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REVIEW

Bimekizumab for Hidradenitis Suppurativa from Trials to Real Life: A Review of the Published Literature

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Abstract: Hidradenitis suppurativa (HS) is a chronic, debilitating inflammatory disorder of the hair follicles affecting areas rich in apocrine glands. Moderate-to-severe forms often require biologic therapies, including bimekizumab, a humanized monoclonal antibody that selectively inhibits IL-17A and IL-17F. Secukinumab, a fully human monoclonal antibody targeting IL-17A, and adalimumab, an anti-TNFα monoclonal antibody and the first biologic approved for HS, also represent established therapeutic options, reducing inflammatory lesions and pain with variable long-term response among patients. Approved in Europe and the USA in 2024 for HS, bimekizumab has shown promising efficacy in the BE HEARD I and II clinical trials, with significantly higher HiSCR response rates compared to placebo and improvements sustained through 48 weeks; the BE HEARD EXT extension study confirmed durable benefits at two years, with reduced disease severity and improved quality of life. This review included six studies (two trials, three case series, and one case report), highlighting consistent results in real-world settings, though limited by the drug's recent introduction. Observational studies reported significant reductions in IHS4, pain, and DLQI scores, with complete remission in some cases. The most frequent adverse events were mucocutaneous candidiasis, generally mild and manageable. Dual inhibition of IL-17A and IL-17F represents an innovative therapeutic approach for HS, with potentially greater efficacy than selective inhibitors. However, further large-scale, long-term real-world data are needed to confirm the drug's safety and effectiveness and to define the optimal role of bimekizumab in HS management.

Keywords: hidradenitis suppurativa, treatment, bimekizumab, adalimumab, secukinumab, pathogenesis

Introduction

Hidradenitis Suppurativa (HS) is a chronic, immune-mediated, inflammatory disorder of the pilosebaceous follicular unit, characterized by recurrent, deep-seated, painful nodules and abscesses that may progress to sinus tract formation and scarring.[1,2]

The disease predominantly involves intertriginous regions with a high density of apocrine glands, including the axillary, inguinal, inframammary, gluteal, and perianal areas. Lesions arise from follicular occlusion due to hyperkeratosis, followed by follicular rupture and subsequent infiltration by inflammatory cells. This cascade results in chronic suppurative inflammation and, in advanced stages, dermal fibrosis.[1,2]

HS typically manifests after puberty and exhibits a relapsing–remitting course. The pathogenesis is multifactorial, involving genetic predisposition, aberrant immune responses, mechanical stress, dysbiosis of the skin microbiome, and environmental factors such as smoking and obesity. Histologically, the disease is defined by dilated follicular infundibula, perifollicular lymphocytic infiltration, and sinus tract epithelium lined with stratified squamous keratinocytes.[3]

Among the key immune pathways implicated in HS, the IL-17 axis plays a pivotal role: lesional skin shows elevated IL-17A and IL-17F expression, driving neutrophilic recruitment, amplifying keratinocyte activation, and sustaining chronic inflammation. Dual blockade of IL-17A and IL-17F targets highly expressed in HS inflammatory circuits

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therefore directly interferes with one of the central cytokine loops promoting tissue destruction and persistent suppurative activity.

Due to its huge impact on quality of life and the extreme variability of clinical response of existing drugs, HS is still a real challenge for the dermatologist; mild-to-moderate forms are usually treated with long-term courses of antibiotics with or without short course of corticosteroids,[4,5] while moderate-to-severe forms are candidates to biological therapies. Up to now, three different biologics are approved for adult moderate-to-severe HS such as Adalimumab, an anti-TNF antibody, since 2015[6–8] secukinumab, an anti-IL17A, since 2025[9,10] and only very recently, bimekizumab, an anti-IL17 A and F, since november 2024. However, the efficacy of adalimumab, the oldest biologic drug available for HS, is highly variable in daily practice,[9,10] with possible primary or secondary lack of efficacy as well as the potential to develop paradoxical reactions (eg paradoxical psoriasis);[10] on the other hand long term efficacy data for secukinumab and bimekizumab are limited; hence the need of long term real life efficacy data is desirable and identifying new therapeutic targets for patients with HS still remain an unmet need.

To date, there are few studies in the literature with anti-IL-17 drugs bimekizumab and secukinumab for HS.[11,12] Particularly, secukinumab presents more data since it is available since 2015 for psoriasis and psoriatic arthritis. In this context, there is a huge and increasing interest in bimekizumab potential for treating HS due to the elevated unmeet clinical needs in HS as well as to observe if bimekizumab could have a somewhat higher clinical efficacy or any peculiarities compared to secukinumab as the case for psoriasis and PASI100 response.[10]

Bimekizumab is a first-in-class humanized monoclonal antibody that selectively inhibits both interleukin (IL)-17A and IL-17F. It was approved for the treatment of moderate-to-severe psoriasis by the European Commission in 2021 and by the Food and Drug Administration in 2023. In April-November 2024 it has also been approved by the EMA and FDA for the treatment of adult patients with moderate-to-severe active HS with inadequate response to conventional systemic HS therapies. The approved dose is 320 mg s.c. every 2 weeks for 16 weeks and then every 4 weeks after.[12] Currently, in our country, Italy, bimekizumab is waiting reimbursement from the Italian Medicines Agency (AIFA); hence at the moment its use in Italy is limited to selected cases with the activation of a bureaucratic procedure of compassionate use which has to be approved by the local health authority. Hence, since its only very recent approval, clinical data, especially real life, on bimekizumab use for HS is very limited. The purpose of our review was to bring together all the existing published evidence, from trials to case report, case series and ongoing studies available in literature regarding bimekizumab use for HS.

Materials and Methods

For this review, a comprehensive literature search was carried out across PubMed, EBSCO, Embase, Google Scholar, Cochrane Skin, and MEDLINE databases, covering publications up to June 2025. The search strategy consisted of combining the following terms: “hidradenitis suppurativa”, “treatment”, “bimekizumab”, “target molecules”, “IL-17”, “IL-17 A–F”, and “real-life”. Eligible publications included meta-analyses, systematic and narrative reviews, letters to the editor, real-world evidence studies, case series, and case reports. All retrieved manuscripts were carefully screened, and relevant data were extracted according to the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines[13] (Figure 1). The reference lists of included studies were also examined to capture any additional relevant articles. Only papers written in English were considered. This article relies exclusively on previously published data and does not involve any studies with human participants or animals conducted by the authors.

Eligibility Criteria and Study Selection

For this review, only randomized controlled trials (RCTs) conducted in humans, using either a placebo or an active comparator, were considered eligible. The selected studies specifically investigated the efficacy and safety profile of bimekizumab in the treatment of hidradenitis suppurativa (HS) and reported at least one of the following outcomes: *Hidradenitis Suppurativa Clinical Response (HiSCR), Dermatology Life Quality Index (DLQI)*, or adverse events. Although the main focus was on controlled clinical trials, the available real-life evidence for these agents remains scarce. Consequently, case reports and case series providing relevant clinical insights were also included and not excluded from the analysis.

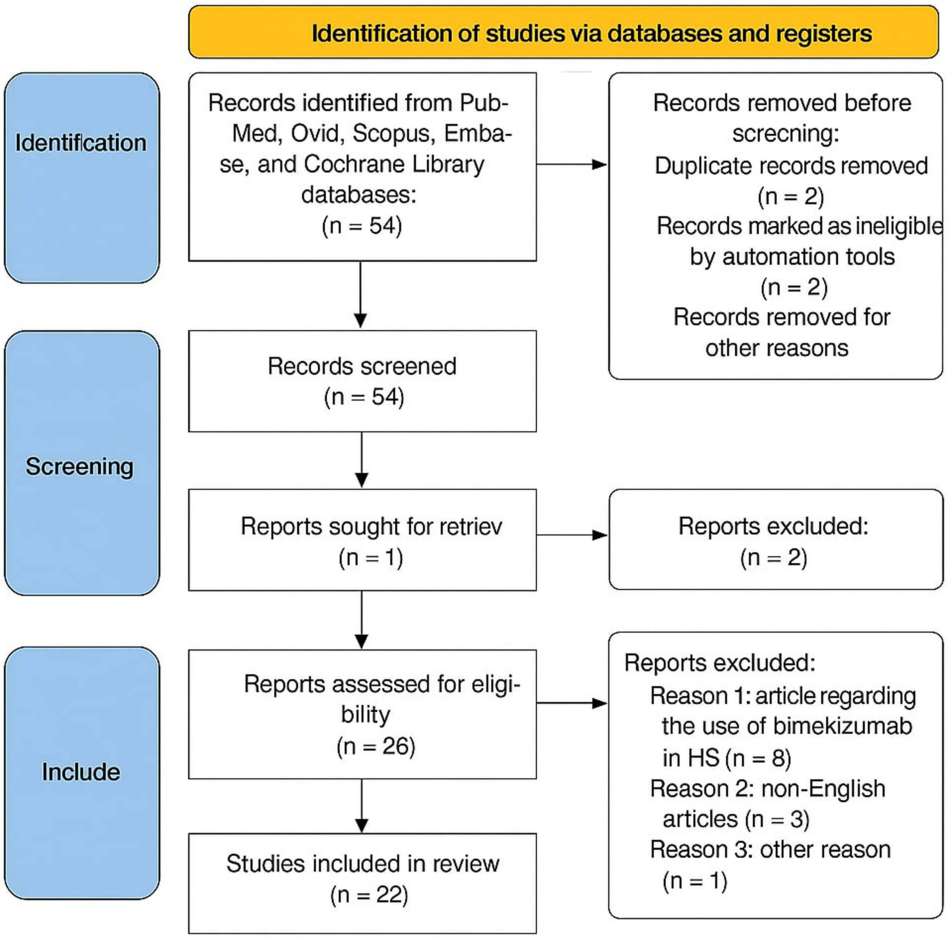

Figure 1 Prisma Check List.

Risk of Bias Assessment

The methodological quality of all included studies was independently assessed by two investigators (F.M. and M.M.) using the revised Cochrane Risk-of-Bias Tool for Randomized Controlled Trials (RoB 2) (14). This evaluation considered various methodological aspects in order to classify each study as having a low risk of bias, high risk of bias, or some concerns. In cases of divergent opinions between the two reviewers, a consensus was reached through discussion; if disagreement persisted, a third reviewer (M.M.) was consulted to provide the final judgment.

Results

A total of 6 articles (2 trials, 3 case series and 1 case report) met the selection criteria for our review.

Bimekizumab was initially investigated by Glatt et al[13] in a Phase 2 randomized, placebo-controlled proof-of-concept trial. At Week 12, among 46 participants receiving 320 mg subcutaneously every two weeks, 57.3% achieved HiSCR, compared to 26.1% in the placebo group. A reduction in IHS4 scores was also observed with bimekizumab (from 40.0 to 16.0), while the placebo group showed a smaller change (from 50.0 to 40.2).

Further evaluation of bimekizumab took place in two identically designed Phase 3 randomized controlled trials—BE HEARD I and BE HEARD II—involving a total of 1014 patients with moderate-to-severe HS. Adults aged 18 and older were randomized according to their worst Hurley stage and baseline use of systemic antibiotics. Treatment included bimekizumab 320 mg every two or four weeks up to Week 16, followed by 320 mg every four weeks through Week 48. The control group received placebo through Week 16, then switched to bimekizumab 320 mg every two weeks.[14]

In both studies, patients treated with bimekizumab every two weeks demonstrated significantly higher HiSCR response rates than those receiving placebo: 48% (138/289) vs 29% (21/72) in BE HEARD I (p = 0.0060), and 52% (151/291) vs 32% (24/74) in BE HEARD II (p = 0.0032). Additionally, in BE HEARD II, a 54% HiSCR response was observed in the group receiving bimekizumab every four weeks, compared to 32% in the placebo group (p = 0.0038). By week 12, more participants on bimekizumab also achieved HiSCR75 and HiSCR90 (46% and 32%, respectively), compared to 10% and 0% in the placebo group and 35% and 15% in the adalimumab group.[14] These improvements were sustained or further enhanced through Week 48.

Moreover, additional evidence was derived from the pooled results of the BE HEARD I & II trials and their extension study, BE HEARD EXT. Specifically, patients who completed Week 48 of BE HEARD I & II were eligible to enter BE HEARD EXT and receive open-label bimekizumab 320 mg either every two weeks or every four weeks, depending on achievement of ≥90% Hidradenitis Suppurativa Clinical Response (HiSCR90; calculated as the average of responses at Weeks 36, 40, and 44 in BE HEARD I & II). Participants originally randomized to bimekizumab from baseline in BE HEARD I & II and subsequently enrolled in BE HEARD EXT were classified as the "Bimekizumab Total group". Out of 556 such patients, 446 completed Week 96 (two years) in the open-label extension phase.[15,16] At Week 16, significantly more patients on bimekizumab every two weeks achieved HiSCR compared to placebo—47.8% vs 28.7% in BE HEARD I and 52.0% vs 32.2% in BE HEARD II.[15] The every-four-week regimen also showed benefit, with statistical significance reached in BE HEARD II. Efficacy was maintained or improved up to Week 48, with HiSCR responses increasing to ~61% and ~64% in BE HEARD I and II, respectively.[15,16]

Extended study results demonstrated sustained improvements in higher thresholds (HiSCR75/90), DLQI, pain, and flare frequency.[15,16] HiSCR90 was achieved by up to 23.2% at Week 16 and rose to 35.9% by Week 48.[15,16] Longer term outcomes are being assessed in the BE HEARD EXT trial (NCT04901195).[16] Preliminary two-year data showed >85% of initial responders maintained their improvements, with 90% sustaining HiSCR50 and 86% maintaining DLQI benefits from Week 48 to 96.[16] Disease severity also declined significantly: mild cases increased from 0% to 53.1%, while severe cases dropped from 87.4% to 20.4% over two years.[14–16] These results highlight bimekizumab durable efficacy and positive impact on quality of life in patients with HS.[14–16]

To date, there is limited real-life evidence given the recent approval of the bimekizumab for HS.[17–20]

Molinelli et al[17] reported on the use of bimekizumab in four female patients (aged 20–62 years) presenting with moderate-to-severe HS and concomitant plaque or inverse psoriasis. Treatment was administered following the psoriasis dosing schedule, consisting of subcutaneous bimekizumab 320 mg at weeks 0, 4, 8, 12, and 16, followed by a maintenance regimen of 320 mg every eight weeks. By week 16, three out of four patients achieved complete remission of HS.[17]

Mansilla Polo et al[18] conducted an ambispective observational study including consecutive adult patients with HS treated with bimekizumab from 13 hospitals in Spain during December 2022 and October 2023. Of the 40 baseline patients, complete follow-up data at week 16 were available for 34 patients (85%). All patients received bimekizumab 320mg, either every 2 or every 4weeks. At week 16, mean IHS4 significantly decreased from 27.1 ± 13.8 at baseline to 15.6±9.9 at week 16 (p<0.001), HS-PGA from 5.1±0.9 to 3.2±1.1 (p<0.001), VAS pain from 8.3±1.7 to 4.7±2.1 (p<0.001) and DLQI from 21.6±6.5 to 12.6±6.1 (p < 0.001).[18] No subanalysis comparing bimekizumab every 2 or 4 weeks was performed. Ureña-Paniegoet al[19] described 8 patients with moderate-to-severe HS treated with bimekizumab until week 16; no data about dosing regimen was available. The authors observed a substantial reduction in IHS4 by −11.625 (4.2), p < 0.05, and a significant improvement in the patient global impression of disease severity by −3.85 (1.42), p < 0.05. No serious adverse effects were recorded during the study period.[19]

Martora et al[20] reported the case of a 24-year-old female patient affected by hidradenitis suppurativa (HS) who had previously undergone multiple systemic treatments, including adalimumab, secukinumab, and guselkumab, without achieving satisfactory disease control. The therapeutic approach was subsequently modified by initiating bimekizumab, administered according to the dosing regimen approved for psoriasis. Following this change, the patient experienced a substantial clinical improvement, with the International Hidradenitis Suppurativa Severity Score System (IHS4) decreasing from 35 at baseline to 15 by week 12. However, during the course of treatment, she developed a severe form of paradoxical scalp psoriasis, which posed additional challenges to the overall management of her condition. This clinical observation underscores both the significant therapeutic potential of bimekizumab in refractory HS and the

importance of vigilant patient monitoring, as uncommon yet clinically relevant adverse events may arise and require timely recognition and intervention.[20,21]

Discussion

HS is a chronic inflammatory condition that primarily affects areas of the body rich in hair follicles.[22] The disease is initiated by the blockage of follicular ducts, which leads to the development of nodules, abscesses, and draining tunnels, often accompanied by an increase in local bacterial load.[22] HS has a profound impact on the lives of patients and their family members. Several factors, such as the presence of comorbidities, unemployment and HS severity, may contribute to making this impact even more severe.

Initial management typically includes antibiotics, anti-inflammatory agents, and corticosteroids as first-line therapies.[23,24] However, lesions recurrence and flare ups are very common after those treatments, making HS management challenging.

Recent research has shifted the focus toward the role of cytokines in the pathogenesis of HS.[25,26] Studies have shown elevated levels of IL-17A, IL-26, IFN-γ, IL-27, and IL-1β, along with reduced expression of IL-22 in the affected tissues of HS patients.[25,26]

These evidences support the use of bimekizumab, an anti-IL17 A and F drug, as a viable therapeutic option for the treatment of HS. Indeed, the identification of T helper 17 (Th17) cells has been a pivotal advancement in T-cell immunology over the past two decades, elucidating their key involvement in the pathogenesis of autoimmune and chronic inflammatory disorders, as well as their role in host defense against bacterial and fungal pathogens.[27]

IL-17A and IL-17F, two cytokines encoded by adjacentgenes on chromosome 6, signal through the same receptor complex (IL-17RA/IL-17RC) and exert similar biological effects. These include the induction of pro-inflammatory mediators such as TNF, IL-1β, IL-6, chemokines, antimicrobial peptides, and matrixmetalloproteinases. Both cytokines can form homodimers or heterodimers (IL-17A/F), with IL-17A homodimers exhibiting the greatest pro-inflammatory activity, followed by IL-17A/F heterodimers and IL-17F homodimers.

Despite being a less potent inducer of inflammation compared to IL-17A, IL-17F is abundantly expressed in HS lesions.[28–30] IL-17A– and IL-17F–producing cells are observed in early HS lesions, while in chronic lesions, IL-17F expression often co-localizes with IL-1–producing cells [12]. Additionally, regulatory T cells in HS demonstrate functional impairment and an upregulation of IL-17F expression.[29]

These observations underscore the critical role of the Th17 pathway in HS pathophysiology and provide a strong rationale for dual inhibition of IL-17A and IL-17F. This combined blockade may offer enhanced suppression of IL-17–mediated inflammation compared to selective IL-17A inhibition alone.[29–31] However, head-to-head clinical trials comparing bimekizumab with IL-17A–selective blocking agents are necessary to substantiate this therapeutic advantage. However, trials data on bimekizumab efficacy and safety for Hs patients are very encouraging.

Beyond randomized controlled trials, current real-life evidence on bimekizumab in HS remains very limited. To date, only four real-world studies and a single case report have been published, encompassing a total of 53 patients, of whom only 47 had complete follow-up to week 16 and 48. Across these reports, mean improvements in disease severity were consistent with trial outcomes: for instance, in the largest cohort (Mansilla-Polo et al, n = 34), the mean IHS4 decreased by ~43% at week 16; similar reductions (~40–45%) were seen in Ureña-Paniego et al, while Molinelli et al reported complete remission in three out of four patients. A recent case report (Martora et al) documented a 57% IHS4 reduction within 12 weeks. While these findings are encouraging, the small sample size and limited follow-up underline the need for further large-scale, long-term real-world studies before definitive conclusions can be drawn.[28–37]

Moreover, another specific point of concern in clinical practice is the potential increased risk of mucocutaneous candidiasis with IL-17 blockade. This so-called “Candida fear” has been highlighted in a recent review by Bilal et al[30] which emphasizes the importance of patient counseling and prompt recognition of fungal infections during therapy. As regards candida and bimekizumab, oral candidiasis were the most frequently reported candida infection with the majority being mild to moderate, resolved following standard anti-fungal therapy, and usually not leading to discontinuation. These findings underline the feasibility of continuing bimekizumab treatment even in the presence of Candida infections, with appropriate and effective management strategies in placed. Moreover, looking up to candida infection rates under

bimekizumab it should be highlighted that these rates are not higher in HS compared to psoriasis patients nevertheless dose regimen for HS is higher.

However, although most candidiasis cases reported in trials and real-life settings have been mild and manageable, ongoing vigilance remains warranted when initiating bimekizumab in HS patients.[38]

A recent network meta-analysis identified bimekizumab as the most efficacious biologic for treating moderate-to-severe HS, with significantly superior HiSCR response rates compared to secukinumab (300 mg every 2 or 4 weeks) and adalimumab (40 mg weekly).[30] Among all treatments evaluated, bimekizumab 320 mg administered biweekly ranked highest across all efficacy endpoints, reinforcingitspotentialroleas a preferredbiologic option in HS management.[30]

The drug is generally well tolerated; commonly reported adverse effects include gastrointestinaldisturbances, headache, and infections, whileseriousadverse events appear to be uncommon.[38] The recommended regimen for HS involves 320 mg administered subcutaneously—either as two 160 mg injections or a single 320 mg injection every two weeks through Week 16, followed by monthly dosing thereafter.[38]

Thanks to its unique mechanism of action involving dual inhibition of IL-17A and IL-17F, bimekizumab represents a novel and potentially more effective approach to HS treatment. While early findings from the BE HEARD EXT trial suggest sustained long-term benefits, comprehensive data are anticipated by the end of 2025.[39,40]

Conclusion

Unfortunately, no single, definitive therapy for hidradenitis suppurativa currently exists, largely due to the multifactorial and still not fully elucidated nature of its pathogenesis. The variable and often inconsistent efficacy of available treatments further highlights the need to identify novel therapeutic targets capable of providing more stable and predictable disease control. Such advances are crucial, as they may allow patients to achieve more durable remission, fewer flares, and reduced reliance on repeated courses of antibiotics or surgical procedures.[30,34–47]

In our review, we assembled all published data to date regarding the use of bimekizumab in HS, showing promising efficacy and a favorable safety profile both in clinical trials and early real-world reports. Compared with currently approved biologics such as adalimumab and secukinumab, the dual inhibition of IL-17A and IL-17F represents a more comprehensive targeting of key inflammatory pathways implicated in HS, which could translate into higher response rates and improved long-term outcomes for patients. (Tables 1 and 2).

However, more extensive real-world studies with longer follow-up are needed to better define the magnitude and durability of bimekizumab's therapeutic benefit, as well as its potential to reduce the need for combination regimens involving antibiotics, anti-inflammatory agents, and surgical interventions. Clarifying these aspects will be essential to understand the drug's actual impact on daily patient care and its place within the evolving therapeutic landscape of HS.

Table 1 Real Life and Clinical Trials Bimekizumab in HS

Study	Type	Sample Size	Dosing	Key Outcomes	Adverse Events
Glatt et al[13] (Phase 2)	Trial	46	320 mg q2w	HiSCR 57.3% vs 26.1% placebo; IHS4↓	Not specified
BE HEARD I & II[14]	Trial	1014	320 mg q2w or q4w	HiSCR ~48–54% vs 29–32% placebo; sustained to 48w	Mucocutaneous candidiasis common, mild
Molinelli et al[17]	Real-life	4	Psoriasis regimen	3/4 complete remission	None serious
Mansilla-Polo et al[18]	Real-life	34	320 mg q2w or q4w	IHS4 27.1→15.6; DLQI, pain improved	None serious
Ureña-Paniego et al[19]	Real-life	8	Not reported	IHS4↓ by −11.6; PGI improved	None serious
Martora et al[20]	Case report	1	Psoriasis regimen	IHS4 35→15 at 12w	Paradoxical scalp psoriasis

Table 2 Biologicals Approved for HS

Biologic	Target	Approval for HS	Dosing (HS)	Key Clinical Notes
Adalimumab	TNF-α	2015	160 mg week 0; 80 mg week 2; then 40 mg weekly	Variable efficacy; paradoxical psoriasis possible
Secukinumab	IL-17A	2025	300 mg q2w × 16 weeks, then q4w	Effective but with variable long-term response; candida risk low-moderate
Bimekizumab	IL-17A and IL-17F	2024	320 mg q2w × 16 weeks, then q4w	Highest efficacy in trials; mild mucocutaneous candidiasis common

Data Sharing Statement

Data are reported in the current study and are on request by corresponding author.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

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