Initial Assessment and Treatment of Diabetic Ketoacidosis (DKA) in the Elderly

Ria E Byrne

Abstract

Diabetic ketoacidosis (DKA) is a medical emergency which affects thousands of people each year in the United Kingdom and requires immediate medical attention. This article explores the pathophysiology and initial assessment and treatments which are essential within the first hour of admission. For DKA to be diagnosed, there must be the presence of hyperglycemia, ketosis and acidosis which may occur for a variety of reasons. It is essential that a rapid assessment is carried out for which the use of a framework or tool can be useful in addition to specific tests such as blood glucose and ketone monitoring, as well as identifying the underlying cause. The initial treatment phase aims to restore circulating volume, reduce blood glucose levels, to correct any electrolyte imbalances and to reduce ketone levels which in turn corrects the acidosis. This involves a combination of intravenous fluids, insulin and potassium, and requires continuous monitoring and adjustment. Communication with both the patient and specialist services is important throughout every stage. A combination of communication techniques should be used and factors such as socioeconomic status and level of heath literacy should be taken into account to ensure the best outcome for the patient, and to prevent future readmissions with DKA.

Introduction

Diabetic ketoacidosis (DKA) is an acute episode which can present in those with insulin dependent diabetes mellitus. According to the National Diabetes Audit (Government Statistical Service, 2013), during the period April 2010 – March 2012, 10,434 people with type I diabetes were admitted to hospital with DKA in the UK. DKA is a complex medical emergency with several stages to the treatment, involving reassessment at every stage. This article will explore the first 60 minutes of assessment and appropriate treatments, pathophysiology of deterioration and the importance of communication with the patient and other applicable services using a case study of Patient A, a 68 year old lady with insulin dependent diabetes mellitus who has been admitted to the Medical Assessment Unit (MAU) with suspected DKA.

Author details
Ria Ellen Byrne
Final Year Student Nurse, BN
Dual Field Child and Adult Nursing, University of Southampton
Email: ria.byrne1@gmail.com

Pathophysiology

In order to be able to effectively assess a patient, recognise signs of deterioration, understand and prioritise treatments and be able to educate the patient, it is important to understand the pathophysiology and evidence behind it. DKA is a state which can occur in those with diabetes, particularly type I diabetes; where the destruction of beta cells causes a complete deficiency of insulin. It is common in patients with newly diagnosed type I diabetes or may be the event which leads to the diagnosis of the common long term condition (Fowler, 2009). DKA can also occur at any time if triggered by another factor, most commonly poor insulin control or infection. Less frequent causes include myocardial infarction, pulmonary embolism, cerebral accidents or protracted vomiting (Wallace and Matthews, 2004), as well as pancreatitis and drugs (Kitabchi et al, 2006).

DKA occurs when three events take place within the body; hyperglycaemia, ketosis and acidosis. Hyperglycaemia occurs as a result of the deficiency of insulin apparent in type I diabetes in combination with an increase in hormones released in response to stress, such as glucagon, cortsol, catecholamine, epinephrine and growth hormone (Fowler, 2009; Noble-Bell and Cox, 2014). Insulin deficiency prevents glucose being utilized by tissues within the body and also increases gluconeogenesis in the liver, both resulting in hyperglycaemia (Fowler, 2009). Insulin deficiency and the increased production of hormones also cause lipolysis to occur. This is the breakdown of fatty acids in the body and results in the release of AcetylCoA, which in turn, is converted into ketones; acetone, acetoacetate and most importantly beta-hydroxybutyrate. This is ketosis and is what causes acidosis to occur. Beta-hydroxybutyrate can initally be present in the body without the presence of acidosis as the acidity is buffered by bicarbonate in the body, resulting in low bicarbonate in the blood until reserves become depleted and acidosis takes over (Laffel, 1999; Wallace and Matthews, 2004).

In order to deal with hyperglycaemia, the body attempts to excrete the excess glucose in the urine along with water, causing polyuria and resulting in dehydration. UK research has found that patients in a state of DKA can experience up to six litres of fluid loss (Freudenthal

Keywords
Diabetic ketoacidosis, elderly, acute, assessment, treatment, communication

et al, 2013). This in turn causes an electrolyte imbalance in the body which also later needs to be addressed once the immediate threat to life has been removed (Eledrisi et al, 2006; Fowler, 2009).

Assessment

The most important action on admission is to carry out an initial rapid assessment of the patient. The ABC framework is commonly used for the initial assessment of trauma patients; however it allows clinicians to identify any immediate, life threatening concerns which is essential when treating any patient. Rapid identification of any life threatening issues allows appropriate interventions to be put into place in order to reduce or prevent risk of death or long term disability as a result (Skinner and Driscoll, 2013). Patient A is drowsy but is able to respond verbally to staff, therefore demonstrating that her airway is patent, she is breathing, and has adequate circulation.

Once any immediate threats to life have been identified and appropriately actioned, a more detailed assessment can be carried out. According to the National Institute for Health and Clinical Excellence clinical guideline 50 (NICE, 2007), a minimum of heart rate, respiratory rate, systolic blood pressure, level of consciousness, oxygen saturation and temperature should be recorded both at initial assessment and at regular intervals; at least every 12 hours. Risk of deterioration of a patient experiencing an acute episode is high, therefore NICE also recommend the use of a track and trigger system in all acute settings in order to detect signs of deterioration as early as possible. One such system that is commonly used is a Modified Early Warning Score (MEWS) (appendix A - Quinton and Higgins, 2012)

A MEWS score can be calculated by collection of vital signs, and should be used in combination with clinical judgement (Alam et al, 2014). This score gives an indication of risk of deterioration and triggers certain actions such as informing the nurse in charge or senior clinician. Calculation of a MEWS has been found to trigger a more timely response to deterioration and therefore an improved outcome for the patient (Ludikhuize et al, 2012). However, research has suggested that an accurate MEWS is not always calculated due to certain vital signs, most commonly respiratory rate, not being recorded (Ludikhuize et al, 2011). On admission to MAU, following the recording of vital signs, Patient A has a total MEWS of six. This means that the nurse in charge, junior doctor and consultant should be contacted for immediate review, as well as the critical outreach team. NICE clinical guideline 50 also recommends that the frequency of calculation of a MEWS should be increased if abnormal physiology is recorded. As Patient A has a MEWS of six, the system denotes that her MEWS should be recalculated every 30 minutes until it has decreased to less than four. Research also suggests that calculation of a MEWS score, not only at the initial assessment and then every 12 hours, but at regular intervals throughout the day, is effective in recognising deterioration (Ludikhuize et al, 2014). This suggests that for best patient outcome and early detection of any deterioration, the MEWS should be calculated at regular intervals for every patient not simply as often as suggested by calculation of the previous score.

It has been found that MEWS is useful in detecting DKA (Pepper et al, 2012) as defined by the criteria set out by the American Diabetes Association (appendix B - American Diabetes Association cited in Pepper et al, 2012). Although this study was not carried out in the UK, the findings may helpful to guide further research within the UK as variations of the MEWS are used throughout the world. Also, the criterion which was used to define DKA was created in the USA which is a country with similar economic status, lifestyles and patterns of ill health to the UK, however the criteria differs slightly to those published by Joint British Diabetes Societies Inpatient Care Group (JBDS, 2010) and so this should be taken into account.

Abnormal vital signs are an indication of the underlying pathophysiology discussed above; the presence of hyperglycaemia, ketoneamia/ketonuria and acidaemia (JBDS, 2010). In order to test for hyperglycaemia and ketonaemia, blood glucose and ketone levels should initially be checked by carrying out a finger prick test. A result of greater than 11mmols/L of glucose and 3mmols/L or more of ketones may be an indication of DKA (Savage, 2011; Savage et al, 2011). If a ketone meter is unavailable, the patient's urine can instead be tested for ketonuria by carrying out a dipstick test; a result of 2+ or more may also indicate DKA. In order to test for acidaemia, a venous blood sample must be taken; venous samples have been found to be as reliable as arterial samples in this instance and obtaining a venous sample is much less invasive for the patient (Kelly, 2006; Savage et al, 2011). If the pH is less than 7.3 or bicarbonate is less than 15mmol/l, this is further indication of DKA (Savage, 2011; Savage et al, 2011). If the results for all the above tests are positive, a diagnosis of DKA can be made and appropriate treatments put in place. Following these tests Patient A is found to have a blood sugar of 28mmol/l, ketones of 3.6mmol/l (and urine ketones of 3+) with a blood pH of 7.1. Bedside monitoring of blood glucose and ketones should be repeated hourly for the first six hours (JBDS, 2010). A venous blood gas also allows the identification of some other abnormal values such as electrolyte levels, however further blood tests including full flood blood count, urea and electrolytes and cultures should be carried out to allow more in depth analysis, as well as to identify the presence of any infection markers. Further investigations such as an Electrocardiogram (ECG) and a chest x-ray should also be carried out as part of the initial assessment (Kitabchi, 2006; JBDS, 2010; Noble-Bell and Cox, 2014).

Treatment

The initial assessment determines the cause of the acute episode and identifies the symptoms which need to be treated first allowing clinicians to prioritise appropriate therapeutic interventions. According to the JBDS (2010) the aim to treatment of DKA is to restore circulating volume, reduce blood glucose levels, correct any electrolyte imbalances and to reduce ketone levels therefore correcting acidosis. The following interventions should take place within the first hour following admission, any part of the interventions which should take place after the first hour are not discussed in detail.

The first step in treating DKA is to administer intravenous fluids (IVI) of 0.9% sodium chloride solution in order to restore circulating volume. As mentioned above, a patient presenting with DKA

may have around six litres of fluid deficit, therefore it is essential that this debt is restored immediately upon admission. If the patient's systolic blood pressure is less then 90mmHg, an initial 500mls should be given over 10-15minutes, this can be repeated if blood pressure remains below 90mmHg. As Patient A has a systolic blood pressure of 92mmHg, she is commenced on the normal regime of 1000mls over the first hour. After the first hour, the fluids should then be changed to include potassium in order to address the inevitable electrolyte imbalance - 0.9% sodium chloride with potassium chloride should then be given at a rate of 500mls per hour for the next four hours (appendix C – JBDS, 2010). This regime also notes that particular caution should be taken with the elderly. This is reiterated by earlier research which states that the regime should be administered at a slower rate if the patient is elderly, as a higher infusion rate may increase risk of respiratory distress syndrome (Savage and Kilvert, 2006).

Once the IVI has been started, the next priority is to address the hyperglycaemia by administration of insulin, with the aim to reduce blood glucose by 3mmol/L/hr. It is recommended that 50 units of quick acting insulin such as Actrapid ®, should be made up to 50mls with 0.9% saline and administered at a rate of one unit/kg/hour, alongside the IVI (JBDS, 2010). Evidence has shown that insulin given according to weight rather than a set dose, allows for more accurate treatment especially in the instance of obesity and other insulin resistant states such as pregnancy (Savage, 2001; Savage et al, 2011), and therefore allows more timely resolution of DKA and improved patient outcome. However, older research suggests that insulin should be given at an initial rate of six units per hour which can then be increased to 10 units per hour if blood glucose has not fallen after two hours (Savage and Kilvert, 2006). It has been suggested that using the older recommendation may be better as it leaves less room for calculation errors and allows flexibility in instances where very high doses of insulin are required which are not relative to weight (Taylor, 2012). Despite this, for Patient A the insulin is successfully calculated and administered using an estimated weight in accordance with modern guidance.

Once the fluid loss and hyperglycaemia have been addressed, the next aim is to prevent further production of ketones, clear the blood of ketones, and reverse the acidosis. The replacement of potassium and reduction of hyperglycaemia should result in the reduction of blood ketones by at least 0.5mmols/L/hr and an increase of bicarbonate by 3mmol/L/hr. If this does not happen, the insulin infusion may be increased by one unit per hour until targets are achieved. Due to the insulin infusion, blood glucose levels may fall more rapidly than ketone levels. If blood glucose levels fall below 14mmols/L, 10% dextrose should be commenced alongside the IVI at a rate of 125mls/hr. Potassium levels should be continuously monitored and treatment adjusted to ensure they remain within normal levels of 4-5.5mmols/L. It is expected that DKA should be fully resolved within 24 hours of the commencement of the treatment regime and patients should be back on their usual insulin regime (JBDS, 2010).

Communication

Guidelines recommend that following admission with DKA, the diabetes specialist team are contacted as soon as possible, ideally within the first hour (JBDS, 2010) and that they are seen by the team within 24 hours (Price et al, 2013). Therefore is it essential that there is timely communication with the specialist team to ensure that this is able to happen. It is also recommended that patients admitted with DKA receive follow up upon discharge, including a review of their condition and medications, and risk assessment of DKA recurring (Noble-Bell and Cox, 2014) enabling interventions to be put into place to reduce this risk. In addition to follow up, patients should have access to relevant education within three months of discharge (Price et al, 2013) to improve patient understanding and further decrease risk of another admittance with DKA.

As mentioned above, poor insulin control was one of the most common causes of DKA and is most likely the cause of Patient A's admission with DKA. Research has found that adherence is lowest in certain health conditions, of which one is diabetes (DiMatteo, 2004). Therefore it is important to investigate why patients, like Patient A, do not adhere to the recommended insulin regime prescribed by medical specialists. Communication with patients has been proven to be essential for patient education and therefore compliance (Zolnierek and DiMatteo, 2009). Talking to Patient A throughout the assessment and treatment brings to light her lack of understanding of her current condition, despite her several previous admissions for treatment of DKA. She seems interested, asking questions about the tests being carried out and what the results mean. Two way communication with patients in which they are asked about their experiences and concerns has been found to be beneficial in increasing patient knowledge, compliance and outcomes (Stevenson et al, 2004). Discussing her condition and treatments with Patient A, both during her hospital stay and through follow up at home, may be influential in improving in her knowledge and adherence and potentially preventing future admissions with the same condition.

A meta-analysis of research into the impact of socio-economic status (SES) of the patient on patient-physician communication has found that patients with lower SES experience less effective communication. Physicians often assume that patients of lower SES have less desire for information or less understanding and therefore physicians are less informative; however, ineffective communication is not entirely the fault of the health care professionals. Patients with lower SES often have a more passive communication style, meaning they ask fewer questions, express less opinions and less desire to make decisions (Willems et al, 2005). This emphasises the need for two way communication and encouraging patients to express their needs, opinions and to make fully informed decisions about their own care. A study into communication between physicians and patients with diabetes found that those with poor functional health literacy, often linked to lower SES, were more likely to be under informed about their condition and how to manage it (Schillinger et al, 2004). This suggests that physicians need to tailor their communication to individual patients, taking into consideration their literacy levels in order to optimise patient understanding and therefore compliance. Although this study was carried out on patients with type II diabetes, it included those whose diabetes was insulin controlled. It has been suggested that basic techniques such as using simple language, giving

additional written advice and speaking slowly can significantly increase patient understanding of health information (Schwartzberg et al, 2007) and therefore may significantly increase adherence, improve patient management of their own condition and reduce unnecessary hospital admissions. Keeping patients and families informed about their care is part of providing psychological support (Noble-Bell and Cox, 2014). Keeping patients fully informed at all stages of investigations and treatment enables patients to feel more in control of their own condition and in turn may improve patient satisfaction and psychological wellbeing (Funnell et al, 2012).

Conclusion

Diabetic ketoacidosis is a medical emergency requiring immediate medical attention. It is important that health care professionals understand the pathophysiology behind DKA in order to effectively assess, prioritise and treat the patient. An appropriate track and trigger tool is essential for detailed assessment and rapid detection of deterioration. Recent guidelines based on the vast available modern evidence should be adhered to for best patient outcome. The cause of DKA should also be taken into account, especially in the case of poor insulin control. Effective communication with healthcare professionals, including specialist services both in hospital and in the community, is essential. This allows timely and effective assessment and treatment, as well as improvement of patient education in order to increase adherence and reduce the number of future admissions. Research also emphasises the need to keep patients and their families fully informed throughout as part of providing psychological support. Unfortunately this is often forgotten due to time constraints but is equally important in improving health outcomes, patient satisfaction and psychological wellbeing.

References

Alam N, Hobbelink E, Tienhoven A, Ven P, Jansma E, Nanayakkara P (2014) The Impact of the Use of the Early Warning Score (EWS) on Patient Outcomes: A Systematic Review. Resuscitation 85(5): 587-594

DiMatteo M (2004) Variations in Patients' Adherence to Medical Recommendations: A Quantitative Review of 50 Years of Research. Medical Care 42(3): 200-209

Eledrisi M, Alshanti M, Shah M, Brolosy B, Jaha N (2006) Overview of the Diagnosis and Management of Diabetic Ketoacidosis. American Journal of the Medical Sciences 331(5): 243-251

Fowler M (2009) Hyperglycemic Crisis in Adults: Pathophysiology, Presentation, Pitfalls, and Prevention. Clinical Diabetes 27(1): 19-23

Freudenthal R, Tufton N, Podesta C, Mulholland R, Rossi M (2013) Fluid Management in Diabetic Ketoacidosis: Are We Adhering to Recommended Guidelines? British Journal of Diabetes & Vascular Disease 13(3): 138-142

Funnell M, Brown T, Childs B et al (2012) National Standards for Diabetes Self-Management Education. Diabetes Care 35(1): S101-108

Government Statistical Service (2013) National Diabetes Audit – 2011-2012: Summary Report. Unknown: Health and Social Care Information Centre

Joint British Diabetes Societies Inpatient Care Group (2010) The Management of Diabetic Ketoacidosis in Adults. Malmesbury: NHS Diabetes

Kelly A (2006) The Case for Venous Rather Than Arterial Blood Gases in Diabetic Ketoacidosis. Emergency Medicine Australasia 18(1): 64-67

Kitabchi A, Umpierrez G, Murphy M, Kreisberg R (2006) Hyperglycemic Crises in Adult Patients with Diabetes: A Consensus Statement from the American Diabetes Association. Diabetes Care 29(12): 2739-2748

Laffel L (1999) Ketone Bodies: A Review of Physiology, Pathophysiology and Application of Monitoring to Diabetes. Diabetes/Metabolism Research and Reviews 15(6): 412-426

Ludikhuize J, Borgert M, Binnekade J, Subbe C, Dongelmans D, Goossens A (2014) Standardized Measurement of the Modified Early Warning Score Results in Enhanced Implementation of a Rapid Response System: A Quasi-Experimental Study. Resuscitation 85(5): 676-682

Ludikhuize J, Jonge E, Goossens A (2011) Measuring Adherence Among Nurses One Year After Training in Applying the Modified

Early Warning Score and Situation-Background-Assessment-Recommendation Instruments. Resuscitation 82(11): 1428-1433

Ludikhuize J, Smorenburg S, Rooij S, Jonge E (2012) Identification of Deteriorating Patients on General Wards; Measurement of Vital Parameters and Potential Effectiveness of the Modified Early Warning Score. Journal of Critical Care 27(1): 424e.7-424e.13 Available from: http://ac.els-cdn.com/S0883944112000160/1-s2.0-S0883944112000160-main.pdf?_tid=9f378538-c0b6-11e3-b2a8-00000aacb360&acdnat=1397137814_1a4b5dff6ff22abdbc54a51e770a0d6d [Accessed 23rd August 2015]

National Institute for Health and Clinical Excellence (2007) Acutely Ill Patients in Hospital: Recognition of and Response to Acute Illness in Adults in Hospital. London: National Institute for Health and Clinical Excellence

Noble-Bell G, Cox A (2014) Management of Diabetic Ketoacidosis in Adults. Nursing Times 110(10): 14-17

Pepper D, Williams P, Haynes D (2012) An Algorithm Using The Modified Early Warning Score To Diagnose Hyperglycemic Emergencies In Resource Limited Settings. IN: Schlessinger, S. (ed) Proceedings Of The Seventh Annual Department Of Medicine Research Day Mississippi 12 April 2012 Mississippi: University of Mississippi Medical Center Department of Medicine 43-45

Price H, Thomsett K, Newton I, Alderson S, Hillson R (2013) Developing Best Practice Tariffs for Diabetic Ketoacidosis and Hypoglycaemia. Practical Diabetes 30(1): 6-8

Quinton S, Higgins Y (2012) Adult Modified Early Warning Score (MEWS) Policy and Escalation Pathway. Heart of England NHS Foundation Trust Available from: http://www.heartofengland.nhs.uk/wp-content/uploads/MEWS.pdf [Accessed 23rd August 2015]

Savage M (2011) Management of Diabetic Ketoacidosis. Clinical Medicine 11(2): 154-156

Savage M, Dhatariya K, Kilvert A et al (2011) Joint British Diabetes Societies Guideline for the Management of Diabetic Ketoacidosis. Diabetic Medicine 28(5): 508-515

Savage M, Kilvert A (2006) ABCD Guidelines for fhe Management of Hyperglycaemic Emergencies in Adults. Practical Diabetes International 23(5): 227-231

Schillinger D, Bindman A, Wang F, Stewart A, Piette J (2004) Functional Health Literacy and the Quality of Physician–Patient Communication Among Diabetes Patients. Patient Education and Counseling 52(3): 315-323

Schwartzberg J, Cowett A, VanGeest J, Wolf M (2007) Communication Techniques for Patients With Low Health Literacy: A Survey of Physicians, Nurses, and Pharmacists. American Journal of Health Behaviour 31(1): 96-104

Skinner D, Driscoll P, eds (2013) ABC of Major Trauma (Fourth Edition). John Wiley & Sons, Ltd.

Stevenson F, Cox K, Britten N, Dundar Y (2004) A Systematic Review of the Research on Communication Between Patients and Health Care Professionals About Medicines: The Consequences for Concordance. Health Expectations 7(3): 235-245

Taylor R (2012) Insulin Dose Requirement in Diabetic Ketoacidosis. Diabetic Medicine 29(1): 153

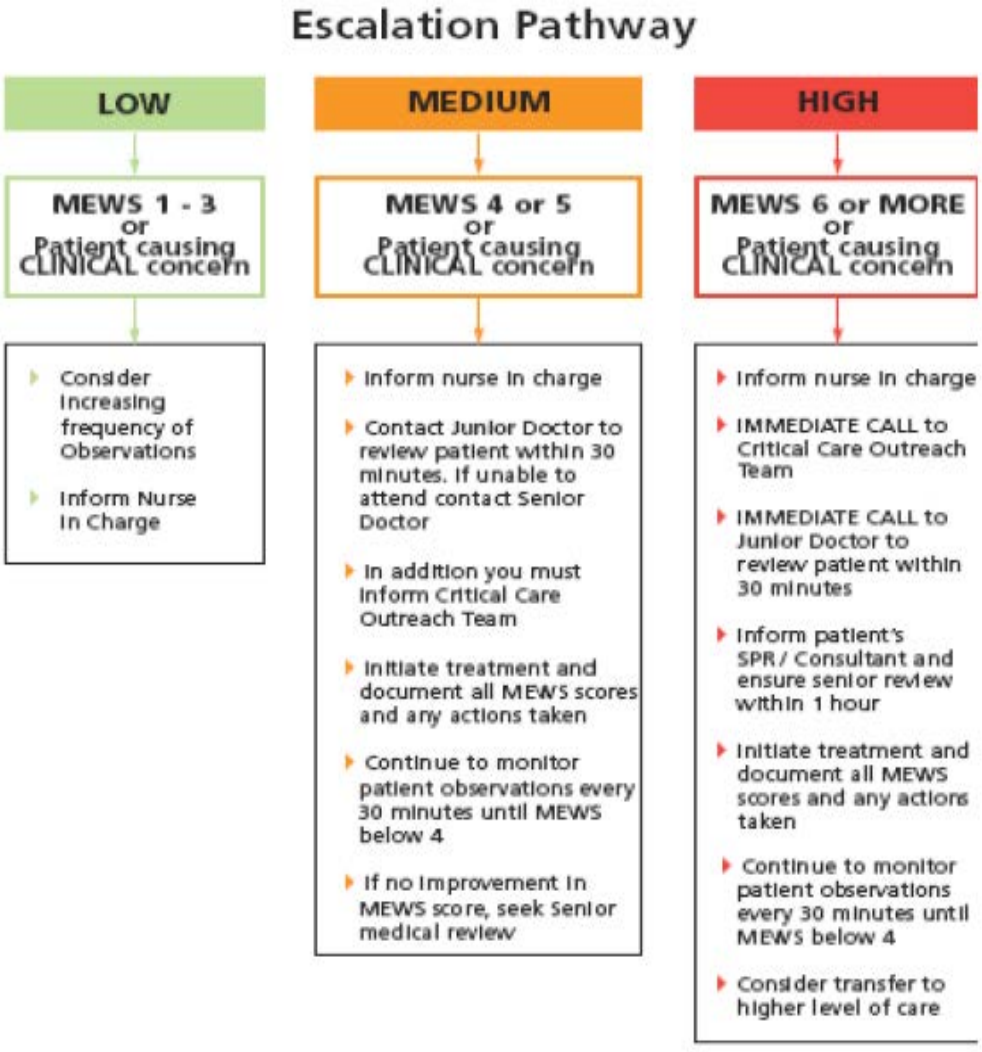
Wallace T, Matthews D (2004) Recent Advances in the Monitoring and Management of Diabetic Ketoacidosis. Quarterly Journal of Medicine 97(12): 773-780

Willems S, De Maesschalck S, Deveugele M, Derese A, De Maeseneer J (2005) Socio-Economic Status of the Patient and Doctor–Patient Communication: Does it Make a Difference? Patient Education and Counseling 56(2): 139-146

Zolnierek K, DiMatteo M (2009) Physician Communication and Patient Adherence to Treatment: A Meta-Analysis. Medical Care 47(8): 826-834

Appendix A MEWS Parameters and Escalation Pathway (Quinton and Higgins, 2012)

Score / Categories	3	2	1	0	1	2	3
Respirations		8 or less		9 -16	17 - 20	21 - 29	30 or more
O^2 SATs				94% or more	90 – 93%	85 – 89 %	84% or less
SBP	70 or less	71 - 80	81 - 100	101 - 199		200 or more	
Pulse				51 - 100	101 - 110	111 - 129	130 or more
AVPU			New Confusion / Agitation	Alert	Voice	Pain	Unresponsive
Temperature		35 or less	35.1 - 36	36.1 – 37.5	37.6 – 38.1	38.2 or more	
Urine				No Concerns	21 - 35	1 - 20	Nil



Appendix B Classification of Hyperglycaemic Emergencies According to Diagnostic Criteria (American Diabetes Association cited in Pepper et al, 2012)

Table 2. Classification of hyperglycaemic emergencies according to diagnostic criteria.

Diagnostic Criteria	Diabetic KetoAcidosis (DKA)		Hyperosmolar Hyperglycaemic State (HHS)	Hyperglycaemia with Dehydration (HD)
	Mild/ Moderate (MDKA)	Severe (SDKA)		
Plasma glucose (mmol/l)	≥ 15	≥ 15	≥ 30	≥ 15
Arterial pH	7.0 – 7.30	< 7.00	> 7.30	> 7.30
Serum Bicarbonate (mEq/l)	10 – 17.9	< 10	> 15	≥ 18
Serum Ketones	≥ 1+	≥ 1+	≤ 1+	≤ 1+
Serum Osmolality (mOsm/kg)	Variable	Variable	≥ 330	< 330
Mental Status	Alert / drowsy	Stupor / coma	Stupor / coma	Alert

Appendix C Typical Fluid Replacement Regimen (JBDS, 2010)

Systolic BP on admission 90 mmHg and over

Below is a table outlining a typical fluid replacement regimen for a previously well 70kg adult. This is an illustrative guide only. A slower infusion rate should be considered in young adults (see Controversial Areas).

Fluid	Volume
0.9% sodium chloride 1L *	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours
Re-assessment of cardiovascular status at 12 hours is mandatory, further fluid may be required	

*Potassium chloride may be required if more than 1 litre of sodium chloride has been given already to resuscitate hypotensive patients