Hyperglycaemic Emergencies – A Need For An Appropriate Diagnosis.

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Abstract: Background: Hyperglycaemic Emergencies, He, Is An Acute Metabolic Complication Of Diabetes Mellitus (Dm) Characterized By Hyperglycaemia, Dehydration, Changes In Plasma Osmolality And Electrolytes Disorders In Which Soluble Insulin And Intravenous Fluids Are Required For Correction. Treatment Outcome Is Determined Largely By Its Precipitating Factors And Co-Morbid Conditions, Accurate Diagnosis And Proper Management. Objectives: To Highlight The Need For Accurate Diagnosis Of Diabetic Keto-Acidosis, Dka, Hyperosmolar Hyperglycaemic State, Hhs Etc, Draw Attention To Errors In Management Of Hyperglycaemic emergencies And Create Awareness For Early Endocrinology Review. Case Reports: Case 1Mrs F.N Is 46 Years Old, Living With Diabetes, Admitted Via The Accident And Emergency (A & E) Department On Account Of Cerebro-Vascular Accident, Cva (Stroke) With Random Blood Glucose, Rbg Of 563mg/Dl. History Of Severe Headache, Vomiting, Fits, Fever And Neck Stiffness Were Absent. An Urgent brain Ct Scan Excluded A Haemorrhagic Stroke. He Presented At A & E To A Medical Unit Who Admitted And Managed For Cva (Stroke). Endocrinology Review Was Sought One Week Post Admission When His Condition Has Deteriorated Case 2Mr Z.O Is A 63 Year Old FarmerAdmitted At The A & E Unit With A Diagnosis Of Poor Glycaemic Control And Right Diabetic Foot Ulcer With Bacterial Infections. Rbg At Presentation Was 1015mg/Dl. He Was Placed On Antibiotics And Subcutaneous T.D.S Soluble Insulin. Endocrinology Review Was Asked For After The Patient Has Been In The Medical Ward For 9 Days And In A Poorer Clinical State Than When He Presented At A & E. Conclusion Hyperglycaemic Emergencies Were Not Diagnosed Appropriately In These Cases Admitted By Other Medical Specialties, Precipitants And Co-Morbid Conditions Were Not Identified For What They Were While Hyperglycaemic emergencies Were Not Managed Based On The Local Treatment Guidelines With Early Involvement Of The Diabetes Care Team.

Key Words: Hyperglycaemic emergencies, Early endocrinology Review, Precipitants of hyperglycaemic emergencies

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I. Introduction

Hyperglycaemic Emergency Refers To Potentially Fatal Acute Metabolic Complications Of Dm Typically Represented By Dka And Hhs¹. The Hallmark² Of Hyperglycemic Emergency (Dka, Hhs Etc) Is Decreased Insulin Levels Which May Be Absolute Or Relative, Decreased Glucose Use And Increased Hepatic Gluconeogenesis From Elevated Counter-Regulatory Hormones Such As Cortisol, Glucagon And Catecholamines, Increased Lipolysis And Ketogenesis In Dka.

Hyperglycemic Emergencies Are Associated With Significant Morbidity And Mortality³. Hyperglycemic Emergency Constitutes One Of The Main Reasons Why A Diabetic Patient Is Admitted In Nigeria⁴,⁵, Accounting For 40% Of Dm Admissions⁶ In An Urban Hospital In Lagos, South Western Nigeria. In Ilorin, The Crude Mortality⁷ Rate For Dka And Hhs Was 22% And 25% Respectively While In Ife, Dka Was The Commonest Cause Of Diabetic Deaths⁸.

In Dka⁹, Absolute Insulin Deficiency Results In Increased Lipolysis, Increased Free Fatty Acid Generation Which Undergoes Beta-Oxidation In The Liver Mitochondria To Form Ketoacids (3-Beta Hydroxybutyrate, Acetoacetate And Acetone) Resulting In Metabolic Acidosis.

There Is, Also, Increased Hepatic Glycogenolysis And Increased Proteolysis With Enhanced Generation Of Amino Acid Residues Which Together With The Increased Glycerol Generated From Lipolysis Serve As Substrates For Increased Gluconeogenesis. With Decreased Glucose Utilization By The Peripheral Tissues Due
To Insulin Deficiency, Increased Glycogenolysis And Gluconeogenesis, There Is Hyperglycemia Which Causes Osmotic Diuresis Leading To Marked Dehydration And Electrolyte Losses. Hhs Occurs In The Setting Of Relative Insulin Deficiency Accompanied By Release Of Counter-Regulatory Hormones (Glucagon, Cortisol, Catecholamines Etc) But Presence Of Some Insulin Reserve Prevents Lipolysis Leading To Minimal Or Absent Ketogenesis.

Precipitating Factors For Dka Include Infections Of Which The Commonest Is Uti And Foot Ulcers/Gangrene. Others Include Stroke, Myocardial Infarction Etc.

The Following Measures Are Involved In Hyperglycaemic emergency Management: General Measures, Fluid Replacement, Insulin Therapy, Electrolytes Replacement, Monitoring, Correction Of Hyperosmolality, Identification And Treatment Of Underlying Cause(S), Resolution And Conversion To Home Therapies. Treatment Of Such Precipitating Factors As Infections, Foot Ulcers And Sepsis Should Be Carried Out Concurrently To Achieve A Favorable Outcome.

In Many Cases, Hyperglycaemic emergencies Present First To The General Practitioner At The Peripheral Hospital Or To The Non Endocrinology Unit Of Tertiary Hospitals (Medical Officers Of Emergency Departments, General Surgeons, Obstetricians Or Other Internists). A Need For This Report, Therefore, Exists To Create Awareness Of The Dangers Of Not Making The Appropriate Diagnosis Of Hyperglycaemic emergency Or Involving The Diabetes Care Team Early In The Management.

Aim/Objectives:
To HighlightThe Need For Accurate Diagnosis Of The Hyperglycaemicemergency Pattern (Dka, Hhs, Etc), Draw Attention To Errors In Management Of Hyperglycaemic emergencies And To The Need For An Early Endocrinology Review.

Case 1:
Mrs F.N Is A 46 Year Old School Teacher Living With Diabetes For More Than 8 Years. While Watching Her Favourite Cartoon Movie One Afternoon, She Developed Sudden Onset Weakness Of The Right Side Of Her Body, Facial Weakness And Speech Impairment. She Was, However, Conscious. On Presentation At The A And E Unit Of Federal Medical Centre, Fmc, Umuahia Some 4 – 6 Hours Later, She Gave No History Of Severe Headache, Vomiting, Fits, Fever Or Neck Stiffness.

Physical Examination Showed A Conscious Middle Aged Woman, Afebrile, Dehydrated, Pulse Rate 96/Min, Bp 150/90mmhg, Had Lateralizing Signs With No Neck Stiffness. Chest And Abdomen Were Essentially Normal. Her Random Blood Glucose At Presentation Was 563mg/Dl And Her Other Results Are As Stated Below:

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Results</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na (mmol/L)</td>
<td>139.0</td>
<td>135 – 155</td>
</tr>
<tr>
<td>K (mmol/L)</td>
<td>4.6</td>
<td>3.5 – 5.5</td>
</tr>
<tr>
<td>Ca (mmol/L)</td>
<td>2.1</td>
<td>2.2 – 2.6</td>
</tr>
<tr>
<td>Cl (mmol/L)</td>
<td>107</td>
<td>96 – 108</td>
</tr>
<tr>
<td>HCO3 (mmol/L)</td>
<td>15</td>
<td>24 – 30</td>
</tr>
<tr>
<td>PO4 (mmol/L)</td>
<td>1.0</td>
<td>0.8 – 1.4</td>
</tr>
<tr>
<td>Urea (mg/dl)</td>
<td>46</td>
<td>15 – 55</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>0.7</td>
<td>0.5 – 1.5</td>
</tr>
<tr>
<td>3-Beta-hydroxybutyrate</td>
<td>6.9</td>
<td>&lt; 5</td>
</tr>
</tbody>
</table>

Urinalysis Showed Glycosuria +++, Ketonuria+++ And Proteinuria+ While Urine Culture Did Not Grow Any Organism. Urgent Brain Ct Scan Done Same Day Showed No Hyper-Dense Lesion (Negative For Hemorrhagic Stroke) And Other Baseline Results Were Essentially Normal. She Was Initially Admitted And Managed By A Non-Endocrinology Unit For Ischemic Cva (Stroke) With Normal Saline, Antioxidants, Anti-Platelet, Oral Anti-Diabetic Agents And Physiotherapy.

One Week Later, The Patient Had Become Unconscious With A Random Blood Glucose Fluctuating Between 400 – 600mg/Dl And The Diabetes Care Team Was Invited To Review Because Of Poor Glycaemic Control. Unfortunately, The Patient Passed On Later.

Case 2:
Mr. Z.O, A 63 Year Old Farmer, Not Previously Known To Be Living With Diabetes, Sustained A Shrub Stump Injury On The Sole Of His Right Foot While In His Farm. A Neighbor-Hood Nurse Gave Him Some Initial Treatment. Three Weeks Later, Mr. Z.O Presented In The A And E Unit Of Fmc, Umuahia With A Foul Smelling Wound On His Right Foot, Fever, General Body Weakness And Impaired Consciousness.
Examination showed an acutely ill-looking man who was febrile (temperature 38.9 degrees centigrade), dehydrated but not pale. Musculoskeletal system examination showed a right foot ulcer on the plantar surface measuring 8 x 13 cm, irregular in shape, involving the underlying muscles and tendons with much slough, oozing offensive, foul-smelling discharges. The surrounding skin was hyperpigmented and the whole foot was swollen. He was conscious but drowsy. His pulse was 104/minute, blood pressure 100/70 mmHg but the other systemic examinations were essentially normal.

Rbg at presentation was “Hi” by a glucometer (laboratory result was 1015 mg/dl). A working diagnosis of poor glycaemic control with right diabetic foot ulcer was made by the managing medical unit. Other investigation results are shown below:

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<th>Reference Range</th>
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<td>Na (mmol/L)</td>
<td>13</td>
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<tr>
<td>K (mmol/L)</td>
<td>4.9</td>
<td>3.5 – 5.5</td>
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<tr>
<td>Ca (mmol/L)</td>
<td>2.3</td>
<td>2.2 – 2.6</td>
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<tr>
<td>Cl (mmol/L)</td>
<td>98</td>
<td>96 – 108</td>
</tr>
<tr>
<td>HCO₃⁻ (mmol/L)</td>
<td>30</td>
<td>24 – 30</td>
</tr>
<tr>
<td>PO₄²⁻ (mmol/L)</td>
<td>1.1</td>
<td>0.8 – 1.4</td>
</tr>
<tr>
<td>Betahydroxybutyrate (mmol/L)</td>
<td>3.2</td>
<td>1.3 – 5.5</td>
</tr>
</tbody>
</table>

Urinalysis showed glucose ++++, ketone +, protein ++, urine culture was negative. Wound swab culture yielded mixed growth of Staphylococcus aureus and coliform organisms sensitive to a number of locally available antibiotics.

The medical team that admitted Mr. Zo started him on a broad-spectrum antibiotics before switching to the sensitive antibiotics, one litre of normal saline daily for 72 hours, antipyretic agents, wound debridement and dressing as well as T.D.S soluble insulin. Endocrinology review was requested by the managing medical unit within the second week of admission on account of difficulty with control of blood glucose. His clinical condition has also become worse than at presentation.

II. Discussion

The main findings in these case reports include failures of the medical units that admitted the patients to make the appropriate diagnosis of DKA and HHS respectively, not recognizing the precipitating factors/co-morbid conditions for what they were and not involving the endocrinologists early in the management of the patients. Blood glucose control, per se, was their main reason for endocrinologist invitation.

The first case had DKA precipitated or complicated by CVA (stroke) and was managed with oral anti-diabetic drugs. The managing medical unit was preoccupied with treating CVA (stroke) and did not involve the endocrinologists early. Failure to utilize soluble insulin to correct hyperglycaemia in this patient was a critical omission. Electrolyte disorders were also, not addressed. It is important to note that hyperglycaemia is a well-known poor prognostic factor in cases of stroke where it independently contributes to stroke outcome by inducing secondary brain damage.

The second case, 63-year old new onset diabetes, presented with HHS precipitated by bacterial infections of a foot ulcer. Diagnosis of HHS, an acute potentially fatal acute complication of DM was not captured by the managing medical unit and the patient was, therefore, at no time treated for such. The electrolyte and fluid deficits characteristic of HHS were not corrected in the index patient and the endocrinologist’s involvement in the management of the patient was delayed till the second week of admission.

It is now recommended that the diabetes specialist team must always be involved in the care of patients admitted to hospital with DKA or HHS as their involvement shortens patient’s hospital stay and improves safety.

III. Conclusion//Recommendations

Diagnosis of hyperglycaemic emergencies was not correctly made in the index cases and the patients were not treated as it should be with the local treatment protocol of the facility. The diabetes care team was not involved early in the management of the two cases of HHS. It is recommended that the diabetes care team should not be involved just for blood glucose control only. Outcome of treatment of the two cases may have been different if the needful was done on each occasion.

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