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Plasma cell mucositis: a review

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ABSTRACT

Plasma cell mucositis (PCM) is an infrequent disease arising from internal dysfunctions of unspecified rationale. In this disease, plasma cells increase rapidly into connective tissue enormously. This condition influences upper aerodigestive tract and one or more body orifices. The motive of this review is to focus on assorted treatment modalities, differential diagnosis and etiology of PCM with emphasis on comprehensive history taking. Inspite of the fact that it is an uncommon condition, it necessitates immense evaluation and should be considered as "diagnosis of evaluation". The literature was searched using "Pub Med" and electronic databases from 1981 to 2017. A total of 98 articles were retrieved from 1952-2017. Among the retrieved articles, most of them were case reports related to medical treatment procedures, 7 were short studies and 5 articles reviewed the differential diagnosis of PCM. In latest documentation, emphasis made on comprehensive history picklings to explore the probable cause and allergen, evaluation and performing particular investigations to reach at conclusion. Substantial researches are essential to approach at ultimate result for specific treatment modality for management of PCM.

Keywords: Management of PCM, differential diagnosis, steroids in PCM and updated treatment modalities of PCM.

INTRODUCTION

Zoon et al elucidated PCM initially affecting glans penis and appeared in different parts of body subsequently [1]. The nomenclature for PCM is diverse and has modified many times. The other names for PCM as follows; Plasma cell orificial mucositis, idiopathic plasmacytosis, mucus membrane plasmacytosis of upper aerodigestive tract and oral papillary plasmacytosis [2]. Atypical gingivostomatitis [3], idiopathic gingivostomatitis[4], plasma cell gingivitis [5-8], gingival plasmacytosis [9] and plasma cell chelitis [10-11] altogether are the assorted terms for PCM involving oral cavity. Plasma cell pervading aerodigestive tract have been

termed as "orificial plasma cell mucositis" [12], documented cases were found identical from one another clinically and histopathologically. PCM is chronic in nature [13]. PCM is scrutinized as non neoplastic disease of adults and no records are fetched regarding progression into plasma cell neoplasm [2].

It is assumed that PCM seems as immunological reaction to definite allergens that exist in chewing gum, flavoring mint, dentifrices and cinnamon flavoring products and continual khat chewers (Catha Edulis) [14]. PCM was presumed to be a hypersensitivity reaction which was depicted as contact stomatitis in late 1960s and early 1970s [15]. Preponderance of PCM is in alliance with autoimmune or immunologically mediated disease [2]. Pretentious mucosa visualizes as enormously erythematous, velvet like and occasionally cobblestone or nodular surface [13]. Manifestations embrace oral pain for extended period, dysphagia, hoarseness and pharyngitis [2]. It is seen as intense erythematous macular areas in oral cavity, invariably include marginal and attached gingival or alveolar mucosa and after pretentious further soft tissues such as maxillary and mandibular sulcus or buccal mucosa. Perhaps epithelial sloughing and desquamation and ulcers are indentified [15].

Histologically, PCM proclaims parakeratosis, epithelial hyperplasia, neutrophilic exocytosis and innumerable spongiotic pustules in deprivation of candida [15]. Extramedullary plasmacytoma (EMP) is another disease, pretentious to oral cavity which is in ration of dense submucosal plasma cell infiltrate [2]. Clinical association is obligatory for discrimination[2]. Management incorporates termination of habit and perpetuation of oral health [14]. Topical steroids aid in truncation of inflammation and expedite healing [15].

The recognition of monoclonal or polyclonal augmentation assists in differential diagnosis of diverse plasma cell diseases. The authentication of PCM entails immunohistochemistry (IHC) visualizing polyclonal expression with kappa and lambda chain limitation other than clinical and histopathological examinations [16]. Immunoglobulin free light chains like kappa and lambda are pondered as marker for plasma cell activation. There is truncation of lambda chain expression while preponderance of kappa chain expression is seen [17]. Monoclonal expansion of plasma cells discerns in neoplasms like multiple myeloma and extramedullary plasmacytoma [18]. The probability of neoplastic amendment is yet unidentified [18].

MATERIALS AND METHOD

The review was orchestrated utilizing medical literature exploration and Retrieval System Online (MEDLINE) database and exploration via Pub Med. Furthermore Google Scholar was also used to explore for any pertinent publications. We used a combination of the following keywords: plasma cell mucositis, treatment of mucus membrane plasmacytosis, and treatment update on plasma cell gingivitis, differential diagnosis of plasma cell mucositis.

A total of 98 articles were recouped from search engine. Among the retrieved articles, most of them were case reports which enclose circumstantial history taking to detect probable allergen and diverse medical treatments under the name of PCM, plasma cell stomatitis (PCS), plasma cell chelitis (PCC) and plasma cell gingivitis (PCG) with variable results using different types of treatment modalities. 7 articles comprise of short clinical research and 5 articles discussed the differential diagnosis of PCM.

DISCUSSION

PCM is an infrequent non neoplastic disease in which aerodigestive tract is invaded by plasma cell proliferation[2]. The usual exhibition of PCM is an inflammatory, either involving large area or restrained at particular small erythematous, edematous, macular lesions with cobblestone/nodular or velvet like features with a distinctive hallmark of precise boundary at mucogingival junction [13]. There are areas of whitening, epithelial sloughing, and erosions have also been recognized [13]. Oral pain, pruritis, burning sensation, and bleeding on manipulation are the congruous manifestations [19].

The mean age of pretentious PCM cases is 56.6 years and imperceptible predisposition in male of approximately 1.2:1 according to review by Solomon et al [2]. There are heterogenous presumptions as etiology of PCM from hypersenstivity reaction to chewing gum, cinnamon and foreign substances but no one validated final[8].

Black pepper, black salt, alum, ajwain, mint, clove, and acasia altogether have been documented in literature [21]. It is presumed that immunologic reaction to some allergic antigen aids in the probable cause [21]. There are diverse allergens which have been intimated in literature like mint in toothpaste, cinnamonaldehyde, strong spices, chilies, chewing of khat, and definite ingredients of herbal toothpastes [21]. Subglottic stricture, stenosis and respiratory obstructions are listed as subsidiary impediment of PCM involving trachea and bronchi [22].

The probable causes mentioned in literature are as following; disease of unspecified origin, disease evolved due to some allergen and disease of neoplastic cause. It is believed that this condition occurs as B-cell mediated disease and T-cells enhancing its reaction [23].

Differential diagnosis of PCM [15, 24] incorporates

- Mucus membrane pemphigoid (MMP) and pemphigus Vulgaris (PV): if a patient is continuously exhibiting to undiagnosed allergen then lesion appears. To differentiate MMP and PV with PCM, a biopsy for both standard histology and direct immunofluoroscence should be done.
- Pubertal or Pregnancy induced gingivitis and plaque associated gingivitis: Non specific gingivitis comprises of plasma cell infiltrate, dissimilarity in histopathology is in thickness of plasma cells. It visualizes as disperse red gingival clinically, history of topical agent aids in correct diagnosis.
- Chronic granulomatous gingivitis occurs due to ingredients present in polishing agents like pumice. It appears as sensitive or painful erythematous gingival, history plays an important role in differentiation.
- Mouth breathers show erythematous and occasionally edematous gingiva, adequate history and interconnection with histopathologic features aid in differentiation.
- Allergic or irritant contact dermatitis can be excluded initially by good history and by position of lesions.
- Dentures or foreign objects can be eradicated to differentiate with contact dermatitis. Patch testing is beneficial to determine any other contact or allergic factors.
- PCM can mimic chelitis granulomatosa, histopathology reveals proof of granulomatous infiltrate comparative to plasma cell infiltrate (seen in PCM).

Other possible clinical differential diagnosis of PCM includes lichen planus, plaque-induced gingivitis, erythroplasia, sarcoidosis and Wegner's granulomatosis [14]. To differentiate between these diverse diseases, history pickling should be comprehensive and qualitative to reach at a diagnosis which requisites oral medicine specialist.

Proper investigations should comprise of full blood count, erythrocyte sedimentation rate (ESR), serum antigen angiotensin converting enzyme (SACE), anti basement membrane, anti-intercellular substance and anti- neutrophil cytoplasmic antibodies (ANCA), serum immunoglobulins, plasma electrophoresis, syphilis serology and tissue culture. Patch testing for contact allergens should be incorporated [9].

Monoclonal or polyclonal augmentation helps in differential diagnosis of diverse plasma cell diseases. IHC visualizes polyclonal expression in PCM with kappa and lambda chain restriction other than clinical and histopathological evaluations [16]. Immunoglobulin free light chains like kappa and lambda are markers for plasma cell activation. There is truncation of lambda chain expression while preponderance of kappa chain expression is seen [17]. Monoclonal expansion of plasma cells discerns in neoplasms like multiple myeloma and extramedullary plasmacytoma [18]. The probability of neoplastic amendment is yet unidentified [18].

Additionally IHC establishes the polyclonality of this disease which helps in kappa and lambda light chain reactivity. There is activation of plasma cell induced expression of CD molecules in neoplastic modification is seen. The CD34, CD43, CD117 and Ki67 are the five more markers which are positively stained in malignant cells and negatively stained PCM.

Concurrently it was seen that plasma cells stained positive for CD44, which is crucial for physiologic tasks of normal cells and pathologic tasks of malignant cells. In association with negative staining outcome for CD34, CD43, CD117, Ki67 and CD44 expression predicted suitable to promote growth of plasma cells into

terminally differentiated cells [25]. Therefore CD44-/+ with CD34-, CD43-, CD117- and Ki67- may be one of the subsequent biological markers to be executed in discrimination of malignant or non-malignant nature of these lesions. However specific cause has not been found but termination of toothpaste has proved to be beneficial. Gene rearrangement studies can be performed if IHC is found indecisive [20].

Treatment is generally symptomatic and is regulated regarding the eradication of any allergic or susceptible factors, if any. Corticosteroids (topical, intralesional, systemic) have been endeavored with some triumph. Other topical immunosuppressant like topical tacrolimus [26] (0.3%) and cyclosporine, topical and systemic antifungals such as nystatin and griseofulvin, topical 2% fusidic acid and oral antibiotics are various treatment modalities utilized in PCM. Utilization of chemotherapy (cyclophosphamaide and vincristine) and low dose radiotherapy has also been promulgated in serious lesion. Debulking techniques such as excision by surgery or by CO_2 lasers, electrocoagulation and cryotherapy have rendered momentary benefits followed by recurrence [15, 20].

Plaque control and traditional therapeutic procedures solely are not sufficient to treat these lesions of unidentified cause. Proper dietary history should be recorded by patient, with information of any other health care products utilized into oral cavity. An effort should be made to eradicate the probable allergen to prompt the cause.

Topical or systemic immunosuppressive medications endeavored in many patients where etiology was unidentified which provided variable results in literature review. Some patients did not acknowledge the treatment, and no probable etiology can be identified, regardless of all investigations and therapeutic interventions [27].

Numerous studies exhibited remarkable amelioration entirely abstaining from causative factors and meticulous oral hygiene care. [28-33] Combined treatment utilizing chlorhexidine gluconate mouthrinse was also recorded. [34-37]

Silverman S Jr, Lozada F[38] in 1977 described triumphant treatment of 6 cases from 12 cases by administering systemic prednisone with the doses not exceeding 40 mg/day.

Sollecito TP in 1992 [8] reported the diminution of symptoms and inflammation, but was inadequate to reach complete healing by topical 0.05% clobetasol proprionate or 0.05% fluocinonide.

Mahler V [39] in 1996 tried topical 2% fusidic acid in a tetracaine-containing adhesive ointment applying 4 times/day with benefit [25].

Vaidya P, Gupta U [37] in 2010 narrated a case used topical Fucibet that contained betamethasone and fusidic acid for applying at least 3 times/day with benefit [26].

Aravinda Konidena et al [40], narrated a case in 2014 in which patient acknowledged to 0.1% topical triamcinolone acetonide and 5 mg Levocetirizine, in addition to oral prophylaxis.

Ranganathan AT [41] in 2015 described a case which was treated with modified application of chlorpheniramine maleate 25 mg tablet crusting into powder above the lesion 3 times/day which resulted in complete resolution [27].

Naruemon Panpradit [21] in 2017, narrated a case in which initial dose of prednisolone 25 mg/day (approximately 0.5 mg/kg) showed enormous retrogression within 6 days. After tapering systemic corticosteroid, it was managed by application of fluocinolone acetonide 0.1% in oral paste. Reported case exhibited complete healing and no recurrence within 2 year-period of follow-up, the time to establish finally normal oral mucosa was as long as 8 months.

Kachapilly Arun et al [18], in 2017 described a case of PCM which was treated with intralesional injections of triamcinolone acetonide 40 mg once a week for 3 weeks and gingivectomy was performed with no proof of recurrence.

The predominant aim for treatment of PCM is symptomatic relief. Numerous treatments resulted in disease stabilization but not retrogression [18]. Corticosteroid is pondered as most incessant treatment modality it has dubious results and is of questionable advantage [22]. When disease is pretentious to tracheobronchial structures of distal airways, progressively invasive treatments may be imperative like tracheostomy, laser ablation or radiation therapy.

CONCLUSION

PCM is distinct type of disease which imposes clinicians with a substantial diagnostic conundrum as it imitates numerous other solitary lesions of clinical importance. The significance of comprehensive history-pickling, evaluation of

lesion and proper investigations should be performed, to reach at a definitive diagnosis which demands the oral medicine specialist. PCM should be comprehended in differential diagnosis of swelling or lesion of aerodigestive tract; stereotypical histology can confirm the diagnosis.

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