of adult patients (aged 36–78 years) with unspecified ear pain, normal otologic examination, and no history of ear surgery. Other less common causes of secondary otalgia from this review included referred pain from specific cranial nerves (CN) such as trigeminal neuralgia, mandibular osteomyelitis, or parotiditis from CN V, acoustic neuroma or herpes zoster from CN VII, pharyngeal tumor or glossopharyngeal neuromas from CN IX, and laryngeal pharyngeal reflux or cricopharyngeal spasm from CN X.

A recent narrative review article noted the differential diagnosis for otalgia due to dental conditions includes acute pulpitis (commonly molar caries), acute or chronic periodontitis, acute apical abscess, acute or chronic periodontal abscesses, and pericoronitis (inflammation of impacted third molars).³

**EVIDENCE-BASED ANSWER**

Vitamin D supplementation in patients with SLE who have vitamin D insufficiency decreases disease activity (SOR: B, single RCT and case series). Vitamin D serum concentration is inversely related to SLE disease activity (SOR: B, cross-sectional study).

A double-blind RCT in 228 premenopausal women and 39 men with SLE examined if vitamin D supplementation altered the SLE disease activity index (SLEDAI), which assesses disease severity.¹ Patients (average age 38.8 years) with a mean SLE disease duration of 8.2 years were randomized to 2000 IU/d vitamin D or placebo. The mean vitamin D level at baseline for patients in treatment and placebo groups, respectively, was 19.8 and 28.7 ng/dL (10–30 ng/dL indicates insufficient level). SLEDAI scores range from 0 to 105 and scores of 5 or more are generally considered to be clinically significant and an indication for treatment.

After 12 months of vitamin D supplementation, the prevalence of vitamin D insufficiency decreased from 69% to 19%, while the SLEDAI significantly improved from a mean score of 4.9 to 3.2 (P<0.01). The prevalences of vitamin D insufficiency at baseline and after 12 months in the placebo group were 68% and 61%, respectively, and the SLEDAI score in the placebo group did not change significantly (4.8 to 4.5; P=0.69). Vitamin D levels correlated inversely with SLEDAI scores (correlation coefficient [r] =–0.583; P<0.05).¹

A case series involving female patients with SLE (N=1,006; mean age 49.6 years) evaluated whether an increase in vitamin D level had any effect on SLE disease activity over 128 weeks.² Disease activity was measured using the SELENA-SLEDAI, which is similar to SLEDAI with the same range of 0 to 105. Because recent studies recommended a serum vitamin D level of at least 30 to 40 ng/mL in adults, patients with vitamin D levels less than 40 ng/mL received vitamin D supplementation.

Seventy-six percent (n=763) of all patients in the study were found to have serum vitamin D levels less than 40 ng/mL, and were given 50,000 units of vitamin D2 weekly and 200 units of vitamin D3 with calcium twice daily.² In patients with vitamin D less than 40 ng/mL, a 20-unit increase in vitamin D level decreased the mean SELENA-SLEDAI score by 0.22 (95% CI, –0.41 to –0.02). This change corresponded with a decrease in the odds of having a SLEDAI score of 5 or more by 21% (95% CI, 1–37). There was no change in SLE disease activity by increasing vitamin D level in patients who had already had levels more than 40 ng/mL.²

A cross-sectional retrospective study in 378 patients from Europe assessed the correlation between SLE disease activity and vitamin D serum concentration.³ SLE disease activity was measured using the SLEDAI–2000 in 278 patients and the European Consensus Lupus Activity Measurement (ECLAM) scores in 100 patients (exact composite for scoring systems was not available). Serum vitamin D level was measured on the same day the disease activity was scored by the investigators. Data from the 2 scoring systems were converted into 1 standardized value to obtain univariate summary. This study showed a weak, but statistically significant negative correlation between SLE disease activity and serum
vitamin D concentration ($r = 0.12; P = .018$). Mean serum vitamin D concentrations were found to be lower in patients with active SLE disease (ie, SLEDAI score $>3$ or ECLAM score $>1$) compared with patients who had quiescent disease (mean $17.8 \pm 24.3$ ng/mL; $P < .0001$).

Kehinde Eniola, MD, MPH
Moses Cone FMR
Greensboro, NC


Is garcinia cambogia more effective for weight loss than diet and exercise?

**EVIDENCE-BASED ANSWER**

No. Garcinia cambogia plus diet and exercise does not cause a meaningful increase in weight loss over diet and exercise alone (SOR: B, meta-analysis of poor-quality RCTs and 2 RCTs).

The active ingredient in garcinia cambogia (Garcinia gummi-gutta) is hydroxycitric acid (HCA), which may prevent fat formation and suppress appetite through different mechanisms.

A systematic review examined 12 RCTs ($N = 706$ patients) of overweight/obese patients comparing oral HCA (1–2.8 g/d) to placebo over 2 to 12 weeks.⁴ All patients continued with general dietary and exercise interventions.

Nine studies ($n = 459$), similar enough to be included in a pooled data analysis, found more weight loss with HCA than placebo (mean difference [MD] $–0.88$ kg; 95% CI $–1.8$ to $0$), although by a clinically insignificant amount. In most studies, no statistically significant difference was found between the HCA and control groups for side effects of headache, skin rash, common cold, and gastrointestinal (GI) symptoms. Limitations of the studies included small sample size; poor descriptions of randomization, blinding, and allocation concealment; and differences in HCA dosage.⁴

The largest RCT from the above review, and the only one with an intention-to-treat analysis, examined the effect of garcinia cambogia extract (1,500 mg/d) on body weight and fat mass versus placebo over 12 weeks in 135 patients (ages 18–65, BMI 27–38 kg/m²).² All patients were prescribed a high-fiber, low-energy diet.

Garcinia cambogia did not produce a statistically significant weight loss compared with placebo (mean weight loss 3.2 vs 4.1 kg; $P = .14$). No significant differences were noted in percent body fat mass loss when accounting for age, sex, and pretest percentage of fat mass (mean percent body fat loss: garcinia cambogia 1.4%, placebo 2.2%; $P = .21$). Adverse events in the garcinia cambogia group (headache, upper respiratory tract symptoms, and GI symptoms) were not more common than in the placebo group. Study limitations included a small sample size and timing/dosage of HCA.

An RCT of 86 patients (ages 20–50, BMI 23–29 kg/m²) examined the effectiveness of Glycine max (soybean) leaves (2 g/d), garcinia cambogia extract (2 g/d), or starch placebo on weight loss versus placebo over 10 weeks.³ Patients maintained their regular diet throughout the study.

Garcinia cambogia did not significantly increase weight loss (0.65 kg) versus placebo (0.68 kg; $P = .05$). No adverse effects were reported. Study limitations included sex differences despite randomization and small sample size.³

An RCT of 98 patients (ages 21–55, BMI 25.2–39.6 kg/m²) examined the effect of garcinia cambogia (1 g HCA twice daily) versus placebo on weight loss over 12 weeks.⁴ All patients were instructed to follow a 1,500 calorie diet and a daily exercise regime.

Garcinia cambogia did not significantly increase weight loss versus placebo (mean weight loss [SD] $–2.0$ kg [2.6] vs 1.5 kg [3.5]; $P = .27$). However, when corrected for amount of exercise, garcinia cambogia resulted in 241 g more weight loss per hour of exercise over placebo ($P = .046$).⁴

Jessica Macdougall, MS
Anne Mounsey, MD
UNC School of Medicine
Chapel Hill, NC