Multinodular Goiter (MNG)

Goiters arise from conditions with decreased thyroid hormone synthesis. Dietary iodine deficiency is a well-known source of some goiters but is not the sole cause of goiter. Multinodular goiter develops from simple goiter. The thyroid gland enlarges from continuous stimulation by TSH due to insufficient thyroid hormone production; the gland hypertrophies and undergoes hyperplasia. The varying histological characteristics of MNG arise from the cycles of hyperplasia and subsequent involution and fibrosis that occur in the gland with changing demands for thyroid hormone. Both polyclonal and monoclonal nodules can be found within the MNG, suggesting that some nodules arise from a genetic mutation that confers a proliferative advantage on the cell.

In addition to testing TSH levels, MNG may be evaluated using CT or MRI. This condition is often asymptomatic, however, compressive symptoms—dysphagia, dyspnea, compression of large vessels in the neck—may manifest and necessitate surgery. Radioiodine may also be used to shrink the goiter; multiple treatments may be necessary and recent research shows recombinant human TSH as an adjuvant may enhance the effects of radioiodine. Some patients may develop a hyperfunctioning nodule within the goiter, a condition called toxic MNG, causing hyperthyroidism. Although carcinoma is uncommon in MNG, a single dominant or enlarging nodule should be evaluated for possible cancer.

References: