Nursing Case Study Analysis

Introduction

The present case study deals with a multiple sclerosis patient who has recently been diagnosed with type 2 diabetes mellitus. This analysis aims to uncover possible pathological and physiological links between the two conditions. It also looks at a few nursing interventions that can be implemented to provide care and support for the patient.

Discussion

Pathophysiology of Multiple Sclerosis

Multiple sclerosis is a chronic autoimmune, neurodegenerative disorder that mainly attacks the central nervous system (CNS). This disorder is mediated by autoreactive lymphocytes that localize in the CNS and cause inflammation, demyelination, axonal loss, and gliotic scarring (Ghasemi *et al*., 2016). These autoreactive lymphocytes are formed in the body due to certain genetic and epigenetic changes in HLA genes of the Major Histocompatibility Complex (MHC). The immune system of individuals affected with multiple sclerosis produce defective B cells that lead to the formation of ectopic lymphoid follicles within the CNS. This in turn leads to antigen presentation, and localized cytokine and antibody production, triggering defense mechanisms against these follicles (Huang *et al*., 2017).

In this disorder, multiple infammatory lesions and plaques are formed throughout the brain and spinal cord of the affected individual. These plaques are largely present in the white matter, but may also be present in the gray matter. They are usually found in layers surrounding the ventricles, optic nerves, spinal cord and brainstem, and corpus callosum. Some of the important components of these plaques are activated macrophages and microglia that release non-specific products such as reactive oxygen or nitric oxide species, cytotoxic cytokines, and excitotoxins. These products attack the myelin sheaths surrounding the axons present in the CNS leading to demyelination. As myelin sheaths are largely responsible for transfer of information between axons, damage to these sheaths leads to severe neurological dysfunction in affected individuals (Huang *et al*., 2017).

Multiple sclerosis is primarily considered a T cell mediated disorder where CD4+ T cells or T helper cells interact with antigen presenting cells in the CNS to mediate immune responses. This leads to the production of cytokines such as IL-12, IL-23, and IL-4 which induces the differentiation of T helper cells into Th1 or Th17. These differentiated Th cells lead to the production of cytokines such as IFN-γ and TNF-α responsible for promoting inflammatory responses in the CNS. Apart from T helper cells, CD8+ T cells are also involved in multiple sclerosis lesions, where they lead to increased vascular permeability, destruction of glial cells, and death of oligodendrocytes (Ghasemi *et al*., 2016).

Apart from genetic and epigenetic factors, several environmental factors are also involved in the pathogenesis of multiple sclerosis. These include exposure to Epstein Barr Virus (EBV), mycoplasma pneumonia, human herpes virus type 6, smoking, UV radiation, and vitamin deficiency. Microbial agents usually have an antigen that is homologous to proteins found on the myelin sheaths. Hence, when

immune mechanisms are targeted to these antigens, they also target the myelin sheath proteins leading to their destruction (Ghasemi *et al*., 2016).

Pathophysiology of Diabetes Mellitus Type 2

Diabetes is a metabolic disorder due to insufficient action of the hormone, insulin, and is characterized by chronic hyperglycemia. It is caused due to a combination of genetic and environmental factors such as obesity, aging, stress, lack of exercise, and overeating. Genetic changes often lead to abnormalities in different stages of glucose metabolism resulting in release of excess glucose in the blood. The pathophysiological effects of type 2 diabetes, regardless of the cause, result from a combination of insufficient insulin secretion and increased insulin resistance (Kaku, 2010).

The onset of type 2 diabetes is characterized by development of insulin resistant cells in liver and muscles, and impaired function of pancreatic beta cells. Abdominal fat, unlike other forms of fat, is immune to the antilipolytic effects of insulin, and this subsequently leads to the production of large amounts of free fatty acids. These fatty acids are responsible for insulin resistance in muscles and the liver. As a result, the process of gluconeogenesis is upregulated in the liver and uptake of glucose obtained from external sources is inhibited. This leads to large amounts of circulating glucose in the bloodstream. The presence of glucose in the blood consequently leads to the production of insulin from pancreatic beta cells leading to a condition known as hyperinsulinemia. Due to insulin resistance in the liver and muscle cells, this excess insulin is not used for glucose metabolism ultimately leading to impaired pancreatic functions and decrease in the production of insulin. At this stage, hyperglycemia starts to become chronic and symptoms of type 2 diabetes start to manifest (Hackett and Jacques, 2009).

Symptoms common to Multiple Sclerosis and Diabetes Mellitus Type 2

1. **Insulin resistance**

Insulin resistance is characteristic of type 2 diabetes and it participates in the pathogenesis of the disease by causing hyperinsulinemia and hyperglycemia. It refers to a decreased capability of the hormone insulin to stimulate glucose metabolism in the tissues. In order to maintain homeostasis and maintain optimal levels of glucose in the bloodstream, the pancreas secrete excess insulin leading to hyperinsulinemia (Kostic *et al*., 2017). Recently, it has been associated with chronic inflammatory processes and oxidative stress seen in multiple sclerosis patients. Although the connection between insulin resistance and multiple sclerosis is not completely understood, it has been detected that metabolic stress might play a role in immune regulation and increase the chances of autoimmune diseases (Ruiz-Arguelles *et al*., 2018).

Several recent studies have identified the presence of insulin receptors and insulin-sensitive glucose transporters in the brain. These regions of the brain play important roles in metabolic regulation, food intake, behavior control, cognition, and memory. The glucose transporter and insulin receptors in the brain mediate the transport of insulin across the blood-brain barrier. However, there is an independent influx of glucose in the brain through other unrelated mechanisms that does not depend on insulin

transport in the brain. When this insulin transport is hindered due to inflammatory processes in the brain, it results in brain insulin resistance leading to peripheral hyperinsulinemia. Further, multiple sclerosis patients take immunomodulatory therapy for the management of their condition, which subsequently leads to glucose dysregulation, contributing to the risk of developing diabetes (Kostic *et al*., 2017).

2. Musculoskeletal effects

Musculoskeletal disease to varying extents is an important characteristic of both multiple sclerosis and type 2 diabetes mellitus. Multiple sclerosis is primarily characterized by an inflammatory attack on the central nervous system which leads to different degrees of disability in the patients. One of the most common symptoms of multiple sclerosis is spasticity along with pain, gait impairment, and abnormal ambulation patterns. It also leads to severe back pain and osteoarthritis, which significantly impairs activities of daily living (Massot *et al*., 2016). In areas of multiple sclerosis lesions in the central nervous system, inflammation and demyelination lead to failure of axonal conduction. Impaired axonal conduction affects several motor-related activities of affected individuals and lead to severe muscle weakness and fatigue (Sa, 2012).

Musculoskeletal effects are also seen in patients with type 2 diabetes mellitus, and these effects are exacerbated if the patient also has multiple sclerosis. The causes for this in case of diabetes include electrocyte imbalance, peripheral neuropathy, peripheral vascular disease, and hypoglycemia. External causes for this may include medications used to treat dyslipidemia such as HMG-coA reductase inhibitor. These medications cause rhabdomyolysis, which refers to the death of muscle fibres and their release in the blood stream. This type of muscle injury leads to severe muscle weakness and muscular defects Wyatt and Ferrance, 2006).

Type 2 diabetes mellitus patients also complain of localized pain in the upper or lower extremities. Although the pathogenesis of the disease in this regard is not very clear, preliminary studies show a connection between this type of pain and increased IgG levels, and also impaired phosphate metabolism. Another common musculoskeletal complication of diabetes is the Diabetic Stiff Hands Syndrome, which is caused by collagen degradation, diabetic microangiopathy, and diabetic neuropathy. It is characterized by thick waxy skin similar to systemic sclerosis or scleroderma. The tendon sheaths of the skin are destroyed and joint range of motion is affected (Wyatt and Ferrance, 2006).

Nursing interventions for the patient in the present case study – Mrs. Jones

- **Providing care for daily activities**

From the given case history, it is evident that the patient's primary caregiver, who is her daughter, is about to leave and her son is about to take up the bulk of her responsibilities. In the nursing assessment meeting where the son and daughter were present, the patient's son refused to take up the responsibility of hygiene and toileting. As the patient has a lot of pain, weakness, and difficulty in performing day-to-day tasks, it is important that a caregiver is provided to help her accomplish these activities with minimal pain. This caregiver should be able to help the patient shower and change her

incontinence pads. She should also be able to supervise administration of medications and provide adequate support if her symptoms worsen (Harris and Halper, 2004).

- **Providing psychological support**

The patient is worried about her daughter leaving and her son having to take care of her on a regular basis. In such a scenario, and in addition to her newly diagnosed diabetes, it is quite possible that she may have an anxiety attack or slip into depression. Also, her mental distress may have negative effects on the prognosis of her multiple sclerosis and diabetes. Hence, it is important to provide psychological support to her and her family so that the transition between caregivers takes place smoothly. It is important for her to understand that she is in safe hands and people around her are happy to provide her with the necessary care and support (Harris and Halper, 2004).

- **Refer the patient to a diabetologist**

The patient has a 13 year history of multiple sclerosis and she has started showing symptoms of diabetes since the past six months. Recently, important links have been uncovered between the two conditions and sometimes, medications given to treat one condition may worsen the other condition. Hence, the patient needs to be referred to a good diabetologist who can study her medical history and the medications that she is already taking. Following this, the doctor will be able to suggest a suitable way to control her diabetes that will not affect her ongoing treatment of multiple sclerosis. The diabetologist will also be able to suggest exercises and dietary changes keeping in mind her history of multiple sclerosis (Harris and Halper, 2004).

Conclusion

For a long time, multiple sclerosis and diabetes were thought to be independent of each other. Recent studies have uncovered important links and susceptibilities between the two conditions based on common inflammatory and metabolic pathways. Both the conditions are debilitating and lead to severe impairment of day-to-day activities. The most important preventive measure for diabetes is exercise, which is not possible for patients of multiple sclerosis. All these factors lead to severe prognosis of diabetes in multiple sclerosis patients. Hence, apart from medications, it is more important to provide psychological and emotional support to such patients so that they can lead a life of self-respect and independence.

References

Ghasemi, N., Razavi, S., & Nikzad, E. (2017). Multiple Sclerosis: Pathogenesis, Symptoms, Diagnoses and Cell-Based Therapy. *Cell Journal,19*(1).

Hackett, E., & Jacques, N. (2009). Type 2 Diabetes Pathophysiology and Clinical Features. *Clinical Pharmacist,1*, 475-478.

Harris, C., & Halper, J. (2004). *Multiple Sclerosis: Best Practices in Nursing Care*(2nd ed.). Bioscience Communications.

Huang, W., Chen, W., & Zhang, X. (2017). Multiple sclerosis: Pathology, diagnosis and treatments (Review). *Experimental and Therapeutic Medicine,13*, 3163-3166. doi:10.3892/etm.2017.4410

Kaku, K. (2010). Pathophysiology of Type 2 Diabetes and Its Treatment Policy. *Journal of the Japan Medical Association,138*(1), 28-32.

Kostic, S., Kolic, I., Raicevic, R., Stojanovic, Z., Kostic, D., & Dincic, E. (2017). Insulin resistance in drug naive patients with multiple sclerosis. *VOJNOSANITETSKI PREGLED,74*(6), 564-571.

Massot, C., & Agnani, O. (2016). Back Pain and Musculoskeletal Disorders in Multiple Sclerosis. *Journal of Spine,05*(01). doi:10.4172/2165-7939.1000285

Ruiz-Argüelles, A., Méndez-Huerta, M. A., Lozano, C. D., & Ruiz-Argüelles, G. J. (2018). Metabolomic profile of insulin resistance in patients with multiple sclerosis is associated to the severity of the disease. *Multiple Sclerosis and Related Disorders,25*, 316-321. doi:10.1016/j.msard.2018.08.014

Sá, M. J. (2012). Physiopathology of symptoms and signs in multiple sclerosis. *Arquivos De Neuro-Psiquiatria,70*(9), 733-740. doi:10.1590/s0004-282x2012000900016

Wyatt, L. H., & Ferrance, R. J. (2006). The musculoskeletal effects of diabetes mellitus. *J Can Chiropr Assoc,50*(1), 43-50.