# Diabetes: Case Study of a 63-year-old Female

The case study discussed in this essay will assess the symptoms experienced by a 63-year-old female. The symptoms include confusion, lethargy, and pale with cool and clammy skin. Based on her past medical history of Diabetes Mellitus type 1, not eating properly, presenting symptoms and a blood sugar reading of 2.9mmol/L, it is plausible to assume that she is experiencing a hypoglycemic episode. Diabetes Mellitus is a disorder that affects the way the body metabolises sugar (glucose) therefore, resulting in high blood sugar. The diabetes subtypes are, diabetes type 1 also called insulin-dependent or juvenile-onset, diabetes type 2 and gestational diabetes (Dunning, 1994). Diabetes Mellitus Type 1 is an idiopathic disease characterized by pancreatic B cell destruction and absolute deficiency of insulin causing hyperglycemia (Cotran, Kumar & Robbins, 2015).

Insulin is produced in the pancreas, which is an organ localised posterior to the stomach, and it is also responsible for aiding in the digestion of fats, carbohydrates, and proteins. The tissue cells that constitute the pancreas are endocrine and exocrine. The endocrine secretory tissue consists of a group of cells, called the Islets of Langerhans. These endocrine cells are grouped around the blood vessel where the hormone is secreted. There are three different groups of secretory endocrine cells with distinct functions, alpha cells, beta cells and delta cells (Shier, Butler & Lewis, 2004) The beta cells produce a hormone called insulin. This hormone is released when the parasympathetic innervation from the vagus stimulate the beta cell to excrete insulin when the levels of sugar in the bloodstream is high (Williams & Pickup, 1999). The insulin opens a channel in the cell's membrane for the glucose to be absorbed, particularly muscle, adipose tissue and liver cells ( Braun & Anderson, 2007).

However, if the beta cell is abnormal or nonfunctional, there will be no insulin release, and the rest of the cells and tissue will not be able to absorb glucose. For instance, in diabetes type 1, the autoimmune process that happens in the Islet of Langerhans (pancreas cells) destroys the beta-cell as a result of the chronic inflammatory process. The immune system, which defends the healthy cell against infections and viruses, mistakenly destroys Beta cells( REF). Cavallaro Goodman & Fuller ( 2015) suggests that the T lymphocytes, macrophages, dendritic cells, B lymphocytes, and Beta cell autoantigens are the cells involved in the autoimmune process or destruction on the Beta-cell. As a result of this cell destruction, the body’s (pancreas) ability to produce insulin is impaired (Georga, Fotiadis & Tigas, 2018).

The hormone insulin allows digested food be used as a source energy. In digestion, food passes through a mechanical and chemical process until it turns into molecules small enough for diffusion and cell absorption. From the small intestine, these molecules are transported by the bloodstream to the cells and tissues and then used as a source of energy (Shier, Butler & Lewis, 2004). If insulin is absent, it does not allow molecules of glucose from the digested food to be absorbed. Therefore, depriving cells of a source of energy would cause a hypoglycemic state.

The plasma glucose level must be kept in a certain range to be able to provide energy.

The National Institute for Health and Care Excellence ( 2019) suggests, in the guideline for

diabetes type 1, a blood glucose level in a range of 5 -7 mmol/L on walking, 4-7 mmol/L before meals or 5-9 mmol/L for a reading 90 minutes after eating. Hypoglycemia occurs when the level of plasma glucose in the body system is below the average or the range suggested for the patient (Kanaka-Gantenbein, Tentolouris, Katsilambros & Makrilakis, 2013). An adult who has a blood glucose level under 4.0 mmol/L is considered to be experiencing a mild to moderate hypoglycaemic episode (Stanisstreet, Walden, Jones & Graveling, 2010). To differentiate between a mild and a moderate hypoglycemic episode, the Glasgow Common Scale (GCS) is used to assess the neurogenic symptoms. A mild patient is conscious and oriented and the moderate patient is more likely to be uncooperative, impaired conscious with low chances of oral treatment due to the risk of choking (Brown, Kumar, Millins & Mark, 2016).

The physical symptoms and signs presenting by this patient is the body responding to the lack of glucose. The sign and symptoms manifest in two different ways. The neurogenic signs and symptoms are triggered during hypoglycemic episode causing physiological changes and activating the autonomic nervous system (Tesfaye & Seaquist, 2010). Frier & Fisher (1999) suggest that the neurogenic symptoms are the mechanism of awareness of hypoglycemia, as it causes physiological changes the patient became aware of its present condition. Some of the physiological changes are palpitation, clammy skin, pale and diaphoresis. For instance, her clammy skin is the result of sweating. The sympathetic cholinergic nerve mediates to maintain homeostasis, the onset of sweat is caused by the increased activity of the sudomotor nerve triggered by a decrease in glucose (Kosaka, 2001).

The neuroglycopenic signs and symptoms result from the brain glucose deprivation in a hypoglycemic episode. As a result of this deprivation, some symptoms are weakness, confusion, lethargy, and hypothermia (Frier & Fisher, 1999). Another symptom presented by this patient is cool skin, the environment has an influence on this factor. If the climate is hot or warm, the sweat mechanism induces the body to lose heat by increasing the heat conduction from vasodilation (Frier & Fisher, 1999).

Like any other patient, scene assessment is required from the beginning to the end. At this stage, several factors can be identified. This includes verifying the patient's safety environment for future hypoglycemic episodes, if there is food in the house, if there is alcohol (as it can affect glucose levels) and if there is a sign that the patient has been in hospital recently (e.g. letter or bracelet) and any other factors depending on the circumstances. As soon as the patient is encountered, airway, breathing, circulation, and disability need to be assessed and corrected if there is a need. The blood glucose is part of the disability assessment, and it needs to be corrected before any further baseline observations are taken, as it can rapidly deteriorate (Pilbery & Lethbridge, 2016).

The patient is found with neuroglycopenic and neurogenic symptoms, and with a blood glucose of 2.9mmol/L. At this stage, the management depends upon the cooperation and condition of the patient. After the first blood sugar reading, if the patient is cooperative and able to swallow, the National Institute for Health and Care Excellence (2019) suggests 10g to 20g of oral glucose in the form of liquid, granulate or lamps. Further, the same procedure is repeated after 10 to 15 minutes. It is advisable to offer some simple carbohydrate or sugar snacks to prevent a blood glucose fall. Some of the food suggested is 100 ml of coca-cola, 18 ml Ribena blackcurrant or 2 teaspoons of sugar. The drinks should not be sugar-free or diet or zero sugar (Pilbery & Lethbridge, 2016).

Another option would be an initial treatment with 15 grams to 20 grams of simple carbohydrate such as chocolate, sugar drink or glucose gel 10 grams ( a glucose tube contains 25 grams glucose with a 40% oral gel) with an interval of five minutes between doses. The blood glucose reading must be checked after 10 min. If the patient refuses to ingest a carbohydrate food or a sugary drink, but is still able to swallow, one to two tubes of oral Dextrose gel 40% can be administered(Brown, Kumar, Millins & Mark, 2016) .

Simple carbohydrates are given to help boost glucose, as they contain single monosaccharide units which are easy and fast to digest. On the other hand, a complex carbohydrates such as disaccharide and polysaccharide contain hundreds of units bonded together. Consequently they take longer to break down and digest (Patton & Thibodeau, 2016).

In case the patient does not want to ingest any glucose, another choice would be intramuscular glucagon (Pilbery & Lethbridge, 2016). By subcutaneous or intramuscular injection, the administrative dosage is the 1mg for adult (Excellence, 2019). The glucagon will induce the liver, activating enzymes in the hepatic cells to convert glycogen ( through glycogenolysis) raising hepatic glucose (Gard, 1998). However, if the hepatic stores of glycogen are too depleted the glucagon will have no effect (Mogensen, 2007).In this case, the patient history present a poor food balance, followed by a (past) physical unwell condition and regular insulin intake. In this presentation is likely to assume that her liver may be depleted of glycogen. Following this assumption, Mogensen (2007) suggests the use of 10% glucose intravenous infusion if it is available, instead of intramuscular glucagon, as it seems to be more efficient and faster.

Consideration before administering the drug is essential such as all the drugs check, allergies and contraindication (Pilbery & Lethbridge, 2016). The intramuscular injection can be given only once, after ten minutes the blood glucose level has to be checked again. It is expected a blood glucose level to raise at least 5 mmol/L, and a change on the level of consciousness (less confuse and more aware of the situation) (Brown, Kumar, Millins & Mark, 2016).

All treatment can be repeated up to three times, except the intramuscular glucagon. If there is no increase in blood glucose, an intravenous infusion of glucose 10% can be considered as another option. This drug has to be administered by a large gauge cannula into a large vein, as it could cause veins irritation due to the 10% concentration. The initial dose is the 10 grams in a 100 ml volume, which can be given in total up to 30 grams (300ml volume) with 5 minutes interval between doses. Furthermore, fluid can be given to avoid patient dehydration. (Brown, Kumar, Millins & Mark, 2016).

If the patient responds well to the previous treatment, and blood glucose is recovered at 5mmo/l and they are able to eat and drink, it may be appropriate to leave the patient at home. However, if the circumstances allow, it could be suggested for the patient to call someone for a few hours only for assurance. On the other hand, if the patient does not recover, they will need to be taken to the hospital and management is continuous en-route. Furthermore, the patient should be taken to the hospital if they present with any disorder or complication (Brown, Kumar, Millins & Mark, 2016).

During a hypoglycemia episode, the fall in blood glucose activates a counterregulatory response to rebalance the levels of glucose in the blood. This response releases hormones such as glucagon, epinephrine, norepinephrine, cortisol and growth hormone. The trigger is caused by

brain glucose deprivation. The brain needs around 120 g - 140 g of glucose daily, any fall in this supply, releases these hormones to counter-regulate to avoid hypoglycemia. (Dunning, 1994). Consequently causing a series of neurohumoral and behavioral responses (McCrimmon & Sherwin, 2010).

However, over time, patients with Diabetes Type 1, will develop difficulties or become less able to detect the symptoms and signs of a hypoglycemic episode. The extent in which the patient perceives neurogenic signs begins at plasma glucose 3.3 mmol/L and neuroglycopenic sign start around 3 mmol/L. The first three defenses against hypoglycemia are decrease insulin, increase glucagon and increase epinephrine. Due to an uncertain fault in the counter-regulatory mechanism, this defense, are attenuated or nonexistent (Cryer, 2010).

It is important, after the patient is stable, to assess their knowledge around their condition. There are many risk factors that need to be addressed to avoid hypoglycemia. One of them is inconsistent or poor diet. It was reported that the patient has been unwell and has not been eating consistently as they should. There are some different factors that could influence a patient’s glucose level. Some of them are food-management such as the time that the food was consumed, type of carbohydrate, intercurrent illness such as urinary tract infection and alcohol intake. Furthermore, poor techniques when monitoring, such as not washing their hand properly before getting the blood sample or not checking the blood glucose meter before using can affect the reading (Dunning, 1994). It is also believed that over the time, patients with diabetes are more likely to have depression due to

Regardless what cause the hypoglycemic episode, London Ambulance Service NHS((LAS), 2019) suggests the hypoglycemic pathway referral. This guide advice the pre-hospital care team which step is more appropriate for a patient who had a hypoglycemic episode. Further the patient will be assessed by a diabetic specialist who will guide and assess their knowledge and independence along the way . For instance, a questionnaire such as Clark and Gold score assessment for hypoglycemia unawareness, allow the health professional to know how aware the patient is before a hypoglycemic episode happens (College Hospital, 2019). Not be able to recognize hypoglycemia signs and symptoms put the patient in great risk.

It is also very important to remind about the communication with the patient. They need to be aware need and informed of all decisions and procedures. However, if the patient refuse for instance a referral, Caldicott Commission(2013) allows health care professional to act in the best interest of the patient. Therefore, making a referral against their wish. Finally, to assess and manage this patient's condition, include not only the physical health. There are factor that also need to be considered in order to maintain this patient out of risk. Furthermore, making the family and friends aware of the condition and emergency procedures will support the patient in case anything happens.

## Reference


1. Braun, C., & Anderson, C. (2007). Pathophysiology. Philadelphia, PA: Lippincott Williams & Wilkins.
2. Brown, S., Kumar, D., Millins, M., & Mark, J. (2016). UK ambulance services clinical practice guidelines 2016. Bridgwater: Class Professional Publishing.
3. Cavallaro Goodman, C., & Fuller, K. (2015) Pathology (4th ed.). St Louis, Missouri:

Elsevier.
4. College Hospital, K. (2019). https://www.nice.org.uk/guidance/dg21/resources/modified-clark-and-gold-score-real-world-example-kings-college-hospital-pdf-2306448398College Hospital, K. (2019).
5. Commission, L. (2013). Data sharing between public bodies. London: Stationery Office.
6. Cotran, R., Kumar, V., & Robbins, S. (2015). Pathologic basis of disease (9th ed.). Philadelphia, PA: Saunders Elsevier.
7. Cryer, P. (1997). Hypoglycemia. New York: Oxford University Press.
8. Cryer, P. (2010). Hypoglycemia in Type 1 Diabetes Mellitus. Endocrinology And Metabolism Clinics Of North America, 39(3), 641-654. doi: 10.1016/j.ecl.2010.05.003
9. Cryer, P. (2016). Hypoglycemia in diabetes (3rd ed.). Virginia: Cenveo Publisher Services.
10. Dunning, T. (1994). Care of people with diabetes. Oxford: Blackwell Scientific Publications.
11. Dunning, T. (1994). Care of people with diabetes. Oxford: Blackwell Scientific Publications.
12. Excellence, N. (2019). GLUCAGON | Drug | BNF content published by NICE. Retrieved from https://bnf.nice.org.uk/drug/glucagon.html#contraIndications
13. Excellence, N. (2019). Hypoglycaemia | Treatment summary | BNF content published by NICE. Retrieved from https://bnf.nice.org.uk/treatment-summary/hypoglycaemia.html
14. Frier, B., & Fisher, B. (1999). Hypoglycaemia in clinical diabetes. Chichester, West Sussex, England: John Wiley.
15. Gard, P. (1998). Human endocrinology. London: Taylor & Francis.
16. Georga, E., Fotiadis, D., & Tigas, S. (2018). Personalized predictive modeling in type 1 diabetes. London, United Kingdon: Elsevier Ltd.
17. Goodman, C. C., & Fuller, K. S. (2015). Pathology: Implications for the physical therapist(Fourth ed.). St Louis, Missouri: Elsevier.( university citation)
18. GREEN, B. (2008). Head Start to AS Biology. Newcastle upon Tyne: Coordination Group Publications Ltd.
19. Holt, R., Cockram, C., Flyvbjerg, A., & Goldstein, B. (2016). Textbook of diabetes(5th ed.). Malden, MA: Wiley-Blackwell.: John Wiley & Son Ltd.
20. Kanaka-Gantenbein, C., Tentolouris, N., Katsilambros, N., & Makrilakis, K. (2013). Diabetic emergencies. Hoboken, N.J.: Wiley.
21. Kosaka, M. (2001). Thermotherapy for neoplasia, inflammation, and pain. Tokyo: Springer.
22. McCrimmon, R., & Sherwin, R. (2010). Hypoglycemia in Type 1 Diabetes. Diabetes, 59(10), 2333-2339. doi: 10.2337/db10-0103
23. Mogensen, C. (2007). Pharmacotherapy of Diabetes. [New York]: Springer Science+Business Media, LLC.
24. National Institute for Health and Care Excellence. (2019). Managing type 1 diabetes in adults. UK: NICE.
25. Patton, K., & Thibodeau, G. (2016). Anatomy & physiology (9th ed.). St. Louis: Elsevier Mosby.
26. Peate, I., & Nair, M. (2016). Fundamentals of anatomy and physiology: For nursing and healthcare students.
27. Pilbery, R., & Lethbridge, K. (2016). Ambulance care. Bridgwater: Class Professional Publishing.
28. Sen, S., Chakraborty, R., & De, B. (2016). Diabetes Mellitus in 21st Century. Singapore: Springer Singapore, Imprint: Springer.

29. Shier, D., Butler, J., & Lewis, R. (2004). Hole's essentials of human anatomy & physiology (10th ed.). Boston: McGraw- Hill.
30. Stanisstreet, D., Walden, E., Jones, C., & Graveling, A. (2010). The Hospital Management of Hypoglycaemia in Adults with Diabetes Mellitus. Retrieved from http://www.diabetologists-abcd.org.uk/JBDS/JBDS_IP_Hypo_Adults.pdf
31. Tesfaye, N., & Seaquist, E. (2010). Neuroendocrine responses to hypoglycemia. Annals Of The New York Academy Of Sciences, 1212(1), 12-28. doi: 10.1111/j.1749-6632.2010.05820.x
32. The London Ambulance Service NHS Trust (LAS). (2019). Implementing the Integrated Hypoglycemic Pathway. LONDON: Health Innovation Network South London.
33. Verberne, A., Korim, W., Sabetghadam, A., & Llewellyn-Smith, I. (2016). Adrenaline: insights into its metabolic roles in hypoglycemia and diabetes. Journal Of Pharmacology, 173(9), 1425-1437. doi: 10.1111/bph.13458
34. Williams, G., & Pickup, J. (1999). Handbook of diabetes (2nd ed.). Oxford: Blackwell Science.
35. Young-Hyman, D., & Peyrot, M. (2013). Psychosocial care for people with diabetes. Alexandria, Va: American Diabetes Assoc.