ISSN: 2591-7951

# Complications associated with cushing's syndrome.

**Filipio Ceccatoiao***

Department of Pathology, Federico II' University, Naples, Italy

*Accepted on September 28, 2021*

## Description

Cushing's syndrome is a rare endocrine disease categorized by cortisol hypersecretion, prompted mostly by a pituitary tumor (Cushing's disease) or, infrequently, by an adrenal or an ectopic neuroendocine tumor. Cushing's syndrome is accompanying with severe morbidities and an increased mortality. The main systemic complications and the main cause of death are signified by cardiovascular disease. The prediction of the disease is mainly affected by the difficulties in the diagnosis and treatment of the disease, which persist a considerable challenge. Cushing's syndrome results from sustained exposure to excess glucocorticoids. Patients with Cushing's syndrome may progress multiple metabolic problems comprising obesity, hyperglycemia, hypertension, depression, low bone mass, muscle atrophy, and hypogonadism. Cutaneous manifestations of hypercortisolism comprise skin atrophy, excessive bruising, purple striations, poor wound healing, facial plethora, vellous hypertrichosis and hirsutism.

Prolonged exposure to high glucocorticoid levels in Cushing's syndrome (CS) is frequently associated with modifications in the hemostatic system and the manifestation of prothrombotic phenotypes. Distended frequency of both atherothrombotic and venous thromboembolic events (VTE) has been reported in patients with CS. In general, cardiovascular difficulties in these patients cause a five-fold increase in mortality compared to the normal population. Although numerous deformities in the hemostatic system have been identified in patients with CS, the fundamental mechanisms of the prothrombotic state are not fully elucidated. Thrombosis is typically a multicausal disease, and all three components of the so-called Virchow triad, namely 1) vascular deformities and endothelial dysfunction, 2) hypercoagulability and 3) stasis, may play a variable role in the pathogenesis of the prothrombotic state in CS patients. Individuals with prolonged hypercortisolemia due to Cushing's syndrome (CS) demonstrate cognitive dysfunction. Because glucocorticoid excess is associated with hippocampal injury in animals and the hippocampus contributes in learning and memory.

Cushing's syndrome is generally complicated by a deficiency of glucose metabolism, which is often clinically demonstrated as diabetes mellitus. The evolvement of diabetes mellitus in Cushing's syndrome is equally a direct and indirect significance of glucocorticoid excess. Indeed, glucocorticoid excess persuades a stimulation of gluconeogenesis in the liver as well as an inhibition of insulin sensitivity equally in the liver and in the skeletal muscles, which denote the most important sites responsible for glucose metabolism. In specific, glucocorticoid excess stimulates the expression of numerous key enzymes involved in the progression of gluconeogenesis, with a subsequent increase of glucose fabrication, and induces a deficiency of insulin sensitivity either directly by interfering with the insulin receptor signaling pathway or indirectly, through the stimulation of lipolysis and proteolysis and the consequential increase of fatty acids and amino acids, which contribute to the improvement of insulin resistance. Moreover, the peculiar dissemination of adipose tissue throughout the body, with the predominance of visceral adipose tissue, considerably contributes to the worsening of insulin resistance and the improvement of a metabolic syndrome, which participates in the existence and maintenance of the loss of glucose tolerance. Finally, glucocorticoid excess is able to impair insulin secretion as well as act at the level of the pancreatic beta cells, where it inhibits different steps of the insulin secretion procedure. This phenomenon is possibly liable for the passage from a loss of glucose tolerance to an overt diabetes mellitus in susceptible patients with Cushing's syndrome.

## *Correspondence to

Filipio Ceccatoiao

Department of Pathology

Federico II' University

Naples

Italy

Email: filipcecato@unipd.it

**Citation:** *Ceccatoiao F. Complications assosiated with cushing's syndrome. Arch Gen Intern Med 2021;5(8):1*