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TNF-dependent regulation and activation of innate immune cells are essential for host protection against cerebral tuberculosis

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Tuberculosis of the central nervous system (CNS-TB) is the most severe form of tuberculosis which often associates with high mortality. The pro-inflammatory cytokine tumor necrosis factor (TNF) plays a critical role in the initial and long-term host immune protection against *Mycobacterium tuberculosis*, which involves the activation of innate immune cells and structure maintenance of granulomas. However, the contribution of TNF, in particular neuron-derived TNF in the control of cerebral *Mycobacterium tuberculosis* infection and its protective immune responses in the CNS were not clear. We generated neuron-specific TNF-deficient ($NsTNF^{-/-}$) mice and compared outcomes of disease against $TNF^{f/f}$ control and global $TNF^{-/-}$ mice. Mycobacterial burden in brains, lungs and spleens were compared and cerebral pathology and cellular contributions analyzed by microscopy and flow cytometry after *Mycobacterium tuberculosis* infection. Activation of innate immune cells was measured by flow cytometry and cell function assessed by cytokine and chemokine quantification using enzyme-linked immunosorbent assay (ELISA). Intracerebral *Mycobacterium tuberculosis* infection of $TNF^{-/-}$ mice rendered animals highly susceptible accompanied by uncontrolled intra- and extra-cellular bacilli replication and eventual mortality. In contrast, $NsTNF^{-/-}$ mice were resistant to infection and presented with a phenotype similar to that in $TNF^{f/f}$ control mice. Impaired immunity in $TNF^{-/-}$ mice was associated with altered genes synthesis in the brain and characterized by a reduced number of activated innate immune cells. Brain pathology reflected enhanced inflammation dominated by neutrophil influx. Our data demonstrate that neuron-derived TNF has a limited role in immune responses but overall TNF production is necessary for protective immunity against CNS-TB.

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Ultrasound-pathology correlation of thyroid tumors

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Ultrasound-guided fine needle aspiration (US-FNA) has become a first diagnostic modality and a gold standard for evaluation of thyroid nodules and thyroid has become the most popular site for FNA. Recently, cytopathologists started to show interest in performing US-FNA themselves. In addition to special training in performing FNA under the US-guidance, familiarity with US features of various thyroid lesions is essential. In addition, up to 15% of thyroid FNAs were reported indeterminate. One way to reduce the "indeterminate" rate is to use team approach and to gain the knowledge of thyroid ultrasound by correlating ultrasound with the final cytologic findings with follow-up surgical pathology including gross pathology and histologic findings of aspirated lesion. Knowing the gross and histopathologic basis of thyroid ultrasound will be tremendously helpful in cytologic interpretation and in minimizing the indeterminate and non-diagnostic rates in thyroid cytology. The presenter has been doing on-site assessment of >100,000 US-FNA of thyroid and would like to share her collection of ultrasound, Doppler, FNA cytology, gross image of resected tumors and histology ranging from scanning to high magnifications. Cases selected for this presentation are adenomatoid nodule, follicular adenoma, follicular adenoma with cystic degeneration, angio invasive follicular carcinoma, follicular variant of papillary carcinoma, encapsulated follicular variant of papillary carcinoma, Hurthle cell adenoma with post-FNA infarction, Hurthle cell carcinoma, classic papillary carcinoma with psammoma bodies, cystic papillary carcinoma, hobnail variant of papillary carcinoma, tall cell variant of papillary carcinoma and poorly differentiated thyroid carcinoma.

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