Case study: adolescent type 1 diabetes



Mary is 19 old female as she just come for higher studies in university. She was diagnosed diabetes type 1 at the age of 11. Her objective data shows the symptoms of uncontrolled diabetes. Diabetes have many complication include diabetic ketoacidosis, hyperglycemia, hyperosmotic non-ketotic state, and hypoglycaemia. In Australia 3. 2% of female and 4. 0 % males reported having diabetes mellitus (Australian Bauru of statistics). Diabetes type 1 is more commonly diagnosed in juveniles formerly it was known as ' juvenile onset' (Robbins, 2008, p. 1348).

Pathophysiology Type 1 diabetes is result from sever or absolute deficiency of insulin due to destruction of beta cells. loss of islet cell is related to genetic susceptibility, autoimmunity and environmental factors. Autoimmune mechanism are related to cell and cytokine-mediated injury of beta cells. Islet cell are damaged by autoantibodies and antibodies to insulin and glutamic acid decarboxylase participate in destruction of islet cells. Auto antibodies are detectable before symptoms appears (Huether, 2004, p. 489). Some environmental factors which triggers the autoimmune injury are viruses , drugs and nutritional intake. Mumps, rubella and cytomegalovirus are the most commonly attacking on the beta cells. High intake in of bovine milk and high level of nitrosamines in food. Alloxian , streptozocin , and pantamide are the drugs which can cause the type1 diabetes. Non immune type 1 diabetes may occur due to pancreatitis (Huether, 2004, p. 490).

Hyperglycaemia occurs if insulin secreting beta cells of islet of Langerhans destroyed and pancreas no more producing insulin. Ones the production of insulin decrease the onset of symptoms is rapid (Robbins, 2008, p. 1350). story of sudden weight loss, and classic symptoms of

Page 3

Patient present the history of sudden weight loss, and classic symptoms of diabetes polydipsia, polyuria and polyphagia.

Polydipsia is excessive thrust due to high level of blood sugar, body cells attract water osmotically result in intracellular dehydration and hypothalamus stimulate thrist. Polyuria increased blood glucose act as osmotic diuretic, glucose filtration in glomeruli is exceeds renal tubules can reabsorb the glucose. Which results glucosuria , water and electrolytes are lost in large quantity in urine. In polyphagia cellular capacity to store carbohydrate, protein and fat is decrease , due to decrease capacity to store energy cells start starvation and increase hunger. Weight loss and fatigue due to loss of fluid and tissues , fat and proteins are utilise as a energy source (Huether, 2004, p. 490).

Mary was found confuse by her flat mate . Reason of confusion might be the cellular dehydration. Serum osmolality and mental status shows strong correlation in diabetic ketoacidosis and hyperglycaemic hyperosmolar state (Kisiel, 2009, p. 1097). Mary's skin is dry and flushed because her organism loses fluid. She has tachycardia due to dehydration and electrolyte imbalance. Her respiration patter is kussmaul breathing because she have metabolic acidosis her pH is 7. 25. Low pH stimulate the respiratory center which increase the depth and rate of respiration. Severity of metabolic acidosis can be determined by level of blood pH . Accepting criterion for sever diabetic ketoacidosis is pH less than 7. 30.(Moore, 2004, p. 47). Her breath is smelling like aceton because the rate of ketone bodies formation is higher than the utilization acetoacetic acid by cells (Mitra. A, 2004, p.

Page 4

1202robinson). Body trying to get rid of excess acetone through skin and lungs.

Diabetes has long term and acute complications. Long term complications are affecting the micro vascular circulation and micro vascular system. When the micro vascular circulation is compromise and small blood vessel are damage in kidney, nerves, and retina leads to end stage renal disease foot ulcers, blindness respectively (Hill, 2009, p. 51)

Most common acute complications of diabetes are diabetic ketoacidosis and hyperglycaemic hyperosmolar state. Nurse must differentiate between these two conditions. They both have many common signs but in diabetic ketoacidosis patient presents deep rapid respirations (Kussmaul breathing), serum ketones and urine kentones presents, serum sodium level increased or decrease, serum potassium level initially increased. Patient can be alert or drowsy, coma in sever case. Pain abdominal due to metabolic acidosis (Kisiel, 2009, p. 1096). On the other hand patient with hyperglycaemic hyperosmolar state shows different symptoms and different values of urine and blood tests. Serum ketones and urine ketones are absent or mild. Respiration rapid and shallow. Serum sodium increased and serum potassium normal or low. Neurological problems like transient haemiparesis or seizures, coma or stupor more likely due to hyperosmolar state (Kisiel, 2009, p. 1096).

Diabetic ketoacidosis is precipitated by infections such as gastro-enteritis, urinary tract infection, or upper respiratory tract infections. Other causes are new onset type 1 diabetes, trauma, steroid treatment, insulin pump failure and deliberate omission of insulin treatment (Dromgoole, 2004, p. 41). Mary had the flu like symptom and her insulin found not working by paramedics. All these symptoms confirm the Mary's diagnosis of diabetic ketoacidosis.

Mary is diabetic her subjective and objective data is important for nursing assessment and her further treatment. In subjective data nurse should collect information about past health history, viral infections like mumps, rubella, coxsackievirus or any other infection. Infection is the most common precipitating factor in diabetic ketoacidosis (Jean-Louis, 2003, p. 861). Family history of diabetes type 1 or 2. History of trauma, pregnancy or recent surgery. Use of medications and regime. Functional health patterns nutritional metabolic: loss of weight, thirst, hunger, nausea vomiting, healing of wound specially on foot whether it is slow or not and compliance with diet. Elimination: constipation or diarrhoea: nocturia, frequent urination, urinary incontinence (Semb susan lewis, 2004p. 1285).

Gather the information about the client activity and exercise is important to find out muscles weakness and fatigue. Cognitive-perceptual: blurred vision, Headache, abdominal pain, tingling and numbness of extremities. Copingstress tolerance: irritability, apathy, and depression. Value and belief: change in life style, diet, medication and activity patterns (Semb susan lewis, 2004p. 1285).

Objective data is important part of history taking. Respiratory assessment she has kussmaul respiration . Neurological she was found confuse. Some shows the strokelike symptoms tachycardia , hypotension, electrocardiogram changes are commonly noticed in diabetic ketoacidosis patients, she has

Case study: adolescent type 1 diabetes – Paper Example

tachycardia. Metabolic or gastrointestinal assessment polyuria and acetone breath. Integumentary assessment dry membrane, flushed skin and poor skin turgor. Her skin is dry and flushed. Vital signs of these patients shows low blood pressure , heart rate more than 100 bpm. History and risk factors for development of crisis are insulin resistance, pancreatitis, infection, and poor control of diabetes (Swearingen, 2004. p. 409).

Mary is presenting kussmauls respiration, tachycardia, high level of blood glucose 42. 5 mmol/L and decreased pH 7. 25. First priority is to maintain her airway patent. Start oxygen via mask or nasal cannula. Intravenous access preferably two sites because patient of DKA often required hydration and medication, Same time take blood for required tests include serum ketones, cardiac enzymes, amylase, lipase and Arterial blood gas. Check capillary blood glucose level (Shaffer, 2007, p. 237). As Mary's level of consciousness very glassgow coma scale should be used to monitor her consciousness. Obtain a 12 lead electrocardiogram to assess any change in cardiac function, or other signs of hyporkalemia and cardiac comparison. Pulse and oximetry should use continuously. Vital signs must monitor half hourly for first hour then hourly for next 4 hours and then 4 hourly until patient condition is stabilize (Jean-Louis, 2003, p. 865). To a assess the signs of renal compromise place indwelling catheter if patient is not able use bedpan. It allow to monitor urinary out and assessment of the adequate fluid resuscitation (Shaffer, 2007, p. 237).

Nursing diagnosis for diabetic ketoacidosis are deficient of fluid volume, risk of infection, ineffective tissue perfusion, and deficient of knowledge. Interventions and rationales related to fluid volume deficient are monitor https://assignbuster.com/case-study-adolescent-type-1-diabetes/

Page 6

Case study: adolescent type 1 diabetes – Paper Example

vital signs because hyperglycemia act as an osmotic diuretic which cause severe electrolyte and fluid loss which can lead to hypovolemic shock. Monitor patient for dry mucosa, poor skin turgor, sunken and soft eyeballs these are the indicators of hypovolemia. Administer IV fluid as prescribed and be alert for fluid overload. Dyspnea, jugular vein distension and crackles are indicators of fluid overload. Administer insulin as prescribed . Use separate IV tubing and use infusion device for maximum accuracy (Swearingen, 2004. p. 410).

Risk of infection is related to inadequate secondary defence. Infection is the most common cause of diabetic ketoacidosis(Jean-Louis, 2003, p. 861). Monitor patient for infection symptoms like fever , chill, pain with urination, and swelling around the IV site (Swearingen, 2004. p. 410). Observe the circulation of extremities, check the colour, warmth, edema, and pulse for early detection of infection. Limit use of indwelling urethral catheter for patient who is unable to void in bed pan. Indwelling catheter increase the risk of infection. Encourage patient to take adequate nutrition and fluaid intake to prevent illness (Semb susan lewis, 2004, p. 1286).

Ineffective tissue perfusion in peripheral of extremities due to interrupted venous or arterial flow secondary to increase blood platelets aggregation and adhesiveness, increase blood viscosity, and decrease patient immobility (Swearingen, 2004. p. 410). Monitor Hct and BUN result Hct return to normal with with in 24-48 after proper fluid replacement. BUN value is indicator of improve renal function and tissue perfusion. Patient suspectible to DVT encourage clients to do the exercise of extremities, calf pumping and ankle circles. Apply pressure stocking or pneumatic pumps to promote venous return (Swearingen, 2004. p. 414).

Deficient of knowledge is the major cause of the uncontrolled diabetes and the complication related to the diabetes. It is important that patient have good understanding of diabetes, so that they can effectively manage their condition (Palmer, 2004, p. 44). Nurse can educate to the patient regarding the diabetes process and common early symptoms of worsening hyperglycaemia explain untreated these conditions can leads to coma or death (Hall, 2008, p. 35). Teach patient about prescribe insulin regimen. Explain that 1-4 time insulin as prescribed everyday to keep the blood glucose level in control (Swearingen, 2004. p. 414). Teach patient to maintain adequate hydration particularly if diarrhea , vomiting , or fever persist (Swearingen, 2004. p. 416).

ABS http://www.abs.gov.au/ausstats/abs@.nsf/mf/4820.0.55.001#In %202004%2C%20the%20age-standardised%20ann

Culliton, G.. (2009, June). An all-in-one approach is necessary in the treatment of diabetes. Irish Medical Times, 43(23), 28. Retrieved April 29, 2010, from ABI/INFORM Trade & Industry

Cypress, M. (2001). Acute complications. RN, 64(4), 26-32. Retrieved from CINAHL Plus with Full Text database.

Dashiff, C., Hardeman, T., & McLain, R. (2008). Parent-adolescent communication and diabetes: An integrative review. Journal of Advanced Nursing, 62(2), 140-162.

Page 9

De Beer, K., Michael, S., Thacker, M., Wynne, E., Pattni, C., Gomm, M., et al. (2008). Diabetic ketoacidosis and hyperglycaemic hyperosmolar syndrome clinical guidelines. Nursing in Critical Care, 13(1), 5-11. Retrieved from CINAHL Plus with Full Text database.

Dromgoole, P. (2004). NSF for diabetes. Standard 7 — emergency care. Primary Health Care, 14(7), 39-42. Retrieved from CINAHL Plus with Full Text database.

E. Solá, S. Garzón, S. García-Torres, P. Cubells, C. Morillas, & A. Hernández-Mijares. (2006). Management of diabetic ketoacidosis in a teaching hospital. Acta Diabetologica, 43(4), 127-30. Retrieved April 30, 2010, from Health Module

Farhat, D. (2001). Disorders of glucose. Topics in Emergency Medicine, 23(4), 27-43. Retrieved from CINAHL Plus with Full Text database.

Hall, G.. (2008). Structured education in diabetes. Practice Nurse, 36(5), 35-37. Retrieved April 29, 2010, from ABI/INFORM Trade & Industry

Gelder, C. (2009). Care of adolescents in transition. Practice Nursing, 20(9), 444-448. Retrieved from CINAHL Plus with Full Text database

Guthrie, R., & Guthrie, D. (2004). Pathophysiology of diabetes mellitus. Critical Care Nursing Quarterly, 27(2), 113-125. Retrieved from CINAHL Plus with Full Text database.

Huether

Pathophysiology : the biologic basis for disease in adults and children / Kathryn L. McCance, Sue E. Huether [editors].

St. Louis, Mo. : Elsevier Mosby, c2006.

Hall, G.. (2008). Structured education in diabetes. Practice Nurse, 36(5), 35-37. Retrieved April 30, 2010, from ABI/INFORM Trade & Industry.

Hill, J. (2009). Reducing the risk of complications associated with diabetes. Nursing Standard, 23(25), 49-55. Retrieved from CINAHL Plus with Full Text database.

Jean-Louis Chiasson, Nahla Aris-Jilwan, Raphaël Bélanger, Sylvie Bertrand, & et al. (2003). Diagnosis and treatment of diabetic ketoacidosis and the hyperglycemic hyperosmolar state. Canadian Medical Association. Journal, 168(7), 859-66. Retrieved April 28, 2010, from ProQuest Health and Medical

Kisiel, M., & Marsons, L. (2009). Recognizing and responding to hyperglycaemic emergencies. British Journal of Nursing (BJN), 18(18), 1094-1098. Retrieved from CINAHL Plus with Full Text database.

McDowell, J., Coates, V., Davis, R., Brown, F., Dromgoole, P., Lowes, L., et al. (2009). Decision-making: Initiating insulin therapy for adults with diabetes. Journal of Advanced Nursing, 65(1), 35-44.

Moore, T. (2004). Diabetic emergencies in adults. Nursing Standard, 18(46), 45. Retrieved from CINAHL Plus with Full Text database.

Palmer, R. (2004). An overview of diabetic ketoacidosis. Nursing Standard, 19(10), 42-44. Retrieved from CINAHL Plus with Full Text database.

Spray, J. (2009). Type 1 diabetes: identifying and evaluating patient injection technique. British Journal of Nursing (BJN), 18(18), 1100-1105. Retrieved from CINAHL Plus with Full Text database.

Trachtenbarg, D. (2005). Diabetic ketoacidosis. American Family Physician, 71(9), 1705. Retrieved from CINAHL Plus with Full Text database.

Verhey, M., Levy, J., & Schmidt, R. (2004). Type I diabetes & patient compliance. Retrieved from CINAHL Plus with Full Text database.

Robinson, N. C., (2005). Lewis