**Critical Thinking Case Study**

The condition affecting the 65-year old woman is Cushing syndrome due to the usage of prednisone that is one of the glucocorticoid drugs. Consumption of the corticosteroid drug affect the adrenal glands that are part of the endocrine system. The use of prednisone has anti-inflammatory effect used in treating various conditions such as rheumatoid arthritis and has effects on the body systems (Bronstein, 2011). The long-term taking of prednisone reduces the functions of the adrenal glands by reducing the natural production of cortisol. Changes in the hormones produced by the adrenal glands due to effects of prednisone produce the adverse effects of the drugs. The changes become evident through changes in body shape and weight of the patients. Therefore, use of prednisone caused exogenous Cushing syndrome that affects the health of the patient.

**The Pathophysiology**

The patient was on prednisone for treating rheumatoid arthritis over an extended period that is the primary cause of the symptoms. Development of Cushing syndrome among patients who take prednisone occurs due to the metabolism of the drug that leads to effects similar to cortisol. When the patient receives the medication through any route, the circulatory system transports prednisone to the liver where metabolism occurs. The liver converts prednisone to prednisolone that is the active form of the drug. Produced prednisolone can diffuse across the cell membranes and bind to glucocorticoid receptors. Consequently, the drug moves to the nucleus and affect the transcription process leading to a reduction of inflammatory proteins. The process also promotes transcription of anti-inflammatory proteins causing the therapeutic effects of the medication among patients with rheumatoid arthritis.

Prednisone also has adverse effects that result in Cushing syndrome. Prednisone is similar to cortisol produced in the human body. Production of cortisol often occurs when an individual is in stress as part of body reactions. Furthermore, the feedback system ensures regulation of the amounts of cortisol present in the body through regulating amounts of adrenocorticotropic hormone (ACTH) released (Manubolu & Nwosu, 2017). However, usage of prednisone over an extended period provides the body effects similar to cortisol without regulation. The drugs cause the adrenal glands to reduce production of cortisol while retaining a high concentration of prednisone. Atrophy of the adrenal cortex may also result due to low amounts of ACTH. Mechanisms in the body have no abilities to control the amounts of prednisone due to continuous administration of the medication. Even with the reduction in amounts of ACTH, the effects similar to those produced by cortisol persist.

The high concentration of prednisone in the body produces effects similar to hypercortisolism. Effects that result from the changes cause an alteration in body physiology identical to the uncontrolled production of cortisol. The changes become evident through the presentation of the symptoms.

**Clinical Presentation**

One of the symptoms presented by the patient is weight gain. The effects result from high levels of prednisone in the body that has similar effects to cortisol. Prednisone cause redistribution of fats in the body of the patients. In normal situations, the release of cortisol stimulates carbohydrates and fats to produce the energy needed in dealing with stressful situations. However, the body does not use the available energy sources causing redistribution and gaining of weight in the trunk. Moreover, lipolysis due to prednisone and cortisol seem not to have significant effects in the abdominal region. Therefore, the patient shows changes in her shape.

The high dosage of prednisone is also responsible for muscle wasting and weakness of the limbs. Studies show that glucocorticoids alter the metabolism of the proteins (Gupta, A. & Gupta, Y., 2013). Reduction in protein synthesis and induced protein catabolism are responsible for muscle wasting and weaknesses of the limbs. Proteolysis in the muscles results from activation of major proteolytic systems at the cellular level including the ubiquitin-proteasome system, calcium-dependent system and lysosomal system. The changes predominantly affect fast-twitch glycolytic fibers. Therefore, the presence of muscle wasting and weakness are symptoms of Cushing syndrome due to the usage of prednisone.

The patient also presented a bone fracture that resulted from the use of prednisone. Long-term intake of glucocorticoids contributes to osteoporosis among the patients. The process involves inhibition of calcium absorption in the gut and enhancement of bone resorption (Kaltsas & Makras, 2010). High concentration of glucocorticoids prevent the function of osteoblasts and increase their apoptosis (Kim, Davydov, Hans & Bockman, 2015). These developments caused the weakening of the bones that resulted in fracture while the patient tried to stand.

The presence of hypertension also results from the development of Cushing syndrome after using prednisone. Development of high blood pressure involves a complex system affecting various physiological functions. One of the mechanism comprises of activation of the mineralocorticoid receptors that increase reabsorption of renal tubular sodium (Isidori et al., 2015). Thus, the development case increase of the intravascular volumes without physiological regulations. Increase in the volume of the blood causes the patients having Cushing syndrome to suffer from high blood pressure.

Therefore, the symptoms presented by the female patient are due to Cushing syndrome. These adverse effects of the medication are due to the metabolism of the glucocorticoids.  Changes in the physiological functions of the body that are hard to eliminate while the patient uses prednisone.

**References**

Bronstein, M. D. (2011). *Cushing's Syndrome: Pathophysiology, Diagnosis and Treatment*. Estados Unidos: Humana Press.

Gupta, A., & Gupta, Y. (2013). Glucocorticoid-induced myopathy: Pathophysiology, diagnosis, and treatment*. Indian Journal of Endocrinology and Metabolism, 17*(5), 913–916. http://doi.org/10.4103/2230-8210.117215

Isidori, A. M., Graziadio, C., Paragliola, R. M., Cozzolino, A., Ambrogio, A. G., Colao, A. (2015). The hypertension of Cushing’s syndrome: controversies in the pathophysiology and focus on cardiovascular complications. *Journal of Hypertension, 33*(1), 44–60. http://doi.org/10.1097/HJH.0000000000000415

Kaltsas, G., & Makras, P. (2010). Skeletal diseases in Cushing’s syndrome: osteoporosis versus arthropathy. *Neuroendocrinology, 92*(Suppl. 1), 60-64.

Kim, S. Y., Davydov, O., Hans, D., & Bockman, R. (2015). Insights on accelerated skeletal repair in Cushing's disease. *Bone reports*, 2, 32-35.

Manubolu, S., & Nwosu, O. (2017). Exogenous Cushing's syndrome secondary to intermittent high dose oral prednisone for presumed asthma exacerbations in the setting of multiple emergency department visits. *Journal of Clinical and Translational Endocrinology: Case Reports, 6*, 4-8.

Sacerdote, A., Weiss, K., Tran, T., Noor, B. R., & McFarlane, S. I. (2005). Hypertension in patients with Cushing’s disease: pathophysiology, diagnosis, and management. *Current hypertension reports, 7*(3), 212-218.