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Evaluation of the effectiveness of Eladi Keram for the treatment of Acne vulgaris: randomised controlled pilot study

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ABSTRACT

Introduction: Acne is a multifactorial and common skin disease which can significantly affect the quality of life of sufferers. In this study, a topical herbal preparation traditionally used in Ayurvedic medicine was evaluated as a treatment for individuals with acne on their shoulders and backs.

Methods: Study participants were randomly assigned to treatment (Eladi Keram) or vehicle control (coconut oil) groups under double blind conditions and instructed on its daily home application. Standardised lesion counting and acne grading were conducted in accordance with US Food and Drug Administration guidelines and with reference to the Leeds Acne Grading Technique. Participants were assessed for severity of the condition at commencement and on day 28 of treatment.

Results: The treatment group showed typical improvements of 42% (p<0.005) on the Investigators Global Assessment scale, a 60% (p<0.05) reduction in inflammatory lesions, a 59% (p<0.05) reduction in non-inflammatory lesions, and a 59% (p<0.005) reduction in combined lesion count. The control group showed no statistically significant changes for these criteria.

Conclusion: This study is the first reported clinical evaluation of Eladi Keram as a treatment for acne and findings suggest that it could be effective in reducing inflammatory and non-inflammatory lesions, warranting further investigation by means of a larger scale clinical trial.
1. Introduction
Acne vulgaris is a common chronic skin disease, predominant in adolescence but also affecting a large number of adults [1,2,3]. Although acne is not associated directly with mortality or conventionally defined morbidity, the discomfort, risk of scarring and often strong emotional distress associated with the condition means it is increasingly considered a valid target for treatment rather than a condition to be endured [4,5]. Acne is defined by the presence of inflamed red papules, pustules, comedones (black or whiteheads) and the pathogenesis of the condition involves a complex sequence of events, including sebum production, hyperkeratinisation, poral occlusion, colonisation by \textit{Propionibacterium acnes} (\textit{P. acnes}) bacteria and a persistent inflammatory immune response [6]. Microcomedones are often the initial subclinical acne lesions which further mature into non-inflammatory comedones and/or inflammatory lesions [7,8]. The development of microcomedone is via hyperkeratinisation (keratin/infundibular plug) in the follicular infundibulum and sebaceous ducts [8]. Follicular epithelial hyperproliferation leads to hyperkeratosis and eventually to the formation of microcomedones and this may be promoted by increased sebum production. Hyperkeratosis is characterised by increased number and size of keratohyaline granules, lipid droplet accumulation, and epidermal scale/keratin flakes [9]. Consequent alteration in lipid composition of sebum, bacterial overgrowth, and hormonal factors elicit stimulation of the immune and inflammatory responses via the action of CD3$^+$, CD4$^+$ (lymphocytes) and macrophages [6,8,9]. The local overproduction of androgen hormones including testosterone, dehydroepiandrosterone sulfate,
and dihydrotestosterone can regulate sebaceous gland growth and sebum production and consequently the formation of acne lesions [7, 9,10,11].

Furthermore, androgen is involved in comedogenesis and hyperkeratinisation via regulating growth factors and IL-1α [8]. Insulin-like growth factor-1 production, stimulated by growth hormone, acts on sebaceous glands by causing their growth and stimulating lipogenesis [7,8,9]. Under certain circumstances, P. acnes, a common commensal Gram-positive anaerobic bacterium (usually a benign inhabitant of sebaceous follicles) may act directly or indirectly on pilosebaceous ducts and activate certain inflammatory proteins (including the pro-inflammatory cytokines) [12] causing inflammation and hyperkeratinisation [8]. However, it has also been reported that comedones can develop in the absence of P. acnes [13].

A range of conventional treatments for acne exist including oral and topical retinoids, antibiotics, benzoyl peroxide and hormonal treatments [14], but concerns over side effects of retinoids including severe depression [15] and the development of antibiotic resistance to P. acnes [16] have created an appetite for novel treatments, including the use of herbal preparations [17,18].

Eladi Keram is a commercially available Ayurvedic herbal formulation with a long standing anecdotal evidence base as an effective treatment for various skin conditions including acne (referred in classical Ayurvedic text as “pidaka” a form of Kushta (skin disease)) [19,20]. The fact that this ancient formula (Eladi Keram) is still widely manufactured and prescribed by Ayurvedic practitioners, highlights the cultural importance of these formulations to the rich
ethnopharmacological tradition of Indian medicine. Although Eladi Keram has a long standing anecdotal evidence base as an effective treatment for various skin conditions, prior to this study, there has been no reported clinical evaluation using a biomedical model of research. It is on this basis together with interest in novel treatments and potential reduced side effects of herbal remedies that Eladi Keram was deemed worthy of investigation.

2. Materials and methods

2.1. Study design and study groups

A randomised controlled pilot study was conducted to evaluate the effectiveness of Eladi Keram for participants with acne on their shoulders and backs in a double-blinded clinical observation. Eladi Keram was selected for the study because it met our selection criteria of being a commercially available traditional medicine with long-standing use, and with claims of efficacy by traditional medicine practitioners and users. The study deliberately focused on subjects with acne on the shoulders and backs rather than on the face. Although greater emotional distress is associated with facial acne [3] and therefore its targeting may be more beneficial, this is countered by the greater likelihood of participants self-treating and/or masking facial acne during the trial. This would lead to the increased possibility of introducing confounding variables in such subjects suggesting that a study on back and shoulder acne would in practice be more scientifically robust. Moreover, any potential benefits noted in the treatment of back and shoulder acne are also likely to be relevant to subjects with facial acne.
There is a higher prevalence of acne in 15 – 24 year olds but some older people have been confirmed as sufferers as evidenced by UK GP returns analysis [21]. In this study, under 18 year olds were excluded from the study due to ethical and recruitment issues. The present clinical observation study was conducted on participants, aged 19 - 50 (mean = 30.05 and median = 30), reporting acne on shoulders or chest unless they reported the following exclusion criteria: being pregnant, breast feeding, taking other medication for acne, using sun tanning lamps/planning travel to sunny climates, or reporting shellfish allergy. A power calculation (see Statistical Analysis) indicated that approximately 20 participants were required to demonstrate a statistically significant and clinically meaningful reduction in acne. From 24 enquiries, three recruits did not fulfil the inclusion criteria. A total of 21 participants (6 male and 15 female) therefore were enrolled. Ten were randomly (using Minitab® statistical software random data sampling function (Minitab Ltd. Coventry, U.K.)) assigned to the treatment group and eleven to the control group.

Ethical approval was granted by Middlesex University Ethics Natural Sciences Sub-Committee and written informed consent was obtained from each participant prior to study commencement.

2.2. Test samples

Eladi Keram was purchased from Nagarjuna Ayurvedic Group (http://www.nagarjunaayurveda.com/) and the constituent herbs were
authenticated by the manufacturers using thin layer chromatography fingerprinting according to the Indian Government Good Manufacturing Practice. Eladi Keram contains 27 dried herbal ingredients (Table 1) prepared in coconut oil (Cocos nucifera) as detailed in Ayurvedic texts including Nishteswar and Vidyanath [19] and Sharma [20]. Except for trace amounts of oyster shell, Eladi Keram does not contain any animal ingredients, nor herbs threatened with extinction as listed in the International Union for Conservation of Nature (I.U.C.N.) Red Data List of Medicinal Plants. Coconut was used as the control skin agent and the oil was purchased from KTC Wednesbury, England.

The treatment and control test samples (200 mL) were placed in plain polyethylene cosmetic bottles (Ampulla U.K. Limited, Hyde, Cheshire) labelled once and randomly (using Minitab® random data sampling function) re-labelled in the primary investigator's absence by a trusted third party. Thus, neither participant nor researcher was aware of the identity of the treatment administered. Unblinding took place after trial completion and results collation/analysis, thus triple-blinding the study.

2.3. Assessment of the effect of Eladi Keram and coconut oil on acne

Participants attended enrolment consultations where they were assessed and provided written consent prior to being given a patch test and being shown how to apply the topical medication, either the treatment formulation (Eladi Keram) or the vehicle control (coconut oil). Briefly, participants were instructed to place the container in warm water to liquefy the contents prior to shaking the bottle
gently and then applying approximately 5 mL to the affected areas once a day. They were also instructed not to apply any other product during the study.

Clinical assessment was undertaken according to the Investigator’s Static Global Assessment Scale (IGA scale), recommended by the US Food and Drug Administration [22]. Grades were recorded, alongside lesion counts documenting inflammatory and non-inflammatory lesions as separate categories on an anonymous record sheet coded with participant identifier number. Photographs were taken of each participant’s back and shoulders for an ancillary visual record. The IGA scale builds on grading systems originating in the seminal research of Burke and Cunliffe [23]. Using this scale, 0 is the absence of lesions and 4 indicates the most severe condition. Reduction to 1 or 0 can be viewed as a successful treatment, as can a 2-point reduction in severity which was also deemed as a clinically meaningful effect size for sample determination (Section 2.4).

On day 28 of self-treatment, participants returned for debriefing and assessment. Nineteen of the 21 participants returned for follow up. Five participants elected to continue with treatment and were monitored beyond 28 days. These participants were requested to return on day 56 for assessment. This allowed longer range data to be collected which are also reported herein.

2.4. Statistical Analysis
Statistical analyses were performed using Minitab® statistical software. Sample size was determined using a power calculation on the basis that a 2-point reduction on the IGA scale was considered a clinically meaningful effect size. A conventional approach of a 4:1 ratio of β:α risk was employed and therefore a power value of 0.8 and a significance value of 0.05 together with a conservative a priori assumption of an anticipated standard deviation of 1.5 points in the sample IGA scores were used for the sample size calculation. Using these parameters, a sample size of 10 in each group achieved a power of 0.805. Significance of shifts within control and treatment groups from day 0 - day 28 (and thence for some subjects to day 56) were analysed using paired two-tailed t tests. Comparisons of these shifts between treatment and control group were conducted using two sample (independent) t tests. Assumptions of homogeneity of variance and underlying normality of distributions were tested using standard equal variance testing and the Anderson-Darling normality test as appropriate. Ordinal data (IGA scale) was analysed using the Mann–Whitney U test. Comparisons to other studies (where raw measurements could not be directly compared) were made using Cohen’s-d measure.

3. Results

3.1. The effect of Eladi Keram and coconut oil on acne

Summarised results for inflammatory lesion counts, non-inflammatory lesion counts, combined lesion counts and the IGA scale for the control (n=11) and treatment (n=10) groups are presented in Fig. 1. The 95% confidence intervals for the mean shifts in lesion counts and IGA scale measures in the treatment
group strongly suggest significant improvements in this group. In contrast, the lesion count and IGA measures show insufficient evidence of any improvement in the control group, with some measures indicating a small deterioration in skin condition.

The mean changes observed at 28 days were directly compared for the treatment group versus the control group for the three lesion counts using independent two sample *t* tests. Inflammatory lesion count showed a mean improvement of -8 (*p*=0.037); non-inflammatory lesion count an improvement of -16 (*p*=0.011) and the combined lesion count gave an improvement of -24 (*p*=0.003). IGA changes were subjected to the Mann-Whitney *U* test which showed a median shift of 1 scale grade unit (*p*=0.007). These results
summarised in Fig. 1 show the significant improvement made in the treatment group compared to the control group across the four assessed parameters. None of the four effect measures yielded significant results in the control group. Two participants did not complete the trial and stated that they had abandoned treatment due to a lack of improvement in their condition. Under standard protocol these subjects were recorded as having static results unchanged after treatment. Unblinding revealed both were in the control group, an outcome which was concomitant with the control group results as well as the overall study findings.

Five participants in the treatment group opted to continue with treatment on an open label basis. After 56 days, these participants continued to improve, with three criteria yielding further statistically significant mean improvements compared to the 28 days results: non-inflammatory lesion count -25 (p=0.033); combined lesion count -31 (p=0.045); IGA score -1 (p=0.033). The exception was the inflammatory lesion count change which had a non-statistically significant improvement of -6 (p=0.178). No adverse reactions were reported by any participant suggesting that a future larger scale study could consider longer treatment duration.

3.2. Review of the anti-bacterial and anti-inflammatory effect of constituents of Eladi Keram from the literature

Eladi Keram contains 27 herbs; 15 have been reported to possess antibacterial properties and 22 to have anti-inflammatory properties. Eleven herbs are reputed to possess both antibacterial and anti-inflammatory properties.
Table 1 Herbs used in the formulation of Eladi Keram and their reported actions

<table>
<thead>
<tr>
<th>Scientific name</th>
<th>Sanskrit name</th>
<th>Anti-bacterial</th>
<th>Anti-inflammatory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Actinopteris dichotoma Kuhn (Actinopteridaceae)</td>
<td>Dhyamakam</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amomum subulatum Roxb. (Zingiberaceae)</td>
<td>Brihadela</td>
<td>![24]</td>
<td>![24]</td>
</tr>
<tr>
<td>Aquilaria agallocha Roxb. (Thymelaeaceae)</td>
<td>Agaru</td>
<td>![48]</td>
<td></td>
</tr>
<tr>
<td>Banksea speciosa J.Koenig (Costaceae)</td>
<td>Pushkarmula</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boswellia glabra Roxb. (Burseraceae)</td>
<td>Saamprani</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boswellia serrata Roxb. (Burseraceae)</td>
<td>Thurushkam</td>
<td>![26]</td>
<td>![27,28]</td>
</tr>
<tr>
<td>Callicarpa macrophylla Vahl (Lamiaceae)</td>
<td>Priyangu</td>
<td>![19]</td>
<td></td>
</tr>
<tr>
<td>Calophyllum inophyllum L. (Clusiaceae)</td>
<td>Thejovathy / Punnag</td>
<td>![30,31,32]</td>
<td></td>
</tr>
<tr>
<td>Cedrus deodora Roxb. (Pinaceae)</td>
<td>Devadaru</td>
<td>![43]</td>
<td>![44]</td>
</tr>
<tr>
<td>Cinnamomum tamala T.Nees &amp; Eberm. (Lauraceae)</td>
<td>Pathram</td>
<td>![143]</td>
<td>![145]</td>
</tr>
<tr>
<td>Cinnamomum zeylanicum Blume (Lauraceae)</td>
<td>Twak</td>
<td>![36,37]</td>
<td>![38]</td>
</tr>
<tr>
<td>Coleus vettiveroides K.C.Jacob (Lamiaceae)</td>
<td>Hreeberam</td>
<td>![19]</td>
<td>![19]</td>
</tr>
<tr>
<td>Commiphora mukul Engl. (Burseraceae)</td>
<td>Gulgulu</td>
<td>![40]</td>
<td>![40]</td>
</tr>
<tr>
<td>Commiphora myrrha Engl. (Burseraceae)</td>
<td>Rasam (Narum pasha)</td>
<td>![41]</td>
<td>![42]</td>
</tr>
<tr>
<td>Crocus sativus Ten. (Iridaceae)</td>
<td>Kumkumam</td>
<td>![43,44]</td>
<td></td>
</tr>
<tr>
<td>Elletaria cardamomum Maton (Zingiberaceae)</td>
<td>E lavakulam</td>
<td>![45,46,47]</td>
<td>![48]</td>
</tr>
<tr>
<td>Ipomoea pes-tigris L. (Convolvulaceae)</td>
<td>Vyagranskhi</td>
<td>![49]</td>
<td>![50]</td>
</tr>
<tr>
<td>Kaempferia galanga L. (Zingiberaceae)</td>
<td>Sati</td>
<td>![51,52]</td>
<td></td>
</tr>
<tr>
<td>Mesua ferrea L. (Clusiaceae)</td>
<td>Nagakesaram</td>
<td>![83]</td>
<td>![84]</td>
</tr>
<tr>
<td>Myristica fragrans Houtt. (Myristicaceae)</td>
<td>Jaathiphalam</td>
<td>![85]</td>
<td></td>
</tr>
<tr>
<td>Nardostachys jatamansi C.B.Clarke (Valerianaceae)</td>
<td>M anchi</td>
<td>![56]</td>
<td>![57]</td>
</tr>
<tr>
<td>Pirus roxburghii Sarg. (Pinaceae)</td>
<td>Sreevasakam</td>
<td>![88]</td>
<td>![90]</td>
</tr>
<tr>
<td>Polygonum alatum Buch. (Polygonaceae)</td>
<td>Sprukka</td>
<td>![60]</td>
<td></td>
</tr>
<tr>
<td>Saussurea lappa (Decne.) C.B.Clarke (Asteraceae)</td>
<td>Kushtam</td>
<td>![61]</td>
<td>![62]</td>
</tr>
<tr>
<td>Shorea robusta C.F.Gaertn. (Dipterocarpaceae)</td>
<td>Sarjarasam</td>
<td>![63,64]</td>
<td></td>
</tr>
<tr>
<td>Taxus baccata Thunb. (Taxaceae)</td>
<td>Thaleesa pathram</td>
<td>![65]</td>
<td></td>
</tr>
<tr>
<td>Valeriana wallichii DC. (Valerianaceae)</td>
<td>Thagaram</td>
<td>![66]</td>
<td>![66]</td>
</tr>
<tr>
<td>Oyster shelfii (Ostreiace)</td>
<td>Sukthi</td>
<td>![67]</td>
<td></td>
</tr>
</tbody>
</table>

The ingredients list was supplied by Nagarjuna Ayurvedic Group, Kalayanthi P.O., Thodupuzha, Kerala, India. All components of Eladi Keram were present at % concentrations of 0.44% (w/v) with the exception of Kumkumam at 0.17% (w/v) and Sukthi at trace levels.

*Anti-bacterial effect against Propionibacterium acnes [exhibited by two of the constituents; Twak and Vyagranskhi]*
3. Discussion
Eladi Keram appears to have a slow but sustained effect on changing the character of acne-troubled skin. There are no reported negative systemic, irritating or drying side effects of Eladi Keram in this study or in the published literature. Therefore, Eladi Keram may be suitable for long term use over several months. In general, inflammatory lesions are often more established than non-inflammatory comedones (white heads and black heads). Recovery from chronic inflammatory lesions takes longer and may result in physical changes to the skin, such as scarring. In contrast, comedones are characterised by occluded pores which can respond positively to treatment within shorter timescales [6]. Nevertheless, in this study Eladi Keram showed comparable improvement in non-inflammatory and inflammatory lesion counts. Thus increasing treatment duration could demonstrate a greater treatment benefit of Eladi Keram in future studies.

No scientific studies on Eladi Keram for acne have been published to date. However, it is worthwhile comparing the findings of this study to those of clinical trials which have used different topical preparations for a similar amount of time. To this end, a Cohen’s d comparison was made between the effect sizes observed from this trial and that conducted by Parveen et al. [68] who undertook a controlled randomised trial of a traditional topical Unani herbo-mineral preparation (treatment group n=20). At 30 days the improvement effect size achieved could be expressed as Cohen’s d = 1.11 (0.93, 1.29 [95% confidence interval]) where the effect size is equivalent to 1.11 standard
deviations. In the currently reported study, the effect size after 28 days could be expressed as Cohen’s d = 1.72 (1.4, 2.15) (treatment group n=10). Thus, based on Cohen’s d comparison, the currently reported treatment with Eladi Keram was 55% more beneficial than another traditional herbo-mineral formula when used for a similar duration.

In comparison to treatment with proprietary topical pharmaceutical preparations used in combination therapy, the effectiveness of Eladi Keram is weaker. Wolf et al. [69] conducted a controlled trial of a topical combination therapy (antibiotic clindamycin and topical retinoid adapalene) compared with base cream (n=125). Their study produced an improvement in inflammatory lesion count of 11.4 (p<0.005) after 28 days treatment. This compares with an improvement of 8 (p<0.05) for Eladi Keram. One could state that combination therapy with retinoid and antibiotic cream was 53% more effective in treating acne when compared to Eladi Keram. The reduced impact of Eladi Keram when compared with conventional treatment can be explained by the assumption that treatment with polyherbal formulations using whole plant extracts at low concentrations will be less pronounced in the short term and may take longer to display positive treatment effects. Further studies are required to evaluate the dose-dependent effect of Eladi Keram by altering the concentrations of the herbal constituents to determine the optimal compositions, in consultations with Ayuverdic practitioners. The currently reported study yielded no adverse reactions, whereas the pharmaceutical study reported two adverse events namely, dryness and stinging/burning [69]. In addition, given issues of antibiotic
resistance and known negative side effects of some acne treatments, a mild alternative may suit some patients. On this basis, comparison of the speed of improvement observed under the apparently milder action of Eladi Keram is not unfavourable. In this trial, treatment was restricted to 28 days with the aim of increasing compliance and reducing drop outs.

Several factors, including bacterial infection by *P. acnes* contribute to the development of acne. However, *P. acnes* is no longer believed to be the cause of acne and the role of this bacterium in the pathogenesis of acne is still unclear [12]. Fifteen of the constituent herbs of Eladi Keram have been reported to exert an antibacterial effect against different strains of bacteria (Table 1). However, there are only two reports on the inhibitory action of herbs in Eladi Keram against *P. acnes* namely *Cinnamomum zeylanicum* (in the form of an essential oil) [36] and *Ipomoea pes-tigris* (compounds in the methanolic extract) [49]. Although the anti-bacterial action of Eladi Keram against *P. acnes* specifically has not been demonstrated, other potential modes of action exist including anti-inflammatory activity. The process of acne lesion formation is initiated by various immune changes and inflammatory responses [6,8]. Twenty-two (out of 27) constituents of Eladi Keram exert anti-inflammatory effects (Table 1) via different mechanisms. For example, Tsai et al. [30] reported that *Calophyllum inophyllum* significantly inhibits the expression of proinflammatory proteins, including the transcriptional factor, nuclear factor-kappaB (NF-κB), cyclooxygenase-2 and inducible nitric oxide (enzymes involved in the production of prostaglandin E2 and nitric oxide, respectively). *C. zeylanicum* is also
reported to exert anti-inflammatory effects (both *in vivo* and *in vitro*) through the inhibition of tumour necrosis factor-α (TNF-α) [38]. Eladi Keram may therefore inhibit pro-inflammatory proteins, thus reducing inflammation, but this effect has yet to be tested. Another potential pathway is a keratolytic effect on hardened pore openings to reduce poral occlusion. Coconut oil (the base oil for the formula) is known generally as an emollient which helps to prevent the epidermis drying, making the skin better able to retain water [70]. This quality may help to prevent poral occlusion and would apply equally to both the control and treatment groups in this study. This could explain the reason some participants in the control group experienced a reduction in non-inflammatory lesion count after treatment with coconut oil, but given the significant improvement in the treatment group this mechanism clearly cannot wholly explain Eladi Keram’s mode of action. Antiseborrhoeic action (reducing excess sebum production) is a further potential pathway that may explain the efficacy of Eladi Keram. Sebaceous glands have been shown to control endocrine and immune type functions within the skin and have both direct and indirect antibacterial functions. Sebum contains an antimicrobial lipid (sapienic acid), which increases in response to skin bacterial presence [6,9]. Excessive sebum production provides an anaerobic growth setting for *P. acnes* in part due to its lipid content, and produces lipases which hydrolyse triglycerides into pro-inflammatory free fatty acids [8]. Macrophages surrounding the pilosebaceous follicles are presented with Toll-like receptors (TLRs), a subtype of pattern recognition receptors [7]. TLR-2 and TLR-4 are specific for acne pathogenesis and they can be stimulated by *P. acnes* ligation [8]. Activated TLR induces NF-
κB thus promoting the transcription of chemokines and adhesion molecules and lead to increased production of the cytokines, IL-1, IL-6, IL-8, IL-10, IL-12, and TNF-α, triggering an inflammatory response in acne patients which presents as visible inflammatory lesions [7,8,9]. The sebaceous glands also work as localised independent endocrine organs responding to hormones, as well as being involved in neurological signalling and immune type responses. This has an important role in responding to stress and maintaining normal functioning [6,9]. This suggests control of sebocytes’ function and maintenance of homeostatic functioning would support a reduction in excess inflammatory response and excess sebum production, phenomena observed in the formation of papules and pustules. The highlighted role of sebocytes indicates that Eladi Keram may therefore act through an anti-seborrhoeic route, a pathway more significant than previously considered, as it may support localised regulation of endocrine and inflammatory signalling and responses. Although we suspect that this proposed mode of action is important we cannot directly link the observed biological effect with any known constituent of Eladi Keram. This highlights the need for further investigation into the precise mode of action, including synergistic effects of herbs contained in herbal treatments such as Eladi Keram.

4. Conclusion
This study is the first reported clinical evaluation of Eladi Keram’s effectiveness in treating acne. We were able to show a clinically meaningful and statistically significant improvement of upper body Acne vulgaris under randomised and double blinded conditions. Given the relatively small sample size employed,
caution should be exercised in extrapolating the findings to a wider population or in extrapolating the findings to the treatment of the more distressing condition of facial acne. Nevertheless, this small-scale study has generated extremely promising data on the efficacy of Eladi Keram. The absence of any participant adverse reaction and the elected continued participation and improvement in some individuals beyond the initial study duration suggest that Eladi Keram may be safely tested for acne treatment. The overall findings warrant further investigation into the mode of action of Eladi Keram and importantly suggest that an extended and larger clinical trial involving facial acne treatment may yield significant findings.

**Disclosure Statement**

The Eladi Keram used in this study is a commercially available product. None of the authors has any historical or current connection with its manufacturer. The authors therefore declare no conflict of interest.

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References


