



# A Brief Note on Transient Schizophrenic Reaction as a Major Cushing's Syndrome Symptom

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## INTRODUCTION

Cushing's syndrome is one cause of secondary juvenile osteoporosis, and one study found it in a young woman with osteoporosis and fragility fractures. Because infertility is one of the symptoms of Cushing's syndrome, it is rarely diagnosed in pregnant women, and the diagnosis is often delayed due to endocrinological, obstetrical and orthopedic diseases. However, if a woman develops Cushing's syndrome during pregnancy, still birth or premature birth may occur and may require surgical treatment; therefore, early detection is critical. Women who develop osteoporosis during pregnancy are at risk for fragility fractures. Osteoporosis associated with pregnancy and breastfeeding is the most common cause of osteoporosis during pregnancy due to hormonal imbalance and low calcium levels. In such cases, vitamin D is given and breastfeeding is not recommended after delivery. In addition, parathyroid hormone preparations are used in some cases. Cushing's disease causes significant neurocognitive and psychiatric symptoms, as well as structural and functional changes in the brain.

## DESCRIPTION

In this review, we have summarized multimodal neuroimaging and neurophysiological studies to highlight the current and historical understanding of structural and functional brain changes in Cushing's disease. Specifically, structural studies revealed gray matter atrophy, loss of white matter integrity, and demyelination in various brain regions. The limbic network, the default mode network, and the executive control network are the three main functional brain connectome networks affected by hypercortisolemia, according to functional imaging studies. After endocrinological remission, gray matter atrophy and impaired functional network activity were partially reversible but widespread. It took years for the changes in white matter integrity to recover. Finally, patients with Cushing's disease have

structural and functional brain connectomic changes that shed light on the neurocognitive and psychiatric symptoms associated with the disease. Cushing's disease is a rare disease with significant consequences in terms of morbidity and mortality. It usually progresses slowly and with non-specific symptoms, which makes early detection difficult. Improvements in screening diagnostic tests have led to earlier diagnosis, contributing to a change in the traditional etiology of CD. Pituitary adenoma accounts for 80% of ACTH-dependent Cushing's syndrome, with 15% due to ectopic origin. Pituitary adenomas can be identified by nuclear magnetic resonance imaging of the hypothalamus and pituitary gland with gadolinium. This is the most sensitive and specific procedure. If MRIHH fails to localize a pituitary adenoma, inferior petrosal sinus catheterization with CRH stimulation is usually performed before recommending transsphenoidal surgery with curative intent. When the adenoma is localized by MRIHH, the remission rate after surgery is around 80% with 15% recurrence. In contrast, when the adenoma is not detected by MRIHH, the remission rate in long follow-up periods is only about 60%. In addition, previous research has found a higher risk of recurrence when the MRIHH is negative but the CIPS test is positive. Corticotroph cell hyperplasia is a rare cause of CD that has been described in several case reports in patients with confirmed pituitary origin of hypercortisolism after pituitary mass removal and histological study of corticotroph cell hyperplasia. Furthermore, it is debatable whether corticotrophic cell hyperplasia is necessary as a first step in CD or not [1-4].

## CONCLUSION

Based on previous findings, we hypothesize that the probability of finding corticotroph cell hyperplasia as a cause of CD is higher in patients with confirmed pituitary ACTH production but not detectable pituitary adenoma by NMRHH than in other

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ACTH-dependent CS cohorts. In this regard, some authors have tried to identify specific characteristics that could be used to differentiate hyperplasia from adenoma, since the diagnosis of corticotrophic hyperplasia is often overlooked. In this regard, our aims were to characterize a cohort of patients with ACTH-dependent CS who did not have a pituitary adenoma detected by MRIHH and underwent a CIPS stimulation test with CRH, as well as to investigate the pituitary pathology in these subjects after transsphenoidal surgery. Furthermore, we observed any differences in diagnostic features between hyperplasia and adenoma.

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## CONFLICT OF INTEREST

The author's declared that they have no conflict of interest.

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