1. No.

   2020G021

2. Title

   Effects of Smoking on Sarcopenic Obesity and Frailty-eating Regulation and Idiopathic Partial Lipodystrophy Syndrome

3. Period

   2020-2022

4. Summary

[Background] Sarcopenic obesity, defined by the co-existence of sarcopenia and obesity, is increasing in an aging society. The present study was designed to evaluate the effects of smoking on sarcopenic obesity, mainly focused on feeding regulation and partial lipodystrophy (PLD), of which pathophysiology seems similar to sarcopenic obesity.

[Methods] We developed a highly sensitive ELISA for leptin, a satiety hormone secreted from the adipose tissue. Using body mass indexes (BMIs) and serum leptin levels of Japanese adult males (n = 549) and females (n = 549), we evaluated the relationship between serum leptin levels and BMIs and determined the normal serum leptin levels in Japanese male and female subjects with BMI between 18.5 and 25. Using first-order correlation between serum leptin levels and BMIs, we could estimate serum leptin level from BMI in each PLD patient. Leptin secretion index (LSI) (%), which is [measured serum leptin level] / [estimated serum leptin level from BMI] was designated as a clinical index for relative leptin deficiency.

[Results] We observed a significant first-order correlation between serum leptin levels and BMIs in both Japanese adult males (r = 0.73) and females (r = 0.75).

We determined the normal serum leptin levels in Japanese adult male and female subjects with BMI between 18.5 and 25, respectively. Serum leptin levels in 14 patients with generalized lipodystrophy (GLD) were all less than the lower limit of normal serum leptin levels.

We examined LSI in 9 patients with PLD with symmetrical partial lipodystrophy in extremities before their onset of diabetes mellitus with insulin resistance. LSI in 9 patients with PLD were from 5% to 83%, which were all smaller than 100%.

There was no correlation between pathophysiology of PLD and smoking.

[Conclusions] Serum leptin levels in 14 GLD patients were all less than the lower limit of normal serum leptin levels, indicating “absolute leptin deficiency”. LSIs in patients with PLD were all smaller than 100%, suggesting “relative leptin deficiency”. LSI may be a useful clinical index for relative leptin deficiency in PLD. Smoking is not correlated with advanced arteriosclerosis in patients with PLD.