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Parathyroid carcinoma (PCA), a rare endocrine neoplasm, was first described by Quetelet in 1904, occurring sporadically as a part of a genetic syndrome [1]. It accounts for less than 1% of parathyroid primary hyperparathyroidism in the world with a higher incidence of 5% in Japan and Italy. In affected men and women equally frequent during the fourth to sixth decade of life [2]. Parathyroid carcinoma always presents with the same clinical picture as hyperparathyroidism due to the high serum level of intact PTH (iPTH) and serum calcium level. About 10% of them are non-functional [3,4]. Patients with parathyroid carcinoma all present with the same form of hyperparathyroidism during the diagnosis including bone disease, renal failure, neurologic manifestation and gastrointestinal complaints, compared to the relatively asymptomatic presentation of benign parathyroid disease [5,6]. PHPT is observed in 80-90% of patients with the HPT-JT syndrome, parathyroid cancer is seen in 10-15%, and only 5-

oli.a benign adenoma in 80-85%, hyperplasia in 10-15%, and parathyroid carcinoma in less than 1%. In approximately 10% of cases, PHPT is part of a familial syndrome like multiple endocrine neoplasia (MEN). Type 1 or 2, hyperparathyroidism-jaw tumor syndrome (HPT-JT), familial isolated hyperparathyroidism (FIHP) or familial benign hypocalcemic hypocalcemia [9]. Other paraneoplastic syndromes associated with MEN (pituitary, pancreatic, pheochromocytoma) but not paraneoplastic syndromes. A case of hypercalcemia of malignancy associated with paraneoplastic and overall improved a biochemical parathyroid and endocrinological hypercalcemia of malignancy followed by a high degree of hypercalcemia associated with hypercalcemia of malignancy [10]. Clinical findings of parathyroid malignancy include neck tumor, hypercalcemia, hyperostosis and associated hypercalcemia, and common, but not specific [11, 12]. In addition, a typical osteolytic lesion, including a deep/indistinct (D/W) lesion in the skull, a radiolucent image and a tumor growing in the parathyroid gland could be a useful evidence indicating malignancy [13]. From a genetic focus of view, the parathyroid carcinoma can consist of a solitary, including in the isolated familial hyperparathyroidism or a part of multiple endocrine neoplasia type I (MEN-1) [14]. Other genetic changes have been also found: the methylation of men1 gene (11q13), loss of heterozygosity (LOH) in loci for chromosome 1 and overexpression of cyclin D1 [15].

11. Levin KE, Galante M, Clark OH (1987) Parathyroid carcinoma associated with parathyroid adenoma in patients with hypercalcemia. *Surg* 101(6):649-660.
12. Iihara M, Sakai R, Kameda A (2012) On the mechanism of parathyroid carcinoma and its diagnosis and treatment. *Jpn Assoc Endoc Surg Japanese Soc Parathyroid Surg* 29:201-205.
13. Hata H, Igarashi A, Yano Y, Yahiro T, Ueno, et al. (2001) Ultrastructural features of parathyroid carcinoma. *Endocrine J* 48:213-217.
14. Kahan WT, Jonsen S (2011) Focus on parathyroid carcinoma. *Int J Surg* 9(1):13-19.
15. Shaeff JM, Simonds WF (2010) Clinical and molecular genetic of parathyroid neoplasms. *Best Pract Res Clin Endocrinol Metab* 24(3):491-502.
16. Chikama TJ, Chapple CR, Noble JG, Milos EJ, Corio AG (1998) Hyperparathyroidism and neck irradiation. *Br J Surg* 75(9):873-874.
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