A rare reason of hyperinsulinism: Münchausen Syndrome by Proxy

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Objectives:

Hyperinsulinemic hypoglycemia results from excess insulin secretion and it is diagnosed by both clinical history and laboratory findings. Laboratory findings include fasting and postprandial hypoglycemia with hyperinsulinemia and elevated C-peptide levels. The causes of hyperinsulinism can be congenital or acquired. Hypoglycemia that occurs later in childhood is most often attributable to pancreatic insulinoma. Toxication of drugs can rarely be reason for hyperinsulinism. Toxication due to hyperinsulinism may occur accidentally or deliberately.

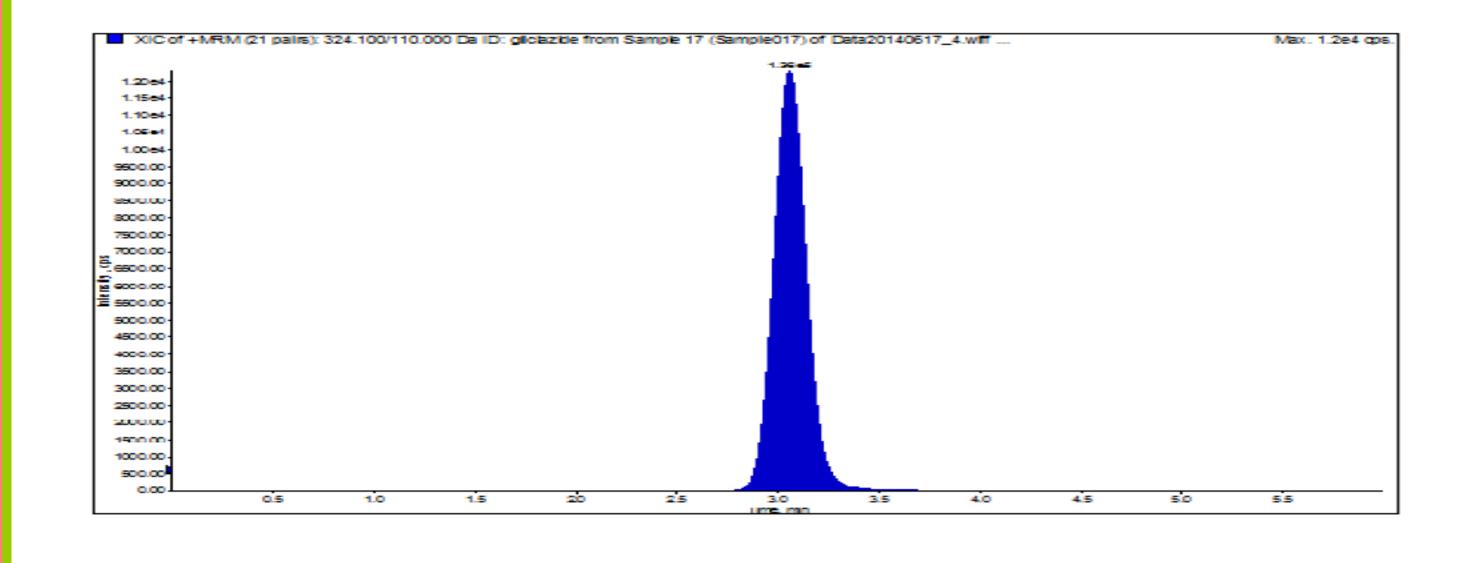
Herein, we report a case who presented with factitious hyperinsulinemic hypoglycemia and diagnosed with Munchausen syndrome by Proxy (MSBP) which is an uncommon condition of child abuse.

Methods:

A 7-year-old girl was referred to our department due to hyperglycemic and hypoglycemic episodes. Our patient was born as 2700 g. at 40 weeks gestation and delivery was uneventful. It was learned that syncope or seizures during hypoglycemic periods started 3 years ago and she was admitted to a pediatric department. Fasting test was performed but hypoglycemia didn't occur during the test. Full metabolic screen also revealed no abnormalities. When she was admitted to our hospital her glycemic levels were high according to glycemia records of the family.

Blood glucose monitoring showed hyperglycemia reaching 300 mg/dl and no hypoglycemia. We didn't detect any other clinical or laboratory findings so that gliclazide (1 mg/kg/day) was initiated and increased to 2 mg/kg/day because of persisting hyperglycemia. However, episodes of hypoglycemia occurred during the patient follow up, thus gliclazide treatment was stopped.

Few weeks later she was admitted to the emergency room with hypoglycemic seizure. Plasma levels of insulin (156 mIU/mI) and C peptide (6 ng/dI) were discordant with the concurrent blood glucose level (33 mg/dI) and urinary ketones were negative, suggesting endogenous hyperinsulinism. Diazoxide and dextrose (10%) were administered and hypoglycemia was prevented. Plasma ammonia and lactate levels along with cortisol and growth hormone levels were within normal limits. Computed tomography and MRI of the abdomen was normal. Severe hypoglycemia persisted in spite of octreotide, diazoxide and glucose infusions up to 20 mg/kg/min. So she was consulted to pediatric surgery department as to pancreatectomy. However, the mother of the patient was looking good discordant with the situation. Due to suspicion of Münchausen by Proxy, blood and urine samples were sent to toxicology laboratory in order to investigate hypoglicemic agents. Toxic levels of gliclazide was detected both in urine and blood samples. Patient was immediately isolated despite unwillingness of mother. All medications were ceased except glucose infusion and hypoglicemia disappeared on the second day of isolation. Glucose infusion was decreased gradually. Daily toxicological analysis of blood and urine samples were performed and gradual decrease in glicalizide levels were observed. Finally on the 4th day of isolation gliclazide was not detected in urine and blood samples. The patient was diagnosed as Münchausen by Proxy.



Gliclazide peak on chromotogram

Conclusions:

Both diagnosis and treatment of factitious hyperinsulinism due to sulfonylureas are difficult. Parents may seem normal and interested in their children. The first important step for diagnosis is suspicion. But when you suspect that the parents may cause the illness, they may modify their methods of inducing illness or they may change their hospitals. Therefore, especially in patients who were evaluated by more than one hospital and have discordant test results, Munchausen syndrome by Proxy should be considered immediately. The second important step is detection of suspicious agent in urine and blood samples are important for diagnosis. After diagnosis the most important part is protecting of children. The case be immediately reported to child care institutes.

