INTRODUCTION

Nowadays, Chylous pleural effusion (CPE) remains an uncommon reason of morbidity but challenging clinical problem in the world. Up to 50% of patients with CPE required surgical treatment, and the mortality is approximately 20% [1]. There are many therapeutic options in the management of CPE, and most of which share the common anatomy of thoracic duct. Conservative treatment is aimed at improving respiratory symptoms by placing a chest tube, volume replacement and nutrition [2]. Because CPE is often refractory to conservative measures, a decision has to be taken with regard to surgical intervention [3]. Among the surgical options is ligation of the thoracic duct through thoracoscopy or thoracotomy [4]. Although the use of innovations of medical therapy and novel interventional techniques in patients with CPE, many patients with CPE have a poor outcome and a prolonged hospital stay, especially patients with recent thoracic surgery and debilitated physical condition, which significantly precluded a thoracotomy approach as well as thorascosopic closure of the thoracic duct [5-6].

CPE is usually a complication of thoracic surgery procedures, accounting for approximately 50% of cases, which leading to a chyle leak into the pleural space that is characterized by an increased triglyceride concentration and the presence of chylomicrons [7]. The current evidences imply that thyroxine treatment can be used to significantly reverse CPE [8]. Thyroid hormones play an important role in maintaining normal neurological function through life. Thyroxine treatment is the cornerstone of therapy for hypothyroidism [9]. However, clinical application of thyroid hormone is hampered probably because of its positive chronoscopic effect on the heart. Recent development of thyroid hormone analogs that are able to uncouple the beneficial effects from unfavorable effects of thyroid hormone is encouraging, which may be suitable for the treatment of CPE in patients with heart disease. Because thyroid hormone receptor isoforms differentially couple to
transcription of target genes and mediate different physiological functions, this thyroid hormone selectively activate or antagonize these isoforms [10]. To date, the rapid resolution of CPE after thyroxine treatment remains unknown. We speculated that it may be due to the important effect of the thyroid hormone. This effect may be the result of controlling lipid metabolism and affecting the development of chyle by thyroid hormone [11]. Thyroid hormone plays an important role in the regulation of adrenergic receptors in the lymphatic system and lungs, thus modulating both the lymphatic flow rate and lung liquid clearance and facilitating the resolution of CPE. More importantly, this could result in a rapid flow of the lymph with engorgement of the lymphatic system, prevention of lymph into the pleura and the interstitial spaces, and the production of CPE [12-13].

THE HYPOTHESIS

Based on the clinical experiences and previous studies about thyroxine treatment, we hypothesize that thyroxine treatment might have a profound effect of resolution CPE. Examination of thyroid functions should be included in the investigation of patients with CPE, including measure the level of acid mucopolysaccharides in the pleural fluid. Because such test would have been of interest for proving the proposed association of CPE and hypothyroidism. But more proofs are needed to confirm this novel idea. Newly developed thyroid hormone mimetics are promising for the treatment of CPE and atherosclerotic cardiovascular diseases.

The hypothesis provides a possible rationale that appropriate thyroxine treatment may use as an effective therapy to enhance CPE control and resolution is intriguing. The ability to facilitate the resolution of CPE would be a huge advantage for a shorter hospital stay of patients with debilitated physical condition. There is a need for future prospective studies and long-term studies to investigate the relationships between thyroid function and CPE to answer these questions.

REFERENCES