PedsCases Podcast Scripts

This is a text version of a podcast from Pedscases.com on "Type 1 Diabetes- Part 1." These podcasts are designed to give medical students an overview of key topics in pediatrics. The audio versions are accessible on iTunes or at www.pedcases.com/podcasts.

Type 1 Diabetes- Part 1:

Developed by Alkarim Velji and Dr. Rose Girgis for PedsCases.com.
January 3, 2013.

Introduction

Hi everyone. My name is Alkarim Velji. I'm a medical student at the University of Alberta. This podcast was developed in conjunction with Dr. Rose Girgis, a pediatric endocrinologist at the University of Alberta.

You're on your final week of a rotation in the emergency department. A 2 year old female comes in an altered level of consciousness. She is also tachycardic, and dehydrated. You notice that she is wearing a medic alert bracelet stating that she has T1DM. What would be the first few things you'd like to know about her?

This podcast is the first part of a two part series on type-one diabetes. The objectives of this podcast are to briefly review the basic pathophysiology of diabetes and elaborate on the management of some of the major acute complications.

Let's start with some pathphysiology:

TIDM is likely caused by an immune mediated destruction of pancreatic beta cells. The loss of pancreatic beta cells results in decreased insulin production. Generally patients do not exhibit signs of diabetes until about 80% of the cells have lost their function. At this point, glucose handling becomes affected. This destruction is triggered by environmental factors in genetically predisposed individuals.

The HLA DR4-DQ8 and HLA DR3-DQ2 on chromosome 6 both confer high risk for developing T1DM. However, T1DM is not caused solely by genetic factors. Fathers with T1DM pass it on only about 5-6% of the time and mothers with T1DM pass it on about 2-3% of the time. Siblings have a 5% of chance of developing it and monozygotic twins have only about a 25-50% chance of developing TIDM if their twin has it.
When discussing TIDM in pediatrics, both the parents and patient should be reminded that they are not alone and this not their fault.

Patients with TIDM are at higher likelihood of developing other autoimmune conditions. Clinical autoimmune thyroid disease (AITD) occurs in 1*S* to 30% of individuals with type 1 diabetes. Celiac disease can be identified in 4-90% of children with type 1 diabetes,

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Addison's disease can occur but it is rare.

The three cardinal symptoms of TIDM are weight loss, polydipsia, and polyuria. What causes these symptoms?

Remember, Insulin is an anabolic hormone that encourages glucose uptake into cells and glycogen synthesis. It also inhibits lipolysis. Therefore, without insulin, very little glycogen is made and lipolysis will continue with fewer checks and balances. In other words, no insulin means no storage of energy in the liver and an increased breakdown of fat. Overall, this causes uncontrolled weight loss.

Acute Complications

Now that we have a handle on the basic pathophysiology of diabetes, let's get back to our patient. She comes into the Emergency Department with an altered level of consciousness and dehydrated. You also note she is tachycardic. Knowing that she has T1DM, what two diagnoses should be at the top of your differential?

Diabetic ketoacidosis and hypoglycemia. So how would we tell them apart?

After establishing her airways, breathing, and circulation, the most important STEP is to assess her blood glucose.

In this case, her blood sugar is high at 1*S* mmol/L. This is clearly high so she is hyperglycemic. The next step then is to assess her blood gases and her ketones. Her blood pH comes back as acidic and she also tests positive for ketones. So, DKA becomes our focus.

In the case of DKA, patients are dehydrated from excessive urination caused by the hyperglycemia. This dehydration results in decreased perfusion of the muscles and further reducing glucose uptake. This yields worsening hyperglycemia.

As well, remember, insulin inhibits lipolysis. Without that inhibition, lipolysis continues and an excess amount of fatty acids are produced. The liver is overwhelmed and the fatty acids are converted into ketones. These ketones are the cause of the acidosis. That produces feelings of nausea that can induce vomiting that will further exacerbate the dehydration.

So, how can we support our patient?

The immediate strategy is to start her on fluids and insulin. The fluids help manage the dehydration and encourage glucose uptake by increasing perfusion. The insulin is essential for inhibiting lipolysis and thus limiting ketone production.

A&!: administering initial fluids and insulin, you continue to monitor the patient's labs and you notice that she is becoming hypokalemic. Your preceptor asks you why? What do you think?

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The answer is actually rather simple: insulin activates a sodium-potassium transporter that pushes potassium into cells, thereby reducing potassium concentration in the blood. Therefore, with insulin administration, potassium needs to be monitored and treated as necessary.

There are three main reasons why patients present with DKA:

- because they are a new diagnosis,
- have T1DM but are suffering from a severe infection or
- they are not taking their insulin. Frequently, teenagers will choose not to take their insulin to stimulate weight loss.

Overall, you should remember the following things:

1. Patients are not alone in their diabetes. It's not their fault they have TIDM. However, they need to be aware of the long-term complications of retinopathy, neuropathy, and nephropathy as well as the acute complications of DKA and hypoglycemia.

2. When treating a patient with DKA, start with your ABCs – airways, breathing, and circulation. Then, administer fluids to help with the hyperglycemia and insulin to help with the acidosis. Finally, make sure you monitor potassium as insulin can induce hypokalemia.

Join us for the second part on our diabetes series

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