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The Use of Glucocorticoids for Better Control of Diabetes Mellitus: The Paradox of Sclerosing Mesenteritis (The Rare Could Become Common)

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Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient: Male, 57-year-old
Final Diagnosis: Sclerosing mesenteritis
Symptoms: Abdominal pain
Medication: Prednisone
Clinical Procedure: CT scan • MRI
Specialty: Endocrinology and Metabolic

Objective: Rare disease

Background: Sclerosing mesenteritis is a rare disease characterized by chronic inflammation of mesenteric adipose tissue. To our knowledge, this is the first case report that presents the effects of glucocorticoid therapy on metabolic control in diabetes mellitus, aggravated by sclerosing mesenteritis. We want to show the significance of this rare disease, which could be underestimated as a cause of decompensation of diabetes mellitus.

Case Report: A 57-year-old man with diabetes type 2 was admitted to the hospital to obtain better metabolic control of this disease. In addition, he reported persistent pain in the left side of his abdomen.
Sclerosing mesenteritis was diagnosed based on the CT and MRI images. Prednisone was administered. The treatment resulted in better glycemic control and abdominal pain reduction. On follow-up after 1 year, the patient reported a decrease in the abdominal pain and an MRI showed a significant reduction of abnormalities in the mesentery.

Conclusions: It is known that glucocorticoids exacerbate hyperglycemia, particularly in patients with diabetes mellitus. However, we noticed contrary effects in the case of our patient. We suggest that the inflammatory process occurring in sclerosing mesenteritis was one of the main causes of metabolic decompensation in our patient. The effect of reduction of inflammation with glucocorticoids was stronger than the hyperglycemic effect of this treatment. That is why, in the presence of this autoimmune disease, the use of glucocorticoids can paradoxically lead to better glycemic control.

Keywords: Diabetes Mellitus • Glucocorticoids • Hyperglycemia • Panniculitis, Peritoneal • Prednisone

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/930453

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Background

Sclerosing mesenteritis (SM), also referred to as retractile mesenteritis, is a rare disease characterized by chronic inflammation of mesenteric adipose tissue. Due to unspecific symptomatology, the disease is hard to diagnose. The clinical presentation includes abdominal pain, fever, weight loss, diarrhea, and vomiting. In the physical examination, the possible symptoms are abdominal tenderness, abdominal mass, and distended abdomen [1,2].

Due to the benign character of the disease, biopsy is usually not recommended. Imaging tests and clinical presentation are enough to establish the diagnosis. In the majority of cases, sclerosing mesenteritis is a self-limited disease, which is why asymptomatic cases are left untreated. In patients that present symptoms, therapeutic options include immunosuppressive agents and surgery [3,4].

To our knowledge, this is the first case report that presents the effects of glucocorticoid therapy on metabolic control in diabetes mellitus, aggravated by sclerosing mesenteritis, and finally compensated by their implementation.

Case Report

A 57-year-old man with type 2 diabetes and hypertension was admitted to the hospital to obtain better metabolic control of those diseases. He also suffered from benign prostatic hyperplasia, hyperlipidemia, and had a 35-pack-year smoking history. His weight on admission was 93 kg (BMI=26.6kg/m^2). He was treated with metformin (3×1000 mg p.o.) and multiple daily injections with human regular insulin before main meals and NPH insulin at bedtime (intermediate-acting insulin) (total mean daily dose of insulin was 51 IU SC). After self-monitoring, he presented with high blood glucose levels (16.65-26.6 mmol/l) and elevated blood pressure. He also reported polydipsia, polyuria, weight loss (16 kg in 3 months). In addition, he presented with persistent pain in the left side of his abdomen, which was aggravated while moving and on palpation. A physical examination revealed no pathological signs in the abdomen.

Blood tests showed a high glycated hemoglobin level (12%, 108 mmol/mol) and leukocytosis (13.57×10^3/µl) without an increase of other inflammatory markers (C-reactive protein, erythrocyte sedimentation rate). An abdominal ultrasound revealed small renal cysts in the left kidney.

As an initial treatment, a continuous intravenous insulin infusion was administered. On the third day of hospitalization, we replaced intravenous insulin infusion with multiple daily injections of insulin. Despite the use of intravenous insulin infusion, the glycemic control still remained unsatisfactory. Due to this fact and the persistence of the abdominal pain, computed tomography (CT) was performed.

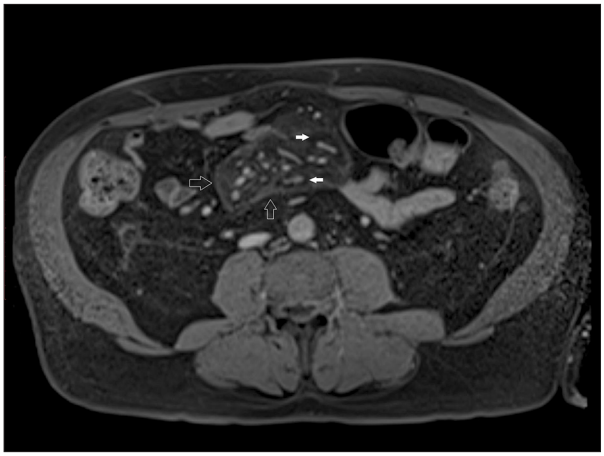

Figure 1. Contrast-enhanced T1 GRE fat-saturated image shows a well-demarcated mass-like lesion in the mesentery with the presence of a capsule (open arrows) and spared fat around traversing vessels (white arrows). The image strongly suggests sclerosing mesenteritis.

CT showed an atypical region of increased attenuation in mesenteric adipose tissue. It also revealed renal cysts in both kidneys; however, they were excluded as the cause of abdominal pain. Colonoscopy and gastroscopy did not detect any abnormalities.

Abdominal contrast-enhanced magnetic resonance imaging (MRI) was ordered, and it confirmed abnormalities in the mesentery (**Figure 1**). Sclerosing mesenteritis was diagnosed on the basis of CT and MRI images. As the patient presented constant abdominal pain, prednisone in a daily dose of 40 mg was administered. The treatment resulted in reduction of abdominal pain and better glycemic control, which was indicated by a lower insulin requirement (total mean daily dose of insulin was 32 IU SC).

On follow-up, after 2 months of glucocorticoid therapy, the patient reported a decrease in the abdominal pain and an MRI showed a significant reduction of the abnormalities in the mesentery. The glycated hemoglobin level decreased to 8.8% (73 mmol/mol). We were also able to gradually lower the daily dose of prednisone to 5 mg per day. One year later, an MRI revealed a further regression of the lesions in the mesentery (**Figure 2**). Mean glucose levels were within the range of 5-8.33 mmol/L, periodically to approximately 10 mmol/L.

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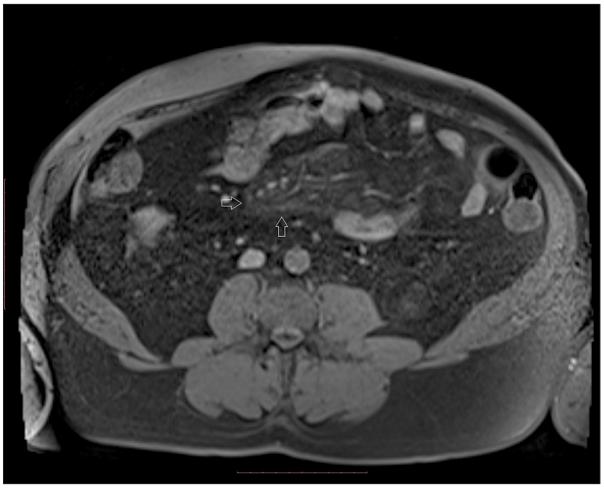

Figure 2. Control MRI study after treatment shows partial regression of the lesion, which is smaller and less demarcated (open arrows).

Discussion

We present a case of accidental diagnosis of sclerosing mesenteritis during investigation of the causes of metabolic decompensation and abdominal pain. However, the connection between SM and diabetes mellitus was suggested before in a series of observational studies. In the literature, the prevalence of diabetes mellitus in patients with SM ranges from 8.7 to 21.5%. Diabetes mellitus was described as a "possible causative and/or associated factor" of sclerosing mesenteritis [5,6].

It was suggested that the inflammation of mesenteric adipose tissue could be a possible etiological factor in the development of insulin resistance. This process is similar to the one that occurs in the development of diabetes mellitus. Dying adipocytes causes inflow of macrophages to adipose tissue and synthesis of proinflammatory cytokines (IL1, IL-6, TNFα), finally leading to increase of insulin resistance via activation of intracellular pathways such as NFκB and JNK. In the SM, the inflammation process is even more intensified and chronic, which presumably makes the development of insulin resistance and diabetes mellitus even easier [7,8]. In connection with the above, it seems that the inflammatory process occurring in the progress of SM was one of the main causes of metabolic decompensation in our patient, which presented as high blood glucose levels and an increased insulin requirement.

Because sclerosing mesenteritis is an inflammatory disease of unknown origin, treatment is based on immunosuppressive agents, including glucocorticoids [3,4].

It is known that glucocorticoids exacerbate hyperglycemia in patients with diabetes mellitus, but they can also induce diabetes mellitus themselves [9,10]. The diabetogenic effect of glucocorticoids is based on many mechanisms, among them: counter-regulatory effect in relation to insulin, increase of insulin resistance and hepatic gluconeogenesis, and glycogenolysis. Furthermore, they decrease the secretion of insulin from beta cells and activate alpha cells in the pancreas and diminish the effects of incretin. The final effect of GC-therapy depends on the dose, the method of administration, and the active ingredient used in formulation of the drug [11]. The data show that oral administration of glucocorticoids is associated with an increased risk of developing diabetes mellitus of up to 2%, while the odds ratio for new-onset diabetes mellitus in patients treated with glucocorticoids ranges from approximately 1.5 to 2.5 [12,13].

Taking into consideration the dose of glucocorticoids used in our patient and the inflammation of his mesentery, we predicted the possibility of significant hyperglycemia. However, in the presented case, we observed the opposite effects of improved metabolic control during treatment with glucocorticoids. It seems that the effect of the reduction of the inflammation with glucocorticoids was stronger than the hyperglycemic effect of this treatment.

We did not find other case reports that document improvement in diabetes mellitus type 2 or insulin resistance by glucocorticoid administration. However, because of the importance of inflammation in the development of insulin resistance, it is questioned whether type 2 diabetes mellitus can be treated as an autoimmune disease. Glucocorticoids are taken into consideration as a one of the possible agents in this novel immunotherapeutic approach. The proposed mechanism via which glucocorticoids inhibit development of insulin resistance is the ability to prevent NF-κB subunits from binding with target genes, and thus inhibit the transcription of DNA. As seen above, the similarity of the inflammatory process of the adipose tissue in the sclerosing mesenteritis suggests that steroids can have analogous action in this disease [14,15].

Conclusions

We should include SM in the differential diagnosis of abdominal pain in patients with diabetes mellitus. In addition, we should think about this disease when we are looking for causes of metabolic decompensation in patients with diabetes mellitus.

In most cases, the use of glucocorticoids causes a significant increase in blood glucose levels, but in the patients with SM, treatment with glucocorticoids can paradoxically lead to better glycemic control.

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