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Stratification of Biochemical Response to Somatostatin Analogs in Carcinoid Syndrome: Implications for Risk of Carcinoid Heart Disease

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BACKGROUND

Carcinoid syndrome, driven by the systemic release of serotonin and other vasoactive substances, can lead to carcinoid heart disease (CHD). Somatostatin analogs (lanreotide and octreotide) are standard first-line therapies to suppress hormone secretion. However, biochemical response is variable, and persistent serotonin elevation may increase the risk of CHD. Identifying partial responders to somatostatin analogs may help guide additional therapeutic strategies.

METHODS

We retrospectively analyzed patients with carcinoid syndrome initiated on standard-dose somatostatin analog (SA) therapy (lanreotide 120 mg SQ every 4 weeks or octreotide LAR 30 mg IM every 4 weeks). Patients underwent determination of serum 5-hydroxyindoleacetic acid (5-HIAA) after 3 months of SA therapy. Biochemical response was stratified into two groups: responders (5-HIAA decreased to <123 ng/mL) and partial responders (5-HIAA decreased from baseline but remained ≥123 ng/mL). Rates of cardiac involvement were assessed using echocardiography and NT-proBNP levels. Patients were included in the TELEHEART study and were randomized to SA + Placebo versus SA + Telotristat Ethyl.

RESULTS

Among 53 patients with carcinoid syndrome treated with somatostatin analogs, 35 (66%) were classified as responders and 18 (34%) as partial responders. All 10 patients randomized to Telotristat have additionally decreased the level of serum 5HIAA, with 80% improving to “responders’ group” at 3 and 6 months follow up, while the eight patients randomized to placebo continued to have serum 5HIAA levels, with a relative linear pattern, above the 123ng/mL threshold, “partial-responders”.

CONCLUSIONS

Biochemical monitoring of serum 5-HIAA allows identification and stratification of patients that may be at increased risk of developing carcinoid heart disease. This approach enables timely consideration of adjunctive serotonin-lowering therapies for partial responders, who fail to suppress 5-HIAA below 123 ng/mL. Long-term studies can establish the survival impact of this approach.

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