Diabetes Update

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Dear Doctor,

Greetings from Micro Labs Limited. We are happy to bring you "DIABETES UPDATE" which contains latest and interesting news in the field of Diabetes and Endocrinology.

- **Effects of a Ketogenic Diet on organ-specific Insulin Sensitivity Among Individuals with Obesity**
- **Impact of delayed medical treatment on the development of Diabetic foot in type 2 diabetic patients**
- **Efficacy of Patient-Centered Self-Management Intervention on Glycemic Control, Self-Efficacy, and Self-Care Practices in Type 2 Diabetes Mellitus**
- **Case report on Diabetic Myonecrosis in a Young Woman with Long-Term Type 1 Diabetes Mellitus**

Should you require any specific article or medical information, please feel free to contact us at medicalservices@microlabs.in

Looking forward to your valuable feedback

Best Regards,

Medical Team, Micro Labs Limited

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Effects of a Ketogenic Diet on organ-specific Insulin Sensitivity Among Individuals with Obesity

INTRODUCTION

OBESITY AND DIABETES

People with obesity who follow a 3-week ketogenic diet had increased skeletal muscle insulin sensitivity.

The ketogenic diet (KD), which limits carbohydrate intake, has been used for many years to manage drug-resistant epilepsy in children.[1] Growing evidence suggests that ketone bodies offer various health benefits, including enhancements in both heart function and metabolic health. The KD promotes weight loss, improves glycemic control, and decreases the need for glucose-lowering medications in people with type 2 diabetes. While the exact mechanism by which KD improves glucose metabolism is still unclear, the observed reduction in blood glucose suggests a possible improvement in insulin sensitivity (IS). This is somewhat surprising that KD was high in fat, which could potentially lead to excess lipid buildup and insulin resistance (IR). Since most studies on KD have focused primarily on glucose levels, it remains uncertain whether the glucose-lowering effect is due to enhanced glucose uptake in skeletal muscle or greater insulin-mediated suppression of endogenous glucose production (EGP).[2] So, this study aimed to examine the impact of the KD on organ-specific IS in individuals with obesity and IR, focusing on skeletal muscle, liver, and adipose tissue. The researchers hypothesized that KD would enhance insulin-stimulated glucose uptake, indicating improved IS in skeletal muscle.

METHODS

This was a randomized controlled crossover study conducted with two phases, separated by a one-week washout period, and included two 3-week interventions: 1) a KD and 2) a standard diet (SDD). Each diet phase ended with a metabolic assessment day. A total of 11 individuals with obesity participated, meeting the inclusion criteria of being 50–70 years old with a BMI between 28–40 kg/m². Participants were randomly assigned in a 1:1 ratio to start with either the KD or SDD. Insulin sensitivity in skeletal muscle was measured by the increase in glucose

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disposal during a hyperinsulinemic-euglycemic clamp (HEC), while hepatic and adipose tissue insulin sensitivity was assessed by the relative suppression of endogenous glucose production (EGP) and palmitate flux during the HEC.

RESULTS

The KD led to a reduction in triglyceride levels, though no significant differences were noted in other biomarkers. After three weeks on the KD, participants experienced a decrease in body weight (2.2 kg), fat mass (1.1 kg), and lean mass (1.4 kg), all statistically significant (P<0.05) compared to the SDD. Plasma levels of β-hydroxybutyrate (OHB) were higher during the baseline period on the KD but returned to comparable levels during the HEC following both diets (intervention: P<0.001; time: P<0.001; interaction: P<0.001). To evaluate skeletal muscle insulin sensitivity, the

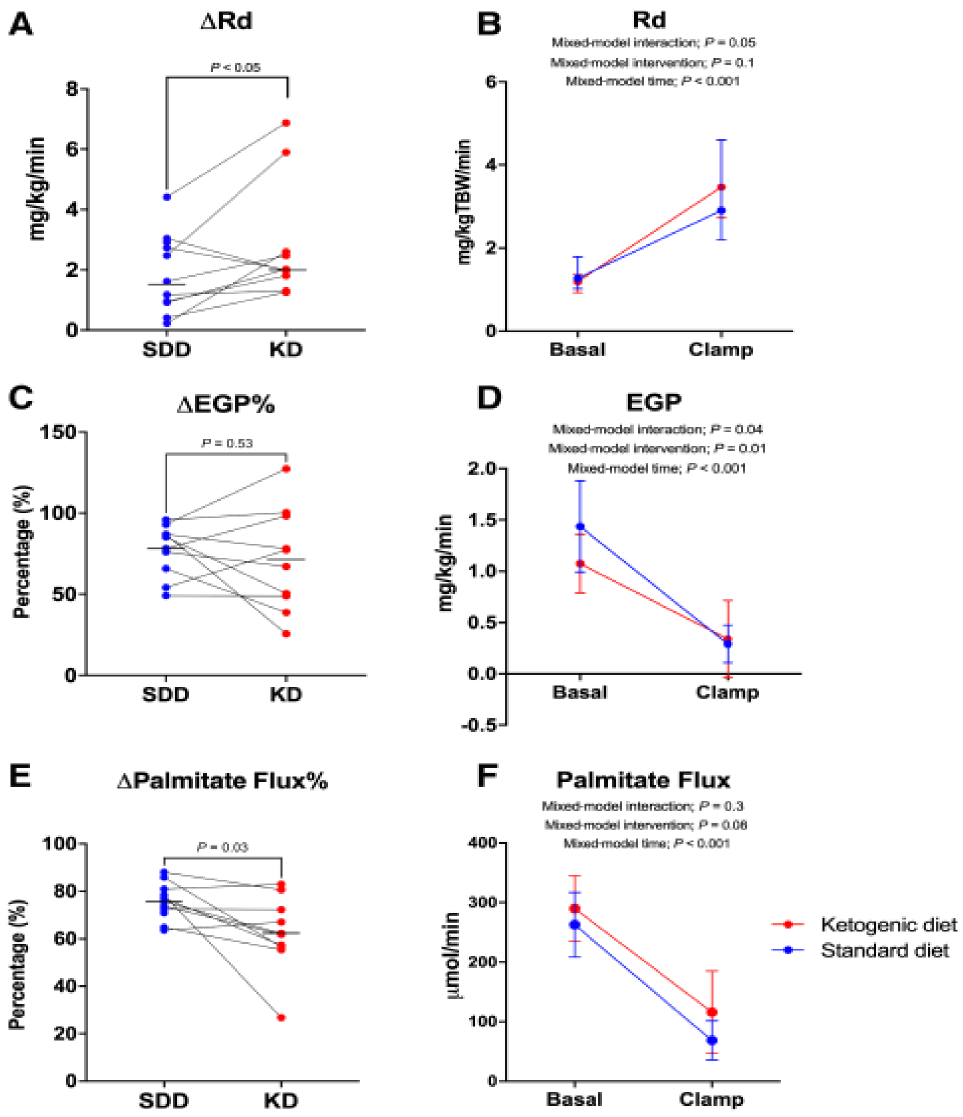

Figure 1 (A-F). Insulin sensitivity measurements for individual organs.

increase in glucose disposal rate (R_d) during the HEC (ΔR_d) was measured, showing a greater increase after the KD compared to the SDD (2.0 [1.7–3.4] vs. 1.6 [0.9–2.9] mg/kg/min; $P<0.05$) (Figure 1A). While glucose R_d was similar during the baseline for both diets, the increase during insulin infusion was more pronounced after the KD (intervention: P=0.1; time: $P<0.001$; interaction: P=0.05) (Figure 1B). For hepatic insulin sensitivity, the relative suppression of endogenous glucose production (EGP) during insulin infusion (ΔEGP%) was similar between the KD and SDD (71 ± 31% vs. 78 ± 15%; P=0.53) (Figure 1C). However, EGP was lower during the baseline period after the KD (1.1 ± 0.3 vs. 1.4 ± 0.4 g/kg/min; P=0.05) and showed a smaller decrease during the HEC (intervention: P = 0.01; time: $P < 0.001$; interaction: P=0.04) (Figure 1D). Additionally, the KD resulted in a lower suppression of palmitate flux compared to the SDD (62±16% vs. 76 ± 8%; $P<0.05$) (Figure 1E). Protein oxidation was greater after the KD during the baseline phase than after the SDD (374 ± 113 vs. 260 ± 83 kcal/24 h; $P<0.05$), though levels during the clamp period were similar between the two diets (260 ± 62 vs. 221 ± 104 kcal/24 h; P=0.13) (intervention: P=0.001; time: P=0.001; interaction: P=0.1).

DISCUSSION

Studies have indicated a direct influence of ketone bodies on insulin sensitivity. For instance, one study found that consuming ketone salts improved glucose tolerance following an oral glucose tolerance test[3], while another study demonstrated that an acute infusion of β-hydroxybutyrate (OHB) boosted insulin-stimulated glucose uptake during an HEC.[4] In the latter study, insulin resistance was induced by stimulating lipolysis with a growth hormone infusion, and the observed increase in glucose uptake was likely facilitated by the effective suppression of lipolysis that occurred with the infusion of OHB. Other studies have shown that an OHB infusion did not significantly affect insulin sensitivity measured during an HEC, although this was noted at relatively low circulating levels of ketone bodies (0.4 mmol/L).[5]

CONCLUSION

A three-week KD resulted in an improvement in insulin sensitivity in skeletal muscle, no change in hepatic insulin sensitivity, and a decrease in insulin sensitivity in adipose tissue. These findings underscore the contrasting adaptive effects of KD on insulin sensitivity in different organs. While the positive impact of KD on skeletal muscle insulin sensitivity suggests potential metabolic advantages of this dietary strategy, additional research with longer durations and definitive outcomes, such as the development of type 2 diabetes, is necessary to confirm the effectiveness of KD.

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Impact of delayed medical treatment on the development of Diabetic foot in type 2 diabetic patients

INTRODUCTION

The likelihood of DF incidence in diabetic patients is greatly impacted by delayed medical therapy. Good predictive value is shown by plasma glucose levels, HbA1c levels, and the combined predictive model of postponed medical treatment.

Type 2 diabetes mellitus is a chronic condition characterized by the body's inadequate use of insulin or diminished insulin effectiveness, resulting in consistently high blood sugar levels. Extended periods of hyperglycemia can lead to the onset and progression of multiple complications.[1] Diabetic foot (DF) is a significant complication associated with diabetes that involves nerve damage in the lower limbs and varying degrees of vascular disease.[2,3] Diabetes patients often neglect to seek foot examinations at healthcare facilities to evaluate their risk of developing DF. As a result, delays in receiving medical attention frequently contribute to the onset of DF. While earlier studies have examined the risk factors associated with DF, there has been no research specifically addressing how delayed medical care influences the likelihood of DF development in individuals with diabetes.[1] Consequently, it is crucial to examine how delayed medical care affects the occurrence of DF in patients with diabetes. So, this study aimed to highlight the effects of postponed medical treatment on the development of DF in individuals with type 2 diabetes and to create a predictive model for DF.

METHODS

This retrospective cohort study involved 292 individuals diagnosed with type 2 diabetes. The participants were categorized into two groups: the diabetic foot (DF) group (n = 82) and the nondiabetic foot (NDF) group (n = 210). Inclusion criteria included: (1) a diagnosis of type 2 diabetes; (2) age between 18 and 80 years; and (3) ineligibility for surgical revascularization.

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Analyses of demographic characteristics, laboratory parameters, and delays in medical treatment were conducted for both groups, including differential and correlation analyses. Logistic regression was utilized to identify influencing factors, and receiver operating characteristic (ROC) analysis was performed to select indicators with strong predictive value for developing a combined predictive model.

RESULTS

The DF group demonstrated significantly higher body mass index (BMI) ($P < 0.001$), longer disease duration ($P = 0.012$), elevated plasma glucose levels ($P < 0.001$), and higher HbA1c levels ($P < 0.001$) compared to the NDF group. Specifically, the DF group had plasma glucose levels of 10.24 ± 4.72, compared to 5.71 ± 0.61 in the NDF group ($P < 0.001$), and HbA1c levels of 9.12 ± 1.65, versus 7.41 ± 1.87 in the ND F group ($P < 0.001$). Furthermore, the NDF group exhibited significantly higher Acute Thrombosis and Myocardial Infarction Health Service System (ATMHSS) scores ($P < 0.001$) and a markedly lower rate of delayed medical treatment (72.38% vs. 13.41%, $P < 0.001$). Positive correlations were found between DF occurrence and factors such as BMI, duration of diabetes, plasma glucose levels, HbA1c, diabetic peripheral neuropathy, and nephropathy. In contrast, ATMHSS scores were negatively correlated with the delay in seeking medical treatment. The ROC analysis showed that plasma glucose levels, HbA1c and delayed medical treatment were strong predictors, achieving an area under the curve of 0.933 for the combined predictive model (Figure 1).

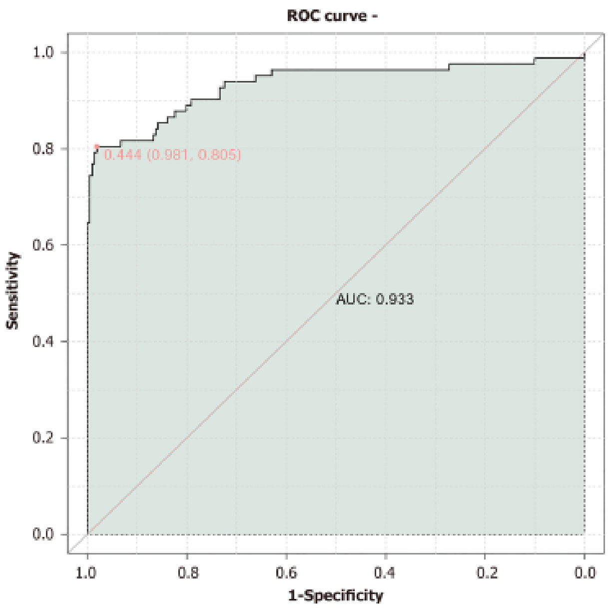

Figure 1. Analysis of the combined predictive model for plasma glucose, HbA1c, and medical delay. ROC: Receiver operating characteristic; AUC: Area under the curve.

DISCUSSION

The main finding of this study was the influence of medical delays on the incidence of DF. One possible explanation for this was that delays in seeking medical attention can result in the disease being diagnosed at a more advanced stage, which significantly diminishes the effectiveness of treatment, increases the burden on patients, and may negatively impact their short- and long-term outcomes. Patients' reluctance to seek medical care could stem from inadequate education, a lack of awareness regarding DF prevention, and an absence of a routine for timely check-ups and healthcare.[4,5] Moreover, the early signs of DF, including coolness in the soles and reduced sensation, were often mild and easily missed by patients, which can contribute to delays in seeking medical attention.

CONCLUSION

Delays in medical care play a crucial role in the likelihood of developing DF among patients with diabetes. The integrated predictive model, which includes plasma glucose levels, HbA1c levels, and the timing of medical care, shows strong predictive capabilities.

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Efficacy of Patient-Centered Self-Management Intervention on Glycemic Control, Self-Efficacy, and Self-Care Practices in Type 2 Diabetes Mellitus

INTRODUCTION

PACE-SMI significantly improved self-efficacy and self-care behaviors in persons with type 2 diabetes, and it resulted in a modest but significant improvement in HbA1c.

Type 2 diabetes mellitus (T2DM) constitutes more than 90% of diabetes cases globally, with a significant portion of those affected residing in low- to middle-income countries (LMIC) in South Asia.[1] Such increasing prevalence of T2DM and its related complications has led to decreased functional capacity in individuals, impairing their quality of life and driving up healthcare costs, which poses significant challenges for both the healthcare system and the economy. Therefore, there is an urgent need for effective interventions to prevent disease progression and minimize the risk of complications. Evidence indicates that lifestyle modifications, often referred to as self-management, are essential for slowing the progression of T2DM and reducing the likelihood of serious complications. Implementing a patient-centered behavioral or social intervention may significantly enhance the effective self-management of T2DM.[2] A recent meta-analysis conducted in developed countries discovered that patient-centered self-management interventions (PACE-SMIs), which combine educational and behavioral elements, can significantly reduce HbA1c levels and enhance self-care behaviors in adults with T2DM compared to standard care.[3] To assess its effectiveness in LMICs, this study evaluated the efficacy of a PACE-SMI in improving HbA1c levels, self-efficacy, and self-care behaviors among adults with T2DM.

METHODS

In this multicenter, parallel two-arm randomized controlled trial, a total of 612 adults with T2DM and HbA1c levels of 7% or higher were recruited and randomly assigned to either the control group (n = 310) or the intervention group (n = 302) through stratified permuted block randomization. Participants included adults aged 18 years and older who had been diagnosed with T2DM for at least six months. The control group received standard care, while the intervention group received standard care along with a nurse-led, theory-based, culturally adapted PACE-SMI, which included eight weekly sessions focused on individualized education, counseling, behavioral training, and home visits. Outcomes were evaluated at baseline, immediately after the intervention, and three months later.

RESULTS

Data at the three-month mark were collected from 583 participants (control: n = 295, intervention: n = 288). Per-protocol analysis revealed that, at T2, the intervention group had a lower mean HbA1c of 8.49% (standard deviation [SD], 1.58) compared to the control group, which had a mean of 8.74% (SD, 1.62). This represented a small but statistically significant mean difference of 0.25% (95% confidence interval [CI], -0.01 to 0.51; Cohen's d = 0.16; p = 0.03). Additionally, self-efficacy and self-care behaviors showed significant improvement in the intervention group (116.89 [SD, 25.50] and 70.01 [SD, 17.97], respectively) compared to the control group (75.43 [SD, 18.99] and 51.54 [SD, 12.04]), with mean differences of 41.48 (95% CI, 37.83–45.13; Cohen's d = 1.84; p < 0.0001) and 18.56 (95% CI, 16.08–21.04; Cohen's d = 1.22; p < 0.0001), respectively. Figure 1 illustrates the changes in HbA1c, self-efficacy, and self-care behaviors at T0, T1, and T2. Furthermore, linear regression analysis indicated that the effect of PACE-SMI on HbA1c was significantly mediated by enhancements in self-efficacy and self-care behaviors ($R_2 = 0.232$, $p < 0.001$).

DISCUSSION

HbA1c is a crucial clinical marker for the effective management of T2DM. In this trial, participants who received PACE-SMI experienced a small but statistically significant improvement in HbA1c after three months. Although the mean difference in HbA1c between the two groups appears modest, the study indicates that even minor reductions in HbA1c can significantly lower the risk of complications associated with diabetes. For example, the UK Prospective Diabetes Study (UKPDS) found that a 1% reduction in HbA1c correlates with a 21% decrease in the risk of any diabetes-related complication.[4] Effective counseling has been linked to enhanced self-efficacy and a decrease in distress related to T2DM.[5]

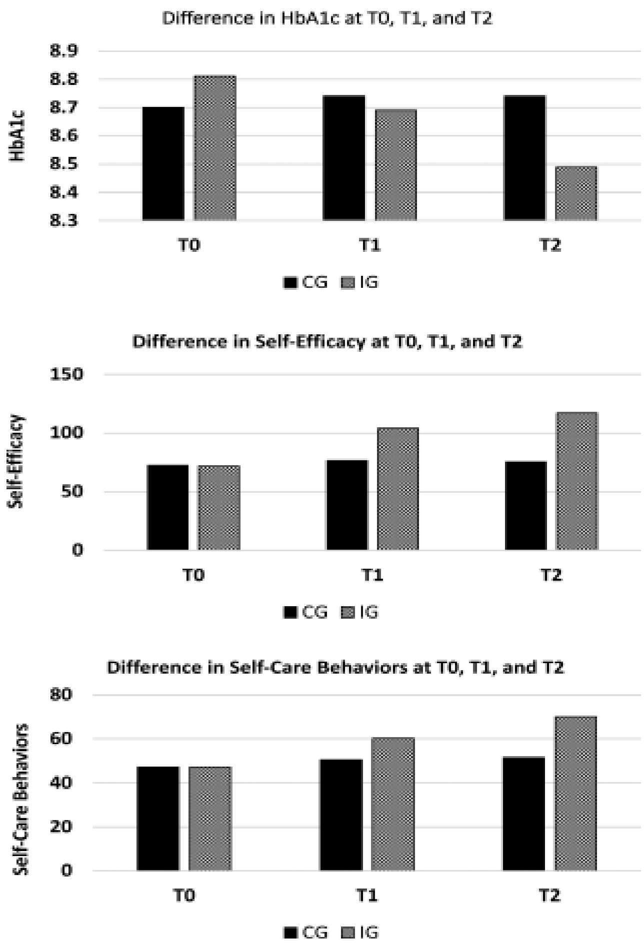

Figure 1. (A) Change in HbA1c at T0, T1, and T2; (B) Change in self-efficacy at T0, T1, and T2; and (C) Change in self-care behaviors at T0, T1, and T2. CG, control group; IG, interventional group

CONCLUSION

This study found that the PACE-SMI led to a modest but significant improvement in HbA1c, along with substantial gains in self-efficacy and self-care behaviors among adults with T2DM. Considering the health and economic implications of T2DM, these results advocate for the incorporation of PACE-SMI into standard practice, offering essential insights for national healthcare policies.

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Case report on Diabetic Myonecrosis in a Young Woman with Long-Term Type 1 Diabetes Mellitus

INTRODUCTION

Diagnosis of Diabetic Myonecrosis relies on clinical and imaging characteristics, necessitating an increased awareness in patients with a history of long-standing diabetes and microvascular complications, as its presentation can resemble several other conditions.

Type 1 diabetes mellitus (T1DM) is a relatively rare condition among the Australian Indigenous population, with an incidence rate of 7 cases per 100,000 individuals, compared to 10 cases per 100,000 individuals in non-Indigenous populations.[1] A retrospective study conducted by Hare et al. on Aboriginal individuals in remote communities of the Northern Territory in Australia revealed that T1DM accounts for less than 0.2% of all diabetes cases.[2] In contrast, the incidence of type 2 diabetes mellitus (T2DM) was approximately four times greater among Indigenous Australians than in non-Indigenous populations. Furthermore, Indigenous Australians experience higher rates of diabetes-related complications and hospitalizations.[1] Managing diabetes and its complications in Indigenous Australians frequently presents difficulties because of considerable social and cultural obstacles, along with geographical isolation.[2] This study discusses a case of diabetic myonecrosis in an Indigenous Australian woman with T1DM.

CASE PRESENTATION

A 24-year-old Indigenous Australian woman from a remote community presented with a three-month history of swelling and pain in her left thigh, along with limited mobility. She had a long-standing history of poorly controlled T1DM, diagnosed at 14 years of age, and her HbA1c was 12.4% at admission, having previously reached as high as 20.9%. Her past medical history featured acute rheumatic fever, syphilis, and recurrent urinary tract infections (UTIs) related to urinary retention. She also had a smoking history of up to 20 cigarettes daily. Upon clinical examination, she appeared pale, cachectic, and undernourished, with a sinus tachycardia of 120 bpm. The left thigh was significantly swollen and tender on palpation. Neurological examination indicated peripheral neuropathy and reduced strength in the proximal left leg. Fundal examination revealed severe bilateral proliferative diabetic retinopathy with macular edema. Investigations showed normocytic anemia, with a hemoglobin (Hb) level of 9 g/L, along with elevated inflammatory markers, including platelets, white blood cell count, neutrophils, erythrocyte sedimentation rate, C-reactive protein, and ferritin. She also presented with impaired renal function. Nutritional analysis indicated severe hypoalbuminemia, low vitamin D, and low vitamin C levels. Urine microscopy and culture indicated a Urinary tract infection (UTI), and the albumin/creatinine ratio was elevated at 840 g/mol, consistent with nephrotic-range proteinuria. Renal ultrasound revealed cystitis and a renal biopsy confirmed Class III diabetic nephropathy with 30% global glomerulosclerosis and 60% interstitial fibrosis and tubular atrophy. A computed tomography (CT) scan of the pelvis and leg showed ill-defined diffuse low attenuation in the left mid-thigh muscle measuring 36 × 34 × 150 mm. Subsequent magnetic resonance imaging (MRI) demonstrated significant muscle edema in the left anterior thigh compared to the right side, affecting the rectus femoris, vastus medialis, vastus lateralis, and adductor muscles (Figure 1). The muscles appeared atrophic with fatty and fluid replacement, and the subcutaneous tissue was edematous. Differential diagnoses at this point included diabetic myonecrosis, nutritional myopathy, and inflammatory myositis. A muscle biopsy indicated significant autolytic changes and extensive muscle atrophy, with partial replacement of muscle mass by interstitial fibrosis and adipose tissue. Management focused on supportive care, optimizing glycemic control and nutrition, and managing pain while treating underlying infections. Due to her persistent inflammatory state, she received empirical treatment with intravenous amoxicillin/clavulanate for one week. Her insulin regimen was adjusted from premixed to basal-bolus insulin. Nutrition was optimized with a high-protein diet, nutritional supplement drinks, and vitamin C, vitamin D, thiamine, and multivitamin supplements. Throughout her hospital stay, pain and mobility improved, and her inflammatory markers normalized. She was then transferred back to her local hospital for

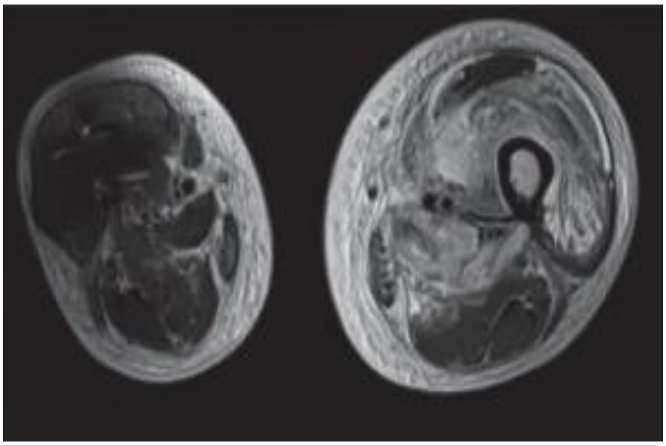

Figure 1. MRI showing significant muscle edema in the left anterior thigh affecting the rectus femoris, vastus medialis, vastus lateralis, and adductor tendons.

ongoing rehabilitation before discharge. Follow-up showed her HbA1c improved to 8.7%, and her nutritional status improved, with her weight increasing from 32.5 kg to 37.5 kg.

DISCUSSION

This case presented a significant diagnostic challenge where the primary differential diagnoses included diabetic myonecrosis, diabetic amyotrophy, and nutritional myopathy. Although the clinical presentation and progression, along with the imaging characteristics, suggested diabetic myonecrosis, the muscle biopsy did not indicate muscular necrosis.[2] The management of diabetic myonecrosis is primarily supportive, similar to this patient's approach, focusing on strict glycemic control, rest, and the use of nonsteroidal anti-inflammatory drugs (NSAIDs) as key components of treatment.[3,4] In this patient, the clinical signs and investigation results did not align with this diagnosis, as there were no indications of hemorrhage or other scurvy-related symptoms, and pain was the more prominent complaint rather than weakness. Additionally, significant vitamin D deficiency has been documented to lead to proximal myopathy, characterized by muscular pain and weakness.[5]

CONCLUSION

Diabetic myonecrosis is an uncommon complication of chronic and poorly managed diabetes mellitus that can lead to considerable morbidity and diminish quality of life. Diagnosis relies on clinical and imaging characteristics, necessitating an increased awareness in patients with a history of long-standing diabetes and microvascular complications, as its presentation can resemble several other conditions.

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