Efficacy of acellular dermal matrix and autogenous connective tissue grafts in the treatment of gingival recession defects among Asians

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Keywords
acellular dermal matrix, Asian, gingival recession, root coverage, subepithelial connective tissue graft.

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Abstract
Introduction: The aim of the present study was to assess the efficacy of acellular dermal matrix (ADM) and subepithelial connective tissue grafts (sCTG) in the treatment of Miller class I and II gingival recession (GR) defects.

Methods: Six patients with eight GR sites were randomly assigned to the test group (GR defects treated with ADM) and control group (GR defects treated with sCTG). Recession height (RH) and width, probing depth, keratinized gingiva, clinical attachment level, and full mouth plaque and bleeding scores were measured at baseline, 3 and 6 months.

Results: The differences in mean changes were insignificant between the two groups in all parameters. In both groups, improvements from baseline to 3 and 6 months were significant for mean RH reduction and clinical attachment gain. A significant increase in the mean keratinized gingiva width was observed in both groups at 3 and 6 months.

Conclusion: ADM and sCTG yield similar outcomes when used in the treatment of GR defects.

Introduction
A variety of treatment regimes have been suggested for the treatment of gingival recession (GR) defects.¹–³ The thin and thick free gingival graft (FGG) techniques are among the earliest strategies used in the treatment of GR defects.⁴–⁶ Although root coverage using FGG has demonstrated acceptable results in shallow GR defects, the procedure is overall ineffective.⁷ It has been reported that the autogenous subepithelial connective tissue grafts (sCTG) are effective in the treatment of GR defects.⁸⁹ Roman et al. reported nearly 70% of root coverage in Miller class I and II GR defects following treatment with sCTG alone, compared to recession sites treated with sCTG and an enamel matrix derivative.⁸

The acellular dermal matrix (ADM) is a derivative of human dermis that is processed to remove the cellular and epidermal components. The intact proteins, collagen fibrillar network, elastin filaments, hyaluronan and proteoglycans, and basement membrane allow ADM to maintain its structural integrity, thereby giving it characteristics of a feasible soft tissue graft material.¹⁰ Studies have shown that root coverage with ADM increases the thickness of the keratinized gingiva compared to when root coverage is attempted without ADM.¹¹,¹² An explanation in this regard could be derived from the fact that collagen fibers present in the ADM membrane facilitate root coverage by stimulating platelet attachment, enhancing fibrin linkage, and having a chemotactic effect on nearby fibroblasts.¹³ However, controversial results have also been reported.¹⁴ Other advantages that suggest ADM as a substitute to FGG include elimination of donor site morbidity and reduction in postoperative discomfort and complications.¹⁵,¹⁶

To our knowledge, from indexed literature, a limited number of studies have compared the clinical efficacy of
ADM with sCTG in the treatment of GR defects; however, the results remain debatable.\textsuperscript{17–20} For example, Novaes \textit{et al.} and Tal \textit{et al.} reported no statistically-significant differences in mean root coverage among GR defects treated with ADM and sCTG.\textsuperscript{21,22} Conversely, Koudale \textit{et al.}, de Souza \textit{et al.}, and Aichelmann-Reidy \textit{et al.} suggested that ADM is a useful substitute for sCTG for root coverage protocols.\textsuperscript{17,19,23}

The biotype of the gingival is usually thin, scalloped, and fragile among the Asian population, which makes them more susceptible to periodontal conditions, including GR, compared to other populations.\textsuperscript{24–26} It is therefore hypothesized that root coverage using ADM could be a suitable alternative in treating both single and multiple recession defects compared to sCTG among Asians. The aim of the present study was to assess the efficacy of ADM and sCTG in the treatment of Miller class I and II GR defects among Asian patients.

\section*{Materials and methods}

\subsection*{Ethical guidelines}

The present study was reviewed and approved by the Ethics Committee of the Faculty of Dentistry, University of Malaya, Kuala Lumpur, Malaysia. All participants were requested to read and sign a consent form before being included in the study.

\subsection*{Inclusion and exclusion criteria}

Only patients of Asian origin with GR defects $\geq 3$ mm in the apico-coronal dimension on facial aspects of incisors, canines, or premolars (with no pathological periodontal pockets, and/or restorations, and/or no alveolar bone loss on periapical radiographs) were included. The following exclusion criteria were imposed: (a) patients with systemic diseases, including poorly-controlled diabetes and prediabetes, HIV/AIDS, renal disorders and hepatitis B and C; (b) habitual tobacco smoking and/or chewing; (c) habitual alcohol consumption; (d) pregnancy; and (e) completely edentulous individuals.

\subsection*{Study participants}

Six patients (three males and three females) with eight sites of Miller class I and II GR defects were included. The mean age of the participants was 37.8 years (range: 23–58 years). The participants belonged to the following ethnic groups: Malays, Chinese, and Indians. These patients were recruited from the outpatient department of the Faculty of Dentistry, University of Malaya, Kuala Lumpur, Malaysia.

\subsection*{Randomization and study grouping}

Randomization was performed using a statistical software program (SPSS version 18; SPSS, Chicago, IL, USA). Depending on the type of treatment adopted for treating GR defects, participants were divided into two groups: the test group and control group. GR defects in the test group were treated using ADM, whereas root coverage in the control group was attempted using sCTG.

\subsection*{Preoperative clinical measurements}

Full mouth plaque scores and bleeding scores were recorded as the percentage of tooth surfaces with plaque and bleeding after probing. Recession height (RH) was measured from the cemento-enamel junction (CEJ) to free gingival margin; recession width at the widest point mesio-distally; probing pocket depths (PPD) and clinical attachment loss as the distance from the base of the pocket to the gingival margin and CEJ, respectively; and keratinized gingiva was measured from the gingival margin to mucogingival junction (MGJ). These measurements were made by one investigator (IMS) to the nearest half millimeter using a graded probe (PCP-UNC 15; Hu-Friedy Manufacturing, Chicago, IL, USA). Acrylic stents were used as reference points to determine the exact sites of measurements, ensuring reproducibility during re-evaluation assessments for all the patients. Following the clinical measurements, the patients performed oral rinses with 0.12% chlorhexidine gluconate solution (Oradex; Cavico [M] Sdn Bhd, Puchong, Malaysia).

\subsection*{Surgical protocol}

All surgical procedures were performed by one operator (IMS). Local anesthesia (Denkan [1] injection, Duopharma (M) Sdn Bhd, Klang, Malaysia) was administered on the buccal aspect of the experimental tooth. Using a no. 15 surgical blade (Hu-Friedy Manufacturing), an intrasulcular horizontal incision was made on the labial aspect of the involved tooth without jeopardizing the gingival margin of the adjacent teeth (Figure 1a and 1b). Two oblique vertical incisions were extended beyond the MGJ, and a mucoperiosteal flap was raised up to the MGJ. The upper parts of the adjacent papillae were de-epithelialized. A partial-thickness flap was reflected (Figure 1b) using a double-sided microsurgical blade (Hu-Friedy Manufacturing). To facilitate passive repositioning of the flap, periosteal fibers were dissected at the apical extent of the flap. The exposed root surfaces were scaled and planed using Gracey’s curettes (Hu-Friedy Manufacturing) and ultrasonic scalers (Satelec, Merignac, France), and any obvious irregularities and/or sharp edges were smoothened with a
pear-shaped diamond bur. These surfaces were then conditioned with tetracycline solution (125 mg/mL saline) (Figure 1c).

In the test group, the denuded root surface was covered with the ADM after being aseptically rehydrated in sterile saline according to the manufacturer’s instructions (Figure 1d). The graft was trimmed in a manner so that the connective tissue (dermal side) was placed against the denuded root surface, with the basement membrane facing up (Figure 1e). The coronal and lateral border of the ADM was sutured to lingual gingival tissue using resorbable sutures (VICRYL; Johnson & Johnson, Livingston, UK) (Figure 1f).

In the control group, GR defects received sCTG harvested from the hard palate. The sCTG procedure carried out in this study was a modification of the method described by Langer and Langer.27 The thickness of the palatal mucosa was assessed by sounding the bone with a probe (PCP-UNC 15; Hu-Friedy Manufacturing). Initially, a horizontal incision almost parallel to the gingival margin was made with a vertical incision on the mesial end creating an L-shaped trap door (Figure 2a). A partial-thickness flap was reflected, and a connective tissue graft of approximately 1.5–2 mm thickness was harvested (Figure 2a). The palatal flap was repositioned and sutured using resorbable sutures (VICRYL, Johnson & Johnson)
(Figure 2b). The donor grafts were then secured to the periosteum and adjacent connective tissue. The overlying partial-thickness mucosal flaps were coronally advanced and positioned to cover the sCTG (Figure 2c and 2d).

**Statistical analysis**

The statistical analysis was performed using a software program (SPSS version 18; SPSS, USA). Descriptive statistics were expressed as means ± standard deviations. As the sample size was small, the effect size was reported to assess the strength of the relationship of the treatment offered and to determine whether a statistically-significant difference is a difference of practical concern. Analysis of the clinical data between the experimental groups was made by comparing the mean values at baseline, 3 and 6 months, as well as mean changes throughout the study period.

**Results**

At baseline, parameters of GR defects were similar among sites in the test and control groups (Table 1). At the 3- and 6-month intervals, there was no statistically-significant difference in the percentage of root coverage among sites in the test group (71.75 ± 19.12%) compared to the control group (58.25 ± 21.56%, 54.25 ± 34.24%). No significant difference in root coverage was observed among the test and control groups at the 3- and 6-month follow-up periods (Table 2). At 3 and 6 months’ follow up, there was significant improvement in the clinical parameters (RH and clinical attachment level) among the GR sites in the test and control groups (Table 3 and Table 4). There was significant difference in the change in clinical parameters among the test and control sites at the 3- and 6-month follow-up periods (Table 5). In the test and control groups, RH was significantly reduced after 3 months of treatment, and the remaining clinical parameters showed no significance at the 3- and 6-month follow-up periods (Table 6).

**Discussion**

The present clinical results showed no significant differences in the efficacy of ADM and sCTG in the treatment of GR defects. Our results are in accordance with a recent experimental investigation. Placement of sCTG under a coronally-repositioned flap allows the connective tissue of the palatal masticatory mucosa to induce differentiation of the epithelial cells of the thin covering flap into a keratinizing cell. As sCTG has been reported to be effective for the treatment of GR defects, it was selected as the treatment regimen in the control group, as compared to sites in the test group, which were treated with ADM.

In the present study, there was no significant difference in the clinical parameters measured at the 3- and 6-month intervals. These results are in contradiction to earlier studies, which showed that root coverage with ADM increased the thickness of keratinized gingiva compared to when root coverage was attempted in the absence of ADM. In an experimental study, Owens and Yukna investigated the resorption rates of various barrier membranes in the oral cavity of dogs. The results showed that at 1 month, all membranes underwent some degree of degradation, whereas at 3 months, all membrane types had undergone severe degradation. In this context, it is possible that the resorption of the ADM (within the first 3 months) could have inhibited new tissue-forming cells from populating at the test sites, thereby yielding results similar to those of the control sites. This implies that both surgical procedures are useful in clinical practice and are almost equally effective in their objective; however, it is notable that the findings might have been influenced by the small sample size.

Previous studies have reported that the size of the defect might influence the overall outcome of root-coverage.
procedures. Pini-Prato et al. showed root coverage to be greater when recession was >4.98 mm. In the present study, the size of the defect was nearly 4 mm; however, no significant influence of defect size on root coverage was observed. Further studies regarding the influence of GR size on root coverage are required.

It is well known that systemic disorders (i.e. poorly-controlled diabetes and impaired glucose tolerance) and habits (e.g. tobacco smoking and chewing) jeopardize periodontal health, as well as healing. We can therefore speculate that the clinical outcomes of ADM and sCTG treatments might vary among immunosuppressed patients and habitual tobacco product users as compared to systemically-healthy individuals and individuals not using tobacco in any form. However, further studies are needed in this regard. Another limitation of the present study is that root-coverage protocols were performed in the absence of adjunct growth factor therapy. In a recent study by Rubins et al. recombinant human platelet-derived growth factor BB (rhPDGF-BB) was combined with sCTG for the treatment of Miller class I or II GR defects. The 6-month follow-up results showed improved outcomes for keratinized tissue gains and the percentage of root coverage as compared to the controls (GR defects covered with sCTG without using rhPDGF-BB). Whether the use of rhPDGF-BB with ADM influences its efficacy in root-coverage protocols requires further investigation.

Conclusion

Within the limits of the present investigation, it is concluded that ADM and sCTG yield similar outcomes when used in the treatment of GR defects.
### Table 5. Changes in clinical parameters from 3 to 6 months’ follow up within the test and control groups

<table>
<thead>
<tr>
<th>Clinical parameter</th>
<th>Study group</th>
<th>3-month follow up</th>
<th>6-month follow up</th>
<th>Change (3–6 months)</th>
<th>P-value</th>
<th>Effect size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recession height (mm)</td>
<td>Test group</td>
<td>1.25 ± 0.96</td>
<td>0.96</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>1.25 ± 0.65</td>
<td>1.38 ± 1.03</td>
<td>–0.13 ± 0.48</td>
<td>0.63</td>
<td>0.26</td>
</tr>
<tr>
<td>Recession width (mm)</td>
<td>Test group</td>
<td>2.00 ± 1.41</td>
<td>2.00 ± 1.41</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>2.75 ± 0.96</td>
<td>2.25 ± 1.50</td>
<td>0.50 ± 2.38</td>
<td>0.70</td>
<td>0.21</td>
</tr>
<tr>
<td>Probing depth (mm)</td>
<td>Test group</td>
<td>1.25 ± 0.50</td>
<td>1.00 ± 0.00</td>
<td>0.25 ± 0.50</td>
<td>0.39</td>
<td>0.50</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>1.25 ± 0.50</td>
<td>2.00 ± 1.00</td>
<td>0.25 ± 0.50</td>
<td>0.39</td>
<td>0.50</td>
</tr>
<tr>
<td>Clinical attachment loss (mm)</td>
<td>Test group</td>
<td>2.50 ± 1.00</td>
<td>2.25 ± 0.96</td>
<td>0.25 ± 0.50</td>
<td>0.39</td>
<td>0.50</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>2.50 ± 0.71</td>
<td>2.38 ± 1.03</td>
<td>0.13 ± 0.48</td>
<td>0.63</td>
<td>0.26</td>
</tr>
<tr>
<td>Thickness of keratinized gingiva (mm)</td>
<td>Test group</td>
<td>4.25 ± 0.50</td>
<td>4.25 ± 0.50</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>5.50 ± 1.91</td>
<td>5.25 ± 1.50</td>
<td>0.25 ± 0.50</td>
<td>0.39</td>
<td>0.50</td>
</tr>
<tr>
<td>Plaque score (%)</td>
<td>Test group</td>
<td>15.55 ± 3.55</td>
<td>12.55 ± 2.83</td>
<td>3.00 ± 2.72</td>
<td>0.11</td>
<td>1.10</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>13.98 ± 2.29</td>
<td>11.25 ± 1.70</td>
<td>3.00 ± 2.72</td>
<td>0.11</td>
<td>1.10</td>
</tr>
<tr>
<td>Bleeding score (%)</td>
<td>Test group</td>
<td>16.15 ± 2.01</td>
<td>14.10 ± 3.49</td>
<td>2.05 ± 2.37</td>
<td>0.182</td>
<td>0.87</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>8.85 ± 5.90</td>
<td>7.50 ± 5.10</td>
<td>1.35 ± 1.57</td>
<td>0.183</td>
<td>0.86</td>
</tr>
</tbody>
</table>

### Table 6. Comparison of the mean changes in clinical parameters between the test and control groups

<table>
<thead>
<tr>
<th>Clinical parameter</th>
<th>Study group</th>
<th>Change in clinical parameters (mean ± SD)</th>
<th>Difference in changes in clinical parameters (mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Baseline to 3-month follow up</td>
<td>Baseline to 6-month follow up</td>
</tr>
<tr>
<td>Recession height (mm)</td>
<td>Test group</td>
<td>2.75 ± 0.50</td>
<td>2.75 ± 0.50</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>1.75 ± 0.65</td>
<td>1.63 ± 1.03</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.049*</td>
<td>0.097</td>
</tr>
<tr>
<td>Recession width (mm)</td>
<td>Test group</td>
<td>1.00 ± 1.41</td>
<td>1.00 ± 1.41</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>1.00 ± 0.82</td>
<td>1.50 ± 1.73</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0</td>
<td>0.32</td>
</tr>
<tr>
<td>Probing depth (mm)</td>
<td>Test group</td>
<td>0.25 ± 0.50</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>0.25 ± 0.50</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0</td>
<td>0.32</td>
</tr>
<tr>
<td>Clinical attachment gain (mm)</td>
<td>Test group</td>
<td>2.50 ± 1.00</td>
<td>2.75 ± 0.50</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>1.50 ± 0.71</td>
<td>1.63 ± 1.03</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.154</td>
<td>0.097</td>
</tr>
<tr>
<td>Thickness of keratinized gingiva (mm)</td>
<td>Test group</td>
<td>3.00 ± 1.41</td>
<td>3.00 ± 1.41</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>2.00 ± 2.16</td>
<td>1.75 ± 1.89</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.468</td>
<td>0.331</td>
</tr>
<tr>
<td>Plaque score (%)</td>
<td>Test group</td>
<td>0.00 ± 0.65</td>
<td>3.45 ± 3.23</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>0.00 ± 0.65</td>
<td>2.73 ± 2.17</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.55</td>
<td>0.75</td>
</tr>
<tr>
<td>Bleeding score (%)</td>
<td>Test group</td>
<td>0.90 ± 1.04</td>
<td>2.95 ± 1.98</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>2.80 ± 3.33</td>
<td>4.15 ± 2.99</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.317</td>
<td>0.528</td>
</tr>
</tbody>
</table>

SD, standard deviation. *P < 0.05.
Acknowledgment

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