



H33 Too Much, Too Little, Too Late: Postmortem Discovery of a Postpartum Endocrinomaly

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After attending this presentation, attendees will be able to: (1) recognize some common and uncommon disease entities associated with maternal deaths; (2) identify gross and microscopic features of postpartum lymphocytic hypophysitis; and, (3) identify postmortem testing applicable to confirming this rare disease entity.

This presentation will impact the forensic science community by serving as an important reminder of a rare cause of maternal death and in turn expand the attendees' knowledge base regarding the subtlety of disease manifestation in order to ensure that the most accurate and etiologically specific cause of death is determined, which directly contributes to public health awareness through mortality data reporting.

The physiological changes of pregnancy are generally self-limiting, extending into the postpartum period. Pathophysiological changes are associated with varying degrees of morbidity and potential mortality if not recognized clinically.^{1,2} Maternal mortality trends are derived directly from cause-of-death information, such as autopsy reports and death certificates, provided by hospitals and medicolegal offices. Publication of these trends, along with disease-specific etiologies, serves as a reminder for the clinical practitioners aiding in timely recognition of signs and symptoms of disease entities, particularly those with lethal potential. Pregnancy-induced hypertension, cardiomyopathy, amniotic fluid embolism, thrombotic embolism, and infection are some of the more common death-related entities that have been reported.³ Endocrine complications associated with pregnancy are clinically recognized, one of which is panhypopituitarism stemming from pituitary necrosis caused by obstetric hemorrhage, also known as Sheehan's Syndrome.^{1,4} Infrequently, pregnancy-associated hypopituitarism can cause or contribute to death.⁵ A much less common cause of hypopituitarism in the postpartum period is lymphocytic hypophysitis, a disease entity with variable and insidious clinical manifestations and with lethal potential.⁶ A more recent report of death caused by this unusual entity is presented, along with distinguishing serological and histological features.

A 25-year-old female with a history of sickle cell trait who was three weeks postpartum following an uncomplicated delivery by cesarean section, died suddenly after a recent onset of lethargy and anorexia. At autopsy, adrenal gland atrophy, a 1.0cm thyroid gland nodule, visceral congestion, and an enlarged pituitary gland were found. Extensive necrosis, acute and chronic inflammation, lymphoid follicle formation with germinal center transformation, and fibrosis were discovered on microscopic examination of the pituitary gland. Immunohistochemical staining highlighted distinct B- and T-cell populations within the follicles, distinguishing the immunological component of this entity, while the fibrosis revealed by trichrome staining confirmed its chronicity. Negative immunoreactivity for Adrenocorticotropic Hormone (ACTH) correlated with the atrophic adrenal glands, marked hypoglycemia, and marked hypocortisolemia detected upon perimortem and postmortem serological testing. Serological evidence of thyrotoxicosis was an additional confounding finding in light of the negative immunoreactivity for Thyroid Stimulating Hormone (TSH). Moreover, the absence of immunoreactivity for TSH-secreting cells was consistent with the destructive inflammatory process of the pituitary gland and the explanation for the markedly low TSH level found on postmortem serological testing and not the result of negative feedback caused by the thyrotoxicosis. Thus, the thyrotoxicosis was due to the functioning thyroid gland adenoma. Lymphocytic infiltrates were seen in adrenal gland sections constituting additional evidence of an autoimmune-mediated disease process, but no such infiltrates were seen in sections of the thyroid gland. Vitreous fluid analysis revealed mild dehydration. The



above findings defined a clinicopathologic state of hypopituitarism with profound adrenal insufficiency caused by lymphocytic hypophysitis, complicated by thyrotoxicosis, terminating in death. Additional microscopic and laboratory examinations revealed evidence of peri-mortem ischemia and hypoxia as part of the terminal mechanism of death in this case.

Reference(s):

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Lymphocytic Hypophysitis, Maternal Death, Sudden Death