CASE STUDY ON PAEDIATRIC TYPE-1 DIABETES MELLITUS WITH DIABETIC KETOACIDOSIS

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Introduction

Type 1 diabetes mellitus (T1DM) is on increasing with a trend of 3–5% increase/year. India has three new cases of Type-1 Diabetes Mellitus /100,000 children of 0–14 years. Type-1 Diabetes is also known as juvenile diabetes. It is a condition in which the body is unable to make sufficient insulin. Juvenile diabetes is an autoimmune disorder. The cells that make the insulin in the pancreas are destroyed by the immune system in the body. The insulin is a hormone which helps the glucose in the blood to enter into the cells which can be used as a fuel to the body. When this glucose is unable to enter the cells, this builds up in the body which causes rise of glucose in the blood also called as hyperglycaemia. This affects all organs in the body eyes, heart, kidneys, nerves etc. It is a chronic condition and can start at any age. The insulin is not producing by pancreas so it must be replaced with insulin injection or insulin pumps. Thus, type 1 diabetes is also called insulin-dependent Diabetes Mellitus. It requires lifelong treatment. The management of diabetes for children should not be extrapolated from adult diabetic care. This is diagnosed by the fasting blood glucose, random blood glucose and also oral glucose tolerance test. Glycated haemoglobin (HbA1c) is also a tool to diagnose. The children with the Type-1 Diabetes commonly present with polydipsia, polyuria and weight loss and approximately a 3rd % with diabetic ketoacidosis. Most people around 90% who are newly diagnosed with type 1 diabetes have antibodies against specific beta cell proteins, insulin, glutamate decarboxylase, islet antigen-2, zinc transporter -8 etc. The transplantation of islets or the clinical pancreas has been considered a feasible treatment option for the patients with T1DM with poor glycaemic control. However, the severe shortage of pancreas and islets derived from human organ donors and the complications that have been associated with transplantations, high cost, and limited availability of procedures remain as limitations in the widespread application of these strategies. Stem cell therapy has a great potential for curing patients with
T1DM. With the advent on stem cell therapy research for various diseases, breakthroughs in stem cell-based therapy for T1DM have been reported.

Case presentation

A 5-year male child came to the hospital on 6/2/21 with the chief complaints of vomiting from last night and abdominal pain from the morning. He had fever 2 days back, low back, low onset, and relieved on medication. Child had 2 episodes of vomiting last night containing food particles, non-blood-stained. Associated with abdominal pain all over the abdomen, polyuria, and polydipsia.

Past history

The patient was diagnosed with type-1 diabetes at the age of 1 year 8 months and at that time he was hospitalised with increased work of breathing and on investigations diagnosed with diabetic keto acidosis and he was on Mixtard Insulin. They have been using 6IU of insulin from then and not titrated the dose according to the age and weight till now. Child is on irregular GRBS (General Random Blood Sugar) monitoring and then gave 1 IU extra dose of Mixtard if GRBS is high.

Antenatal history, Natal history, Post-natal history and Developmental history

No abnormalities.
Immunisation history

Immunised completely till now.

Family history

No history of similar illness in family and siblings.

Anthropometry

Weight: 14.8 kg
Height: 98 cm
BMI: 15.4

Vitals

Temperature: Afebrile
Pulse rate: 98 bpm
Respiratory rate: 28 cpm
Blood pressure: 95/70 mm Hg
SPO2: 100% at RA
GRBS: 410 mg/dL
Pulse volume: low
CRT < 3 seconds

General examination

Child is alert and active. No signs of dehydration, pallor, icterus, clubbing, cyanosis, lymphadenopathy and oedema.
Lab investigations

Complete urine examination:
sugar:3 +            ketones:1+

Arterial blood gas test

<table>
<thead>
<tr>
<th>Test</th>
<th>observed value</th>
<th>normal value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBC (Standard Bicarbonate)</td>
<td>17.97 mmol/L</td>
<td>21-27 mmol/L</td>
</tr>
<tr>
<td>PH</td>
<td>7.209</td>
<td>7.35-7.45</td>
</tr>
<tr>
<td>PCO2</td>
<td>46.74 mmHg</td>
<td>35-45 mmHg</td>
</tr>
<tr>
<td>PO2</td>
<td>114.0 mmHg</td>
<td>80-100 mmHg</td>
</tr>
<tr>
<td>TCO2 (Total CO2)</td>
<td>20.23 mEq/L</td>
<td>23-29 mEq/L</td>
</tr>
<tr>
<td>HCO3</td>
<td>18.83 mEq/L</td>
<td>22-28 mEq/L</td>
</tr>
</tbody>
</table>

Liver function test

<table>
<thead>
<tr>
<th>Test</th>
<th>value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspartate aminotransferase</td>
<td>62 U/L</td>
</tr>
<tr>
<td>Alanine transaminase</td>
<td>67 U/L</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>152 U/L</td>
</tr>
</tbody>
</table>

Electrolytes

Sodium – 134 mEq/L
Potassium-3.6 mEq/L
Chloride-105 mmol/L
Calcium-8.3 mg/dL

Renal function test-normal

Complete blood picture - Normal

Diagnosis: type -1 diabetes mellitus with mild diabetic ketoacidosis

Day 1

Treatment:

- Inj.regular insulin 11 U/day (0.7u/kg/day) 3U-3U-3U-2U(7AM-1PM-7PM-1AM)
- Iv fluids NS @50ml/hour+5 ml KCl at 500 ml
- Inj.ceftriaxone 375 mg iv twice daily.
Day 2

GRBS

9:30 am 186 mg/dl

1:00 pm 386 mg/dl

Treatment:

- Inj.regular insulin 11 U/day (0.7u/kg/day) 3U-3U-3U-2U(7AM-1PM-7PM-1AM)
- Ivf ns @68 ml/hr
- Inj.ceftriaxone400 mg IV BD 6 AM/6PM

Day 3

Complete urine examination (CUE): sugar-4+

GRBS:

<table>
<thead>
<tr>
<th>Time</th>
<th>Pulse rate (bpm)</th>
<th>Respiratory rate (cpm)</th>
<th>Spo2 (%)</th>
<th>Blood pressure (mmHg)</th>
<th>GRBS (mg/dL)</th>
<th>IVF (normal saline)</th>
<th>Insulin infusion (0.5 kg/hr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4PM</td>
<td>100</td>
<td>25</td>
<td>98</td>
<td>94/56</td>
<td>131</td>
<td>NS</td>
<td>0.75ml/hr</td>
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<tr>
<td>5PM</td>
<td>90</td>
<td>28</td>
<td>99</td>
<td>89/50</td>
<td>129</td>
<td>NS</td>
<td>0.75ml/hr</td>
</tr>
<tr>
<td>6PM</td>
<td>92</td>
<td>24</td>
<td>99</td>
<td>90/50</td>
<td>79</td>
<td>NS</td>
<td>0.75ml/hr</td>
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<tr>
<td>7PM</td>
<td>94</td>
<td>26</td>
<td>99</td>
<td>92/52</td>
<td>128</td>
<td>NS</td>
<td>0.75ml/hr</td>
</tr>
<tr>
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<td>96</td>
<td>24</td>
<td>98</td>
<td>90/50</td>
<td>149</td>
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<tr>
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<td>95</td>
<td>24</td>
<td>98</td>
<td>90/50</td>
<td>149</td>
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<td>99</td>
<td>94/50</td>
<td>140</td>
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<tr>
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<td>96</td>
<td>26</td>
<td>98</td>
<td>92/52</td>
<td>168</td>
<td>NS</td>
<td>0.75ml/hr</td>
</tr>
<tr>
<td>12AM</td>
<td>94</td>
<td>24</td>
<td>97</td>
<td>96/66</td>
<td>163</td>
<td>NS</td>
<td>0.75ml/hr</td>
</tr>
<tr>
<td>1AM</td>
<td>93</td>
<td>27</td>
<td>99</td>
<td>94/50</td>
<td>147</td>
<td>NS</td>
<td>0.75ml/hr</td>
</tr>
<tr>
<td>2AM</td>
<td>96</td>
<td>26</td>
<td>98</td>
<td>96/40</td>
<td>162</td>
<td>NS</td>
<td>0.75ml/hr</td>
</tr>
<tr>
<td>3AM</td>
<td>94</td>
<td>26</td>
<td>99</td>
<td>97/40</td>
<td>121</td>
<td>NS</td>
<td>0.75ml/hr</td>
</tr>
<tr>
<td>4AM</td>
<td>96</td>
<td>26</td>
<td>98</td>
<td>96/40</td>
<td>78</td>
<td>NS</td>
<td>0.75ml/hr</td>
</tr>
</tbody>
</table>

Plan to convert to regular insulin SC

Day 4:

- U/O:500ml, highly coloured
- GRBS:259 mg/dL
- Inj. Regular insulin 11u /day 3U-3U-3U-2U (7AM-1PM-7PM-1 AM
Day 5:

- Occasional wheeze present
- **GRBS:** 288 mg/dL (10:15 am)

**Treatment:**

- SYP.SALBUTAMOL-5 ml PO TID (0.2 mg/Kg /Day)

**DAY 6:**

CST (continue same treatment)

**Day: 7**

**Treatment:**

- Inj:mixtard insulin(0.8 U kg/day) 7U-0-5U(7AM-0-7PM)
- Syp: salbutamol 5ML PO TID(0.2 MG/KG/DAY)

If GRBS increases above 250 add 1 IU of regular insulin for every increase in 100 mg/Dl

**Day-8**

- Weight: 13.2 kg
- Temperature: 101.8 f
- Treatment: CST with SYP.PCM 9ml PO SOS

**Day- 9**

- Target blood glucose range: 80-150
- Mixtard insulin:8u -0 -6U
- Retina dilated, referred to ophthalmologist – B/L Anterior segment NORMAL, B/L Pupils- Non-Sensitive and Reactive to Light.
- CST

**Day-10**

- 2 loose stools
- Inj.mixtard insulin-8u-0-5u
- Syp.zinc 5ML PO OD
Type-1 dm, mild diabetic ketoacidosis(recovered)

Discussion:

A 5-year male child came with the complaint of vomitings and abdominal pain. He was diagnosed with type-1 diabetes with mild diabetic ketoacidosis. The patient was diagnosed with type-1 diabetes at the age of 1 year 8 months and he was on insulin mixtard 6 IU and not titrated the dose according to weight and age till now. Andalso, on irregular GRBS monitoring, frequent GRBS monitoring is done and lab investigations like arterial blood gases, liver and renal function tests, electrolytes and haematological tests were done. IV fluids and insulin were given to the patient. He was recovered from ketoacidosis while discharge. The cause of the diabetes in this case is unknown might be due to the genetical changes. Lifelong treatment is frequent GRBS monitoring is required.

Conclusion:

The fundamental treatment for T1D is insulin, either by infusions or through an insulin pump. Individuals with T1D need to figure out how to compute their sugar consumption and how to check their glucose level. They additionally need to figure out how to compute the insulin portion they need each time dependent on their glucose level and starch consumption. Commonly, individuals with T1D check their glucose level five to seven times each day and give insulin infusions four to seven times each day, depending upon glucose levels and recurrence of eating. Patients need to follow their endocrinologist at regular intervals and work intimately with their diabetes group (medical caretaker, dietitian and social specialist). Right now, there are no known treatments that can change the immune framework, and prevent destruction of beta cells, and thus prevent T1D.

Stem cell-based treatment has been viewed as a promising helpful strategy for diabetes treatment, particularly for T1DM. As referenced, significant advances in research have improved our opportunity of restoring glucose-responsive insulin discharge in patients with T1DM. However, some of the clinical trials in stem cell therapy have been dissatisfactory, and numerous inquiries and specialized obstacles actually should be addressed. The major issues are the following aspects:

- How to develop more functional and mature β- cells in vitro from hPSCs (Human pluripotent stem cells).
- How to improve the efficiency of differentiation of IPCs (Insulin-producing cells) from hPSCs.
- How to protect the implanted IPCs from the attack of immune system.
- How to create adequate quantities of desired cell types for clinical transplantation and
- How can insulin independence be established?

In spite of these limitations, the utilization of stem cell treatment for T1DM addresses the most advanced approach to cure Type 1 diabetes.

References:

- https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6661119/#!po=12.9121
- https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4413384/