Potentially Malignant Disorders – the Case for Intervention

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Abstract

Potentially malignant disorders are recognisable mucosal conditions preceding invasive squamous carcinoma development. Established oral cancer remains a lethal and deforming disease, with a rising incidence. Management techniques for identifiable oral precursor lesions have traditionally been polarised between observational and interventional surgical techniques. By defining salient management goals for treating potentially malignant disease, and examining the evidence supporting the efficacy of treatment intervention, this paper presents the case for interventional laser surgery as a definitive diagnostic and treatment modality.

Introduction

Potentially malignant disorders (PMD) are recognisable oral mucosal lesions, primarily leukoplakia, but also erythroleukoplakia, erythroplakia and proliferative verrucous leukoplakia (PVL), which precede in an unpredictable manner invasive squamous cell carcinoma (SCC) development[1,2]. Whilst variable malignant transformation rates have been quoted, a systematic review estimated an overall 12% cancer risk over a mean transformation time of 4.3 years[3].

The ensuing morbidity and high mortality consequent upon SCC diagnosis and treatment remains significant in contemporary clinical practice and, whilst the objectives of treating PMD have been poorly defined, most authorities agree that prevention of malignancy is the priority[4]. This is complicated, however, by the widespread, often multi-focal nature of disease throughout the upper aerodigestive tract[1].

We have previously defined salient management goals supporting an interventional strategy for PMD management, as outlined in Table 1, and also published a series of long-term patient cohort studies demonstrating the efficacy of intra-oral CO_2 laser surgical excision, both as an accurate diagnostic tool and a reliable treatment modality particularly for managing 'high-risk' PMD[4-8]. Following initial histopathological diagnosis and risk assessment, mucosal lesions are excised by laser with margins ablated by vapourisation allowing healing by secondary intention[5]. Evidence from a 590 PMD cohort confirmed that interventional surgery provided definitive diagnosis and treatment, facilitated early SCC recognition, identified patients at risk of progressive disease, rationalised follow-up and helped define clinical outcome data[8].

In a recent journal editorial, however, Guneri & Epstein[9] repeat a number of previously raised concerns regarding the use of surgical intervention in PMD management. Whilst these are summarised in Table 2, of particular concern are the suggestions of 'over-treatment', 'increased cost of care' and the view that PMD excision 'does not show benefit with respect to progression to cancer'.

For many years, of course, PMD management was controversially polarised between surgical excision to remove identifiable mucosal disease and conservative

medical or observational techniques[10]. Shiu & Chen[11] attempted a systematic review of treatment efficacy for leukoplakia, but found wide variation in diagnostic criteria, extensive heterogeneity of treatment protocols, disorganised patient compliance and incomplete follow-up data rendering meaningful analyses impossible.

Review of non-surgical managements, including antioxidant and chemotherapeutic approaches, have shown no proven efficacy, demonstrated high risk for side effects, substantive recurrence following treatment cessation and, most pertinently, no evidence of cancer prevention[10,12,13]. One report observed an increased 31.4% malignant transformation rate in leukoplakia treated with isoretinoin and β-carotene[14], whilst other studies confirmed only 2 to 4% of clinicians would use chemo-preventive agents, emphasising the contemporary redundancy of this modality[15,16].

Whilst 'potential malignancy' is undoubtedly a difficult concept for both patients and clinicians, it intuitively warrants interventional management rather than passive observation[17,18]. van der Waal[19], astutely noting that most PMD patients prefer treatment intervention, recommended surgical excision of localised lesions followed by long-term, specialist follow-up; a view supported by many clinicians[20,21].

The aim of this article, therefore, is to review contemporaneous literature regarding PMD treatment and, by addressing each of the stated PMD treatment goals, attempt to clarify the case for interventional surgical management.

Accurate and Definitive Histo-Pathological Diagnosis

Characteristic of PMD is the variable presence of epithelial disorganisation and dysmaturation, identified microscopically as dysplasia and graded subjectively for severity[1,2]. Although evidence remains weak, it is generally assumed that more severe dysplasia is at greatest risk of cancer development[1,2]. With no predictive biomarkers available, accurate histo-pathological diagnosis remains essential in clinical practice, yet incision biopsies are not necessarily representative of the true and evolving nature of PMD, particularly large and widespread disorders. A

significant ‘under-diagnosis ratio’, varying between 14 to 36%, is seen when incision specimens are compared with their excision counterparts[22-25].

Incision biopsy, therefore, can only be regarded as a ‘provisional’ diagnosis, with whole lesion excision deemed mandatory for ‘definitive’ PMD diagnosis and grading; the latter is facilitated by CO_2 laser excision[2,4,8,26]. Whilst thermal cytological artefacts at excision margins have been suggested as a limitation of laser biopsy, these do not adversely affect histopathology assessment and reporting by experienced oral pathologists[5-8]. In addition, it is also recognised that the absence of dysplasia in incision biopsies does not rule out the risk of pre-existing or developing cancer in individual PMD lesions[25,27].

Multiple lesion disease, particularly widespread and pan-oral presentation which affects around 25% of PMD patients, is undoubtedly more challenging to manage but the technique of ‘field mapping’ whereby multiple-site incision biopsies are performed to delineate more significant foci of dysplastic disease facilitates pragmatic, targeted intervention to high-risk regions[28,29].

Prediction of Clinical Behaviour

The ability to accurately predict clinical outcome for individual patients or lesions remains elusive in clinical practice[4,5]. However, long-term follow-up of defined PMD patient cohorts undergoing coordinated interventional treatment has facilitated documentation of clinical outcome and defined categories of disease free, further PMD disease, malignant (same-site) transformation and new-site oral cancer development[4]. Retrospective analyses have identified predictive features: disease free status is more likely in cases of mild dysplasia, but significantly less common with erthroleukoplakia and lesions exhibiting lichenoid inflammation, whilst further disease occurs more frequently in PVL and in the absence of laser excision[8.] The likelihood of SCC development is increased in erythroleukoplakia, in severe dysplasia and in lesions arising on the floor of mouth and ventro-lateral tongue[8].

As a general observation, the incidence of further disease increases with length of patient follow-up, with non-homogeneous leukoplakia, extensive lesions, more

severe dysplasia and floor of mouth and ventral tongue sites at greatest risk[7]. Continued tobacco smoking and alcohol remain persistent risk factors following laser surgery risking development of further PMD[7,30].

Early Recognition of Malignancy

Sufficient evidence exists to confirm that PMD excision facilitates early recognition of cancer. Review of surgically treated cases shows that 'unexpected' SCCs, often at early invasive stages, are identified histo-pathologically in 7 to 12% of excision specimens [8,23-25,31]; a figure not dissimilar to the 12% transformation rate quoted in systematic review[3] and an important diagnostic and treatment success. The efficacy of intervention is confirmed in our study cohorts because few patients required post-laser oncology treatment and long-term follow-up revealed excellent clinical outcomes[7,8,25].

SCC detection at an early stage enables curative treatment with simple and minor surgical intervention[4,32]. In contrast, the limitations of PMD observation are highlighted by a 25% malignant transformation rate reported in a UK dysplasia clinic where there was no coordinated treatment protocol and SCC diagnoses only made following clinically evident malignant change[32].

Effective Removal of Premalignant Tissue

As mucosal-only conditions, PMDs do not require the extensive treatment necessary for SCC removal or destruction. It seems self-evident to intervene early and remove dysplastic mucosa whilst 'pre-invasive'. Surgery can be performed by scalpel, cutting diathermy or photodynamic therapy but the efficacy of dysplasia excision by CO_2 laser is confirmed in patient cohort studies[5-8]. Dysplasia-free margins or residual foci of mild dysplasia are seen in 75% of treated cases[7,30], but with no significant association between dysplasia in resection margins and clinical outcome, almost certainly due to the ability to extend treatment zones by laser ablating oral cavity margins 2-4mm beyond specimen excision[4].

Whilst minimal intervention has been proposed for less severely dysplastic lesions, treatment of all mucosal pre-malignancy is recommended to reduce risks of recurrence and disease progression consequent upon observing even mildly dysplastic lesions[33,34].

Prevention of Further Disease

Well-defined clinical outcome categories are essential prerequisites to inform clinical practice and improve understanding of PMD natural history[1,2]. Review of CO_2 laser treatment studies shows that 51 to 89% of PMD patients are rendered free of disease, with only 10 to 34% exhibiting further disease[8,35-40]; this contrasts with observational treatment in which 77% of lesions persist[41].

Following successful primary therapy, local PMD recurrence and development of new-site lesions may occur[42], but careful patient follow-up facilitates early identification and further laser treatment can be administered. Whilst most patients in our study series required only 1 laser treatment, a mean number of 2.26 treatments over a mean time of 32.9 months ultimately rendered over 74% of 590 PMD patients disease free despite initial presentation with significant dysplasia[8].

Prevention of Malignant Transformation

It is a fundamental hypothesis, and the most significant justification for PMD intervention, that surgical removal of a dysplastic lesion will reduce the risk of malignant transformation. Studies have shown 15% transformation rates for patients whose lesions were not excised, compared with around 5-6% where lesions were removed[3,43].

Excluding SCCs excised ‘unexpectedly’, we have noted between 2 to 5% of PMD patients developing same or new-site cancer formation during post-treatment follow-up[5-8]. Local surgical excision of dysplastic lesions appears to decrease the risk of same-site malignant transformation, but does not eliminate the risk of new-site oral cancer development[13,34,44]. The clinical consequence is the realization that continued

patient surveillance, regular clinic monitoring and risk factor profiling remain pertinent for all PMD cases following treatment[4].

Patient Acceptability and Minimal Morbidity

As CO_2 laser surgery is recommended as the preferred PMD management choice, it is important that it is both acceptable to patients and post-operative morbidity is low. Significant complications following CO_2 laser treatment are rare and, whilst some patients report post-operative pain, submandibular salivary gland swelling following floor of mouth procedures and lingual nerve dysaesthesia after tongue surgery, these are usually transient, self-limiting and rarely require additional treatment[6,45]. A small number of patients report prolonged complications but these are usually after more extensive surgery, or in those who continue to smoke heavily or consume alcohol after treatment[45].

In general, laser surgery is well tolerated and accepted by patients, aids haemostasis, promotes excellent healing and produces minimal scarring with little functional deficit or patient morbidity[5]. A particular advantage is the ability to repeat excisions or ablations at the same site without compromising oral healing or function[4,5]. No significant adverse quality of life outcomes for PMD patients have been reported, particularly in contrast to the known physical and psycho-social consequences of SCC treatment[46].

Whilst no consensus exists to determine the nature or duration of PMD follow-up, a policy of active surveillance offers patients a number of additional treatment advantages and helps advance understanding of PMD natural history and disease progression[5]; Table 3. Interventional management should be considered cyclical in nature, passing from active surgical excision through to surveillance but returning to surgical intervention upon diagnosis of further disease. It is, therefore, a consistent and determined approach to patient management[4,5].

Cost Effectiveness

Few data exist in the literature to support or refute the concept that early, minimal intervention, essentially secondary or tertiary SCC prevention in 'at risk' populations, is cost-effective, but it is not an unreasonable hypothesis. Rather than increasing cost of care, targeted PMD excision is likely to prove less expensive than the alternative of multiple, repeat incision biopsies performed over many years of observational follow-up[44]. There may be additional cost savings in centralizing services for PMD patients within specialist oral oncology centres[5].

In considering wider aspects of health economics, it is necessary to balance PMD treatment efficacy, cost and value for money with anticipated costs of oncology treatment. Recently introduced UK Department of Health Patient-Level Information and Costing Systems (PLICS) allow estimates of costs incurred by NHS healthcare organizations providing specific treatments. PLICS data from the Newcastle upon Tyne Hospitals NHS Foundation Trust over 6 months (April-October 2015) contrasted diagnostic and surgical services for PMD laser surgery (averaged at around £997.90 per treated patient) with multi-disciplinary SCC care (£5,471.58 per patient). Initial SCC treatment cost more than 5 times PMD management, without considering the significant additional costs of head and neck cancer care, including adjuvant radiotherapy and/or chemotherapy treatments.

Conclusions

Clinical outcomes for oral cancer will only improve by earlier detection of SCC and effective management of precursor lesions with malignant potential[47]. The consequences for an individual patient progressing to SCC are devastating. There seems little to gain from observational strategies, particularly as substantive evidence, summarised in Table 4, supports the diagnostic and treatment efficacy of intervention as a definitive treatment modality. Interventional laser surgery provides readily available, effective, low morbidity treatment which is successful in excising PMD mucosal lesions, facilitates early diagnosis of occult SCC and may help reduce overall risk of SCC development.

In the future, PMD patients will require better stratification into ‘high’ and ‘low’ risk categories, with individually tailored treatment protocols based upon bio-molecular and genetic profiling of cancer risk. Whilst awaiting such refinements, and in the absence of meaningful multi-centre, prospective randomised trials, the contemporary case for interventional management is presented.

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Competing Interests

None declared.

References

1. Thomson PJ. Oral Carcinogenesis. In: PJ Thomson (ed) *Oral Precancer – Diagnosis and Management of Potentially Malignant Disorders.* Chichester: Wiley-Blackwell; 2012. p31-47.

2. van der Waal I. Potentially malignant disorders of the oral and oropharyngeal mucosa; terminology, classification and present concepts of management. *Oral Oncology* 2009 **45** : 317-323.

3. Mehanna HM, Rattay T, Smith J, McConkey CC. Treatment and follow-up of oral dysplasia – a systematic review and meta-analysis. *Head & Neck* 2009 **31** : 1600-1609.

4.Thomson PJ. The role of interventional surgery in oral potentially malignant disorders 2014. *Faculty Dental Journal* **5**: 84-89.

5. Thomson PJ. Management of Oral Precancer. In: PJ Thomson (ed) *Oral Precancer – Diagnosis and Management of Potentially Malignant Disorders.* Chichester: Wiley-Blackwell; 2012. p107-137.

6. Thomson PJ, Wylie J. Interventional laser surgery: an effective surgical and diagnostic tool in oral precancer management. *International Journal of Oral & Maxillofacial Surgery* 2002 **31** :145-153.

7. Diajil AR, Robinson CM, Sloan P, Thomson PJ. Clinical outcome following oral potentially malignant disorder treatment: a 100 patient cohort study. *International Journal of Dentistry* 2013 Article ID 809248, 8 pages http://dx.doi.org/10.1155/2013/809248.

8. Thomson PJ, Goodson ML, Cocks K, Turner JE. Interventional laser surgery for oral potentially malignant disorders: a longitudinal patient cohort study. *International Journal of Oral & Maxillofacial Surgery* 2017 **46** : 337-342.

9. Guneri P, Epstein JB. Why are we still unable to accurately determine the malignant potential or the behaviour of oral mucosal lesions? *Oral Oncology* 2017 http://dx.doi.org/10.1016/j.oraloncology.2017.04.011.

10. Carnelio S, Rodrigues GS, Shenoy R, Fernandes D. A brief review of common oral premalignant lesions with emphasis on their management and cancer prevention. *Indian Journal of Surgery* 2011 **73**: 256-261.

11. Shiu MN, Chen TH. Intervention efficacy and malignant transformation to oral cancer among patients with leukoplakia. *Oncol Report* 2003 **10**: 1683-1692.

12. Lodi G, Sardella A, Bez C, Demarosi F, Carrassi A. Interventions for treating oral leukoplakia. *Cochrane Database of Systematic Reviews* 2006 Issue 4. DOI: 10.1002/14651858.CD001829.pub3.

13. Kumar A, Cascarini L, McCaul JA, Kerawala CJ, Coombes D, Godden D, Brennan PA. How should we manage oral leukoplakia? *British Journal of Oral & Maxillofacial Surgery* 2013 **51**: 377-383.

14. Lee J-J, Hong WK, Hittelman WN, Mao L, Lotan R, Shin DM, Benner SE, Xu X-C, Lee JS, Papadimitrakopoulou, Geyer C, Perez C, Martin JW, El-Naggar AK, Lippman SM. Predicting cancer development in oral leukoplakia: ten years of translational research. *Clinical Cancer Research* 2000 **6**: 1702-1710.

15. Marley JJ, Cowan CG, LameyP-J, Linden GJ, Johnson NW, Warnakulasuriya KAAS. Management of potentially malignant oral mucosal lesions by consultant UK oral and maxillofacial surgeons. *British Journal of Oral & Maxillofacial Surgery* 1996 **34**: 28-36.

16. Kanatas AN, Fisher SE, Lowe D, Ong TK, Mitchell DA, Rogers SN. The configuration of clinics and the use of biopsy and photography in oral premalignancy: a survey of consultants of the British Association of Oral and Maxillofacial Surgeons. *British Journal of Oral & Maxillofacial Surgery* 2011 **49**: 99-105.

17. Mandal S, Agrawal R, Salgotra V, Kohli S. Management of potentially malignant disorders of oral cavity: role of medical therapies. *International Journal of Dental and Health Sciences* 2014 **1**: 187-193.

18. Thomson PJ. Managing oral potentially malignant disorders: A question of risk. *Faculty Dental Journal* 2015 **6**: 186-191

19. van der Waal I. Oral potentially malignant disorders: is malignant transformation predictable and preventable? *Med Oral Patol Oral Cir Bucal* 2014 **19**: e386-390.

20. Thomson PJ, McCaul JA, Ridout F, Hutchison IL. To treat...or not to treat? Clinicians' views on the management of oral potentially malignant disorders. *British Journal of Oral & Maxillofacial Surgery* 2015 **53**: 1027-1031.

21. Field EA, McCarthy CE, Ho MW, Rajlawat BP, Holt D, Rogers SN, Triantafyllou A, Field JK, Shaw RJ. The management of oral epithelial dysplasia: The Liverpool algorithm. *Oral Oncology* 2015 **51**: 883-887.

22. Pentenero M, Carrozzo M, Pagano M, Galliano D, Broccoletti R, Scully C, Gandolfo S. Oral mucosal dysplastic lesions and early squamous cell carcinomas: underdiagnosis from incisional biopsy. *Oral Diseases* 2003 **9**: 68-72.

23. Holmstrup P, Vedtofte P, Reibel J, Stoltze K. Oral premalignant lesions: is a biopsy reliable? *J Oral Pathol Med* 2007 **36**: 262-266.

24. Lee J-J, Hung H-C, Cheng S-J, Chiang C-P, Liu B-Y, Yu C-H, Jeng J-H, Chang H-H, Kok S-H. Factors associated with underdiagnosis from incisional biopsy of oral leukoplakic lesions. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2007 **104**: 217-225.

25. Goodson ML, Thomson PJ (2011). Management of oral carcinoma – benefits of early precancerous intervention. *British Journal of Oral & Maxillofacial Surgery* 2011 **49**: 88-91.

26. Balasundaram I, Payne KFB, Al-Hadad I, Alibhai M, Thomas S, Bhandari R. Is there any benefit in surgery for potentially malignant disorders of the oral cavity? *J Oral Pathol Med* 2013 **43**: 239-244.

27. Goodson ML, Sloan P, Robinson CM, Cocks K, Thomson PJ. Oral Precursor Lesions and Malignant Transformation – Who, Where, What and When? *British Journal of Oral & Maxillofacial Surgery* 2015 **53**: 831-835.

28. Hamadah O, Goodson ML, Thomson PJ. Clinicopathological behaviour of multiple oral dysplastic lesions compared with that of single lesions. *British Journal of Oral & Maxillofacial Surgery* 2010 **48** : 503-506.

29. Thomson PJ, Hamadah O. Cancerisation within the oral cavity. The use of 'field mapping biopsies' in clinical management. *Oral Oncology* 2007 **43**: 20-26.

30. Hamadah O, Thomson PJ. Factors affecting carbon dioxide laser treatment for oral precancer: A patient cohort study. *Lasers in Surgery and Medicine* 2009 **41**: 17-25.

31. Chiesa F, Sala L, Costa L, Moglia D, Mauri M, Podrecca S, Andreola S, Marchesini R, Bandieramonte G, Bartoli C. Excision of oral leukoplakias by CO_2 laser on an out-patient basis: a useful procedure for prevention and early detection of oral carcinomas. *Tumori* 1986 **72** : 307-312.

32. Ho MW, Field EA, Field JK, Risk JM, Rajlawat BP, Rogers SN, Steele JC, Triantafyllou A, Woolgar JA, Lowe D, Shaw RJ. Outcomes of oral squamous cell carcinoma arising from oral epithelial dysplasia: rationale for monitoring premalignant oral lesions in a multidisciplinary clinic. *British Journal of Oral & Maxillofacial Surgery* 2013 **51**: 594-599.

33. Huff KD, Garren KC, Huff MS. A novel, minimally invasive approach to managing mild epithelial dysplasia. *General Dentistry* 2010 March/April: 126-129 www.agd.org.

34. Arnaoutakis D, Bishop J, Westra W, Califano JA. Recurrence patterns and management of oral cavity premalignant lesions. *Oral Oncology* 2013 **49**: 814-817.

35. van der Hem PS, Nauta JMK, van der Wal JE, Roodenburg JL. The results of CO_2 laser surgery in patients with oral leukoplakia: a 25 year follow up. *Oral Oncology* 2005 **41**: 31-37.

36. Yang SW, Tsai CN, Lee YS, Chen TA. Treatment outcome of dysplastic leukoplakia with carbon dioxide laser – emphasis on the factors affecting recurrence. *J Oral Maxillofac Surg* 2011 **69**: 78-87.

37. Jerjes W, Upile T, Hamdoon Z, Al-Khawalde M, Morcos M, Mosse CA, Hopper C. CO_2 laser of oral dysplasia: clinicopathological features of recurrence and malignant transformation. *Lasers Med Sci* 2012 **27**: 169-179.

38. Del Corso G, Bartolomeo Gissi D, Tarsitano A, Costabile E, Marchetti C, Montebugnoli L, Foschini MP. Laser evaporation versus laser excision of oral leukoplakia: a retrospective study with long-term follow-up. *Journal of Cranio-Maxillo-Facial Surgery* 2015 **43**: 763-768.

39. Matsumoto K, Suzuki H, Asai T, Wakabayashi R, Enomoto Y, Kitayama M, Shigeoka M, Kimoto A, Takeuchi J, Yutori H, Komori T. Clinical investigation of carbon dioxide laser treatment for lingual leukoplakia. *Journal of Oral & Maxillofacial Surgery, Medicine & Pathology* 2015 **27**: 493-497.

40. Mogedas-Vegara A, Hueto-Madrid J-A, Chimenos-Kustner E, Bescos-Atin C. The treatment of oral leukoplakia with the CO_2 laser: a retrospective study of 65 patients. *Journal of Cranio-Maxillo-Facial Surgery* 2015 **43**: 677-681.

41. Thomson PJ, Goodson ML, Hamadah O. Cohort studies in oral precancer management: intervention vs observation. *Oral Oncology* 2009 **3(Suppl)**: 116.

42. Wan Y, Liang F, Nakasone T, Sunakawa H. Development of new leukoplakias and leukoplakia-associated second/multiple primary oral cancers: A case report and literature review. *Journal of Oral & Maxillofacial Surgery, Medicine & Pathology* 2014 **26**: 166-169.

43. Lumerman H, Freedman P, Kerpel S. Oral epithelial dysplasia and the development of invasive squamous cell carcinoma. *Oral Surg Oral Med Oral Pathol* 1995 **79**: 321-329.

44. Dost F, LeCao K, Ford PJ, Ades C, Farah CS. Malignant transformation of oral epithelial dysplasia: a real world evaluation of histopathologic grading. *Oral Surg Oral Med Oral Pathol Oral Radiol* 2014 **117**: 343-352.

45. Goodson ML, Sugden K, Kometa S, Thomson PJ. Complications following interventional laser surgery for oral cancer and precancerous lesions. *British Journal of Oral & Maxillofacial Surgery* 2012 **50**: 597-600.

46. Tadakamadla J, Kumar S, Johnson NW. Quality of life in patients with oral potentially malignant disorders: a systematic review. *Oral Surg Oral Med Oral Pathol Oral Radiol* 2015 **119**: 644-655.

47. Mishra R. Biomarkers of oral premalignant epithelial lesions for clinical application. *Oral Oncology* 2012 **48**: 578-584.

TABLE 1: MANAGEMENT GOALS IN TREATING PMD

Management Goals
Accurate Diagnosis
Prediction of Clinical Behaviour
Early Recognition of Malignancy
Removal of Dysplastic Mucosa
Prevention of Further Disease
Prevent Malignant Transformation
Patient Acceptability and Minimal Morbidity
Cost-Effective

TABLE 2: PERCEIVED CRITICISM OF INTERVENTIONAL PMD TREATMENT

Risk of 'over-treatment' of benign lesions unlikely to progress to cancer
Cost of treatment
Intervention does not reduce the risk of cancer
Uncertainty over placement and significance of excision margins
Risk of PMD recurrence
No ability to predict clinical behaviour
Poor functional outcome following treatment of widespread lesions

TABLE 3: ADVANTAGES OF PATIENT FOLLOW-UP AND SURVEILLANCE STRATEGIES

POST- PMD TREATMENT

Assess efficacy of treatment intervention
Recognise treatment complications
Early identification of recurrent or further PMD disease
Early identification of cancer development
Optimal timing and coordination of further treatment intervention
Opportunities to modify patient risk factor behaviour
Effective assessment of long-term patient risk
Improve understanding of PMD natural history

TABLE 4: PMD MANAGEMENT GOALS AND TREATMENT EFFICACY

		Treatment	Modality	
Management Goals	**Observation**	**Medical Therapy**	**Laser Surgery**	**References**
Accurate Diagnosis	No	No	Yes	22, 23, 24, 25, 26, 27, 28, 29
Prediction of Clinical Behaviour	No	No	Possible	4, 7, 8, 30
Early Recognition of Malignancy	No	No	Yes	8, 23, 24, 25, 31, 32
Removal of Dysplastic Mucosa	No	No	Yes	4, 5, 6, 7, 8, 30, 33, 34
Prevention of Further Disease	No	No	Possible	8, 35, 36, 37, 38, 39, 40, 41, 42
Prevent Malignant Transformation	No	No	Possible	3, 4, 5, 6, 7, 8, 13, 34, 43, 44
Patient Acceptability and Minimal Morbidity	No	No	Yes	4, 5, 6, 45, 46
Cost-Effective	?	?	Yes	5, 44