kind of spontaneously developing capillary hemangioma is called LCH PG. It is usually corroborated by the distinct arrangement of blood vessels, the lack of etiological elements, and the sparseness or absence of inflammation in non-ulcerated lesions or in deeper lobules of arteries. As a result, LCH PG may be a benign vascular tumor with an unclear etiological origin, similar to other benign neoplasms of the oral cavity. This work has unquestionably demonstrated the differences between LCH and non-LCH PG in terms of immunohistochemistry, histology, and clinical symptoms. It is plausible that the identified etiological variables in non-LCH PG result in a lesion that histologically resembles a benign vascular tumor, like lobular capillary hemangioma, even if the condition is clinically more frequently sessile. There might be additional variables at work in reparative granulation tissue, which is more often pedunculated and has blood vessels with vascular diameters distinct from those found in LCH PG. When dealing with non-LCH PG, it is important to identify and eradicate the etiological factors in order to stop the lesion from getting bigger or from happening again [10].

## 5. Correlation with Pregnancy

Up to 5% of pregnancies may result in gingival PG development. Pregnancy-related hormonal imbalances increase the organism's sensitivity to irritation [1], but gingivitis is not caused by subclinical hormone abnormalities alone—bacteria plaque and gingival inflammation are both necessary. Because this type of gingivitis develops during pregnancy and resembles that which does not occur in non-pregnant women, it appears to be related to the hormonal condition that is found during pregnancy. An occasional sign of pregnant gingivitis is a predisposition to develop pregnancy granuloma, a form of localized hyperplasia. It typically appears in the second or third month of pregnancy, with a tendency to bleed and possible mastication issues. The persistent presence of plaque in the early months of pregnancy results in gingival catarrhal inflammation, which sets the stage for the later months' development of hyperplastic gingivitis, which is impacted by hormone accumulation. Unmanaged circumstances can lead to PG development. Pregnant ladies are often associated with PGs. Whitaker et al. reported that progesterone or estrogen receptors were more prevalent in PGs. It was thought that these pregnancy-related hormone levels were the reason behind the increased frequency of PGs. This lesion is unusual in women with poor oral hygiene in areas with local exacerbating factors such dental calculus or ill-fitting restorations.

Inadequate dental hygiene or incomplete excision may cause PG to return after surgery performed during pregnancy. The molecular mechanisms governing the rise and regression of PG during pregnancy have been extensively studied. Pregnancy causes a major disruption in the form and function of the skin and mucosa's blood and lymph microvasculature, a condition known as endocrine disruption. Recent studies have discovered that sex hormones have a variety of effects on the body's biochemistry and immune system. In order to form granulation tissue, estrogen stimulates the production of Basic Fibroblast Growth Factor (bFGF), Transforming Growth Factor beta1 (TGF-α1) in fibroblasts, Nerve Growth Factor (NGF) in macrophages, and Granulocyte-Macrophage-Colony Stimulating Factor (GM-CSF) in keratinocytes. These reactions occur more quickly following injury. The effects of estrogen on macrophages that raise VEGF (Vascular Endothelial Growth Factor) production and may contribute to the development of pregnancy-induced gingivitis are countered by antagonists. These sex hormone regulation effects may be modified in PG as a therapeutic or preventative measure. Progesterone suppresses the immune system in the gingival tissues of pregnant women, allowing for a greater chronic tissue reaction rather than a quick acute inflammatory response to plaque, which causes an exaggerated appearance of inflammation in humans, according to research by Ojanotko-Harri et al. It was suggested by Yuan et al. that PG expressed significantly higher amounts of VEGF and bFGF compared to healthy gingiva and periodontium. Additionally, angiostatin was expressed far less in PG than in periodontally involved gingiva and healthy gingiva.

Notably, the exact biochemical mechanism causing pregnancy PG to regress after parturition is yet unknown. Angiopoietin-2 (Ang-2) has been proposed to cause blood vessel regression in the absence of VEGF. Tumor necrosis factor-α (TNF-α) was found to increase the expression of Ang-2 in all studied endothelial cell types, according to Yuan and Lin. The granulomas had the highest level of Ang-2 protein during pregnancy, followed by normal gingiva and those that developed after parturition. VEGF levels were raised in the granulomas during pregnancy and virtually vanished after delivery. Since Ang-2 did not affect the quantity of apoptotic cells after parturition in comparison to pregnancy, microvessels may be protected against apoptosis by VEGF alone or in combination with Ang-2. Pregnancy-related alterations in the mouth are similar to the unique oral symptoms of hormonal oral contraceptive use, such as hyperplastic gingivitis, PG, and enhanced gingiva vascularity. While the precise method by which oral contraceptives influence the vascular changes in the periodontium is unclear, prostaglandin E2 has been proposed as a possible inflammatory process modifier. Nichols et al. discovered that the periodontium lacks steroid hormone receptors, suggesting that progesterone or estrogen may not have a direct function in the development of this lesion, despite the association between PG and oral contraceptives and pregnancy. The unique oral symptoms associated with the use of hormonal oral contraceptives, such as increased gingiva vascularity, PG, and hyperplastic gingivitis, are similar to the changes in the mouth that happen during pregnancy. Prostaglandin E2 has been proposed as a possible regulator of the inflammatory process, even if the precise method by which oral contraceptives effect the vascular changes in the periodontium is unknown. Though there is a connection between PG and oral contraceptives and pregnancy, Nichols et al. discovered that progesterone and estrogen may not directly cause this lesion because the periodontium lacks steroid hormone receptors.

The terms "granuloma gravidarum" and "pregnancy tumor" are commonly used since PG of the gingiva can develop in as many as 5% of pregnancies. Pregnancy-related hormonal imbalances cause the body to react to irritation more intensely. However, bacterial plaque and gingival irritation are necessary for the subclinical hormone changes that lead to gingivitis. It typically appears in the second or third trimester of pregnancy, sometimes with associated bleeding issues and mastication difficulties. Because estrogen increases the production of vascular endothelial growth factor (VEGF) in macrophages, a function that androgens reverse, there may be a connection between estrogen and the development of pregnancy tumors. Progesterone functions as an immunosuppressant in the gingival tissues of expectant mothers, preventing a rapid acute inflammatory response to plaque but allowing for a greater chronic tissue reactivity. This causes an appearance of inflammation that is clinically exaggerated. How pregnancy cancers decrease molecularly after giving birth is yet unknown. Recently, flash lamp pulsed dye laser, sodium tetradecyl sulfate sclerotherapy, and cryosurgery have all been used. 100% ethanol and corticosteroids could be injected intralessionally, particularly for highly recurring lesions.

## 6. Differential Diagnosis

Pyogenic granulomas (PG) can resemble Kaposi sarcoma, infantile hemangiomas, vascular malformations, and Kaposi form hemangioendothelioma, among other vascular tumors. Parulis, peripheral gaint cell granuloma, peripheral ossifying fibroma, hemangioma, peripheral fibroma, and leimyosarcoma are a few potential diagnosis for peripheral glioblastoma (PG). Patients referred to as "Kaposi-like PG" were confirmed to have human herpes virus type 8 [4]. These lesions are not PG; they are real Kaposi sarcomas. Other malignancies that may mimic PC include basal cell carcinoma, malignant melanoma, and benign lymphomas. Individuals with compromised immune systems may be more vulnerable to infections of the deep soft tissues, like phaeohyphomycosis or bartonellosis. In addition, parulis, hemangioma, peripheral fibroma, leimyosarcoma, peripheral gaint cell granuloma, peripheral ossifying fibroma, and post extraction granuloma are included [3]. Histological analysis of removed tissue is required to make a definitive diagnosis of pyogenic granuloma. Surgeon excision along with careful scaling and curettage of neighboring teeth and root surfaces is the corrective strategy for progressive gingivitis (PG), as calculus is a common irritation that can cause recurrence. Since PGs are rarely encapsulated, if surgical excision is not performed completely, they may return. Although the exact cause of PPG is unknown, trauma and elevated levels of progesterone and estrogen have been proposed as possible contributors. [4] Peripheral gaint cell granuloma, peripheral ossifying fibroma, hemangioma, pregnancy tumor, conventional granulation tissue, hyperplastic gingival inflammation, Kaposi's Sarcoma, bacillary angiomatosis, angiosarcoma, and non-Hodgkin's lymphoma are among the conditions that can be differentiated as PG [7]. Warthin-Ostarry stain-positive masses of bacilli are represented by extensive extracellular deposits of pale hematoxyphilic granular material in bacillary angiomatosis, another AIDS-related condition [7]. The lobular growth pattern, well-formed arteries, and cytologically bland endothelial cells that characterize PG set it apart from angiosarcoma [7].

## 7. Treatment

Even though PG has a wealth of treatment options available, selecting the best course of action can be challenging if the condition is big or arises in a place that is challenging to operate on. Excisional biopsy is recommended for the treatment of PG, unless the technique would result in a noticeable deformity in which case incisional biopsy is required. Therefore, the intensity of symptoms determines how PG is managed. Clinical monitoring and follow-up are recommended if the lesion is tiny, painless, and bleeding-free. While the usual treatments for gingival lesions involve conservative surgical excision and removal of causative irritants (calcus, plaque, foreign materials, trauma source), the excision should reach the periosteum and the adjacent teeth should be thoroughly scaled to eliminate the source of ongoing irritation.

Other therapeutic plans have recently been put forth in place of precise surgery. Nd:YAG laser excision of this lesion was described by Powell et al. because to its lower bleeding risk when compared to other surgical procedures. They chose the Nd:YAG laser over the CO2 laser, because of its superior coagulation characteristics. White et al. proposed that laser excision is well tolerated by patients with no adverse effects. They also stated that CO2 and Nd:YAG laser irradiation is successful in surgical treatment.

Meffert et al. found that previously resolute tissue responded effectively to a series of treatments with the pulsed dye laser after using the flash lamp pulsed dye laser on a mass of granulation tissue that did not respond to the standard treatment procedures. Cryosurgery, according to Ishida and Ramos-e-Silva, is a very effective method for treating PG. They said that the oral mucosa is the perfect location for this approach due to its smoothness and wetness. It exhibits excellent cosmetic results and could be the primary option or a backup plan in place of traditional surgery.

In patients experiencing recurrence as a result of insufficient cryosurgery, Ichimiya et al. tried an alternative approach using an injection of absolute "ethanol." They concluded that this therapy was less invasive than surgical excision and appeared to be an alternative therapy for PG. Although conservative treatment by techniques such as cryosurgery, laser surgery, and electrodessication is usually adequate, excisional treatment can often result in scars.

According to Moon et al., most patients had effective lesion clearance using sodium tetradecyl sulfate (STS) sclerotherapy without experiencing significant side effects. Even though this approach requires numerous treatment sessions, they feel that it offers a superior choice than excision due of its simplicity and lack of scarring. The particular and nonspecific activities of STS, which obliterates artery lumina and selectively damages endothelial cells, may mediate the mechanism of their technique's therapeutic effects. Furthermore, non-specific necrotic alterations may result from STS infiltrations into stromal tissues. Pregnancy-related treatment concerns are crucial. Using soft toothbrushes, maintaining good oral hygiene, and removing dental plaque are crucial at this time to prevent pregnant tumors. In the event of uncontrollable bleeding, treatment options should be tailored to the patient's specific needs and may include blood transfusions, supportive therapy, medication to hasten fetal lung maturity, and even termination of the pregnancy in order to preserve the patient's life, as in the case of uncontrollable eclampsia. According to Steelman and Holmes, it is advisable to maintain good dental hygiene and schedule frequent follow-up consultations during pregnancy. When feasible, surgical and periodontal treatment should be finished in the second trimester, with follow-up at home care until after delivery. However, other experts contend that since recurrence is likely in gravid patients, treatment should wait until parturition for best results. Lesional shrinking following delivery may eliminate the need for surgery in pregnant women. Additionally, Parisi et al. treated PG with a series of intralesional corticosteroid injections, especially for lesions that were extremely recurrent.

Re-excision may be required in certain circumstances since up to 16% of lesions return after excision. It is thought that

insufficient excision, neglecting to eliminate etiologic factors, or re-injuring the area are the causes of recurrence. Certain recurrences show up as numerous deep satellite nodules encircling the initial lesion site (Warner-Wilson Jones syndrome). It is important to note that the recurrence incidence of gingival cases is significantly higher than that of lesions from other oral mucosal locations. There have also been suggestions for various therapeutic methods, including sodium tetradecyl sulfate sclerotherapy, intralesional injection of ethanol for corticosteroid injection, cryosurgery, flash lamp pulsed dye laser, and Nd:YAG laser. Following extragingival pyogenic granuloma surgery, recurrences are not common. at conclusion, given the uncommon nature of labial PPG, physicians need to be aware of this growth at this unusual site in order to make an accurate differential diagnosis and ensure appropriate treatment. Its clinical manifestation and gynecological history allow for easy differentiation [4].

## 8. Conclusion

This paper presented an overview on pregnancy tumor.

## References


[1] Gondivkar, S. M., Gadbail, A., & Chole, R. (2010). Oral pregnancy tumor. Contemporary clinical dentistry, 1(3), 190–192.

[2] Fowler, E. B., Cuenin, M. F., Thompson, S. H., Kudryk, V. L., & Billman, M. A. (1996). Pyogenic granuloma associated with guided tissue regeneration: a case report. Journal of periodontology, 67(10), 1011–1015.

[3] Canivell-Zabaleta, M., Martin-Lozano, G., Olmos-Juarez, E., Fontillon-Alberdi, M., & Infante-Cossio, P. (2018). Extragingival Pregnancy Pyogenic Granuloma on the Lip. The Journal of craniofacial surgery, 29(1), e49–e50.

[4] Rockafellow, A., Florin, W., Philipone, E., & Koslovsky, D. (2015). Pregnancy Tumor in a 31-Year-Old Female with a Facial Port-Wine Stain. Case reports in dentistry, 2015, 472605.

[5] Jafarzadeh, H., Sanatkhani, M., & Mohtasham, N. (2006). Oral pyogenic granuloma: a review. Journal of oral science, 48(4), 167–175.

[6] Vilmann, A., Vilmann, P., & Vilmann, H. (1986). Pyogenic granuloma: evaluation of oral conditions. The British journal of oral & maxillofacial surgery, 24(5), 376–382.

[7] Epivatianos, A., Antoniades, D., Zaraboukas, T., Zairi, E., Poulopoulos, A., Kiziridou, A., & Iordanidis, S. (2005). Pyogenic granuloma of the oral cavity: comparative study of its clinicopathological and immunohistochemical features. Pathology international, 55(7), 391–397.

[8] Bragado, R., Bello, E., Requena, L., Renedo, G., Texeiro, E., Alvarez, M. V., Castilla, M. A., & Caramelo, C. (1999). Increased expression of vascular endothelial growth factor in pyogenic granulomas. Acta dermato-venereologica, 79(6), 422–425.

[9] Eversole LR. 3rd ed. Hamilton: BC Decker; 2002. Clinical outline of oral pathology: Diagnosis and treatment; pp. 113–4.

[10] Kanda N, Watanabe S. Regulatory roles of sex hormones in cutaneous biology and immunology. J Dermatol Sci. 2005;38:1–7.

[11] Ojanotko-Harri AO, Harri MP, Hurttia HM, Sewon LA. Altered tissue metabolism of progesterone in pregnancy gingivitis and granuloma. J Clin Periodontol. 1991;18:262–6.

[12] Neville BW, Damm DD, Allen CM, Bouquot JE. 2nd ed. Philadelphia: WB Saunders; 2002. Oral and maxillofacial pathology; pp. 437–95.

[13] Powell JL, Bailey CL, Coopland AT, Otis CN, Frank JL, Meyer I. Nd: YAG laser excision of a giant gingival pyogenic granuloma of pregnancy. Lasers Surg Med. 1994;14:178–83.

[14] Lindenmuller IH, Noil P, Mameghani T, Walter C. CO2 laser-assisted treatment of a giant pyogenic granuloma of the gingival. Int J Dent Hyg. 2010;8(3):249–52.

[15] Gordon-Nunez MA, de vasconcelos M, Benevenuto TG, Lopes MF, et al. Oral pyogenic granuloma: a retrospective analysis of 293 cases in a Brazilian population. J Oral Maxillofac Surg. 2010;68(9):2185–8.

[16] Saravana GH. Oral pyogenic granuloma: a review of 137 cases. Br J Oral Maxillofac Surg. 2009;47(4):318–9.

[17] Goncales ES, Damante JH, Fischer Rubira CM, et al. Pyogenic granuloma on the upper lip: an unusual location. J Appl Oral Sci 2010;18:538–541.

[18] Adeyemo WL, Hassan OO, Ajayi OF. Pregnancy-associated pyogenic granuloma of the lip: a case report. Niger J Med 2011;20:179–180.

[19] Dal Pra KJ, Bernabe DG, Miyahara GI, et al. Extra-gingival pyogenic granuloma displacing teeth. J Craniofac Surg 2017;28:e185–e186.

[20] Mulinari-Santos G, Garcia BT, Taveira LA. Pyogenic granuloma on the upper lip: a rare location. J Craniofac Surg 2017;28:577–578

[21] Manegold-Brauer G, Brauer HU. Oral pregnancy tumour: an update. J Obstet Gynaecol 2014;34:187–188.

[22] Asha V, Dhanya M, Patil BA, et al. An unusual presentation of pyogenic granuloma of the lower lip. Contemp Clin Dent 2014;5:524–526.

[23] Ravi V, Jacob M, Sivakumar A, et al. Pyogenic granuloma of labial mucosa: a misnomer in an anomolous site. J Pharm Bioallied Sci 2012;4(suppl 2):S194–196.

[24] Gondivkar SM, Gadbail A, Chole R. Oral pregnancy tumor. Contemp Clin Dent 2010;1:190–192.

[25] Ashok KA, Ashok L, Sujatha GP. Pyogenic granuloma on the upper labial mucosa: a case report. J Clin Diagn Res 2013;7:1244–1246.