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Multisystemic Complications in a Patient with Diabetes Mellitus: A Case of Spinal Cord Compression with Chronic Kidney Disease and Diabetic Neuropathy

I Putu Eka Dharma Putra[1]
[1]Kertha Usadha hospital Singaraja, Bali, Indonesia, vios333vaa@gmail.com

Corresponding Author: vios333vaa@gmail.com[1]

Abstract: Diabetes mellitus is a major cause of multisystemic complications, particularly in patients with poor glycemic control since a young age. Chronic hyperglycemia induces vascular, neurological, and renal damage, predisposing patients to severe functional impairments and infections. **Case Report**: We present a 21-year-old male with long-standing uncontrolled diabetes mellitus who developed compressive myelopathy, diabetic kidney disease (DKD), diabetic neuropathy, and neurogenic bladder. The patient exhibited progressive lower limb weakness, urinary and fecal incontinence, chronic back pain, severe hyperglycemia, electrolyte imbalances, and a significant decline in renal function. **Discussion**: This case illustrates the complex interplay between diabetic microvascular and neurological complications. Chronic hyperglycemia led to glomerular hyperfiltration and progressive renal injury, while peripheral and autonomic neuropathy resulted in debilitating sensory-motor symptoms and recurrent urinary tract infections. The overlapping pathologies highlight the necessity for early screening, aggressive metabolic control, and a multidisciplinary approach to management. **Conclusion**: Early diagnosis and prompt multidisciplinary intervention are critical to prevent irreversible multisystem damage in patients with longstanding diabetes. Comprehensive management strategies focusing on infection control, renal protection, neuropathic pain relief, and glycemic stabilization are essential to optimize patient outcomes.

Keyword: Diabetes Mellitus; Compressive Myelopathy; Diabetic Kidney Disease; Diabetic Neuropathy; Neurogenic Bladder; Urinary Tract Infection; Chronic Kidney Disease; Multisystemic Complications.

INTRODUCTION

Myelopathy, particularly when caused by spinal cord compression, is a significant neurological condition that can lead to severe motor and sensory deficits. In regions like Indonesia, the prevalence of cervical spondylotic myelopathy (CSM) is notable, with studies indicating that

degenerative changes in the cervical spine are common among adults over 40 years old. These degenerative changes can result in chronic spinal cord compression, leading to progressive neurological impairments if not addressed timely. Early diagnosis and intervention are crucial to prevent irreversible damage and improve patient outcomes. (1)

Diabetes mellitus (DM), especially when poorly controlled, is known to cause various neurological complications, including peripheral neuropathy and autonomic dysfunction. Chronic hyperglycemia leads to metabolic and vascular changes that damage nerve fibers, resulting in symptoms such as numbness, tingling, and muscle weakness. These neurological manifestations can significantly impair a patient's quality of life and may complicate other underlying conditions, such as myelopathy, by exacerbating motor and sensory deficits. (2)

Diabetic kidney disease (DKD) is a common microvascular complication of long-standing diabetes, characterized by progressive kidney damage leading to chronic kidney disease (CKD). The pathophysiology involves glomerular hyperfiltration, thickening of the glomerular basement membrane, and mesangial expansion, ultimately resulting in proteinuria and decreased glomerular filtration rate. DKD is a leading cause of end-stage renal disease worldwide, emphasizing the importance of early detection and management of diabetes to prevent renal complications. (3)

METHOD

Autonomic neuropathy in diabetic patients can lead to neurogenic bladder dysfunction, characterized by impaired bladder sensation and contractility. This condition results from damage to the autonomic nerves controlling the bladder, leading to symptoms such as urinary incontinence, retention, and recurrent urinary tract infections. Neurogenic bladder significantly affects the patient's quality of life and can further complicate the management of diabetes and its associated complications.

Case Report

A 21-year-old male patient named IKAS from Pemogan, Denpasar Selatan, presented to the emergency unit with a chief complaint of chronic back pain. The patient reported that the pain had been ongoing for approximately four years and had significantly worsened since the previous day. Over the past four months, he had been experiencing progressive weakness in both lower extremities. Initially, he found it difficult to climb stairs, which gradually progressed to an unsteady gait and stiffness in both legs. In the last two weeks, his mobility had become severely limited, and he remained mostly on bed rest. The pain was described variably as stabbing and pulling, often accompanied by tingling sensations from the knees downward. He also reported numbness in the affected areas, and occasionally, touching the soles of his feet would trigger an electric shock-like pain or exacerbate the discomfort. Although the pain worsened with changes in position, it did not disturb his sleep. The patient also complained of urinary and fecal incontinence, along with a lack of sensation during these events. He mentioned recent unintentional weight loss but was unable to specify the amount. He denied any history of coughing or recent sexual activity.

The patient has a known history of Diabetes Mellitus since 2016, diagnosed at the age of 12. He discontinued insulin therapy in 2019 and has not undergone regular medical follow-up since. He denied any other chronic conditions such as hypertension or cardiac disease. His mother also has Diabetes Mellitus. Socially, the patient is currently unemployed and denied any history of smoking or alcohol consumption. He is unable to perform even light household tasks due to profound weakness and fatigue.

On physical examination, the patient was alert, moderately ill, and hemodynamically stable. His vital signs were as follows: blood pressure 124/87 mmHg, pulse rate 103 beats per minute (regular), respiratory rate 20 breaths per minute, temperature 37.4°C, and oxygen saturation of 99% on room air. Neurological assessment revealed a gibbus deformity in the lumbar region approximately 8 cm in size with overlying crusts and local tenderness. Muscle power in both upper limbs was 5/5 bilaterally, while in the lower limbs it was reduced to 4/4. There was no pitting edema, and both limbs were warm to touch. ABI values were within normal range: 1.07 on the right and 1.08 on the left. His conjunctivae appeared anemic, though no scleral icterus was noted. Cardiovascular and respiratory examinations were unremarkable, with normal heart sounds (S1 and S2) and clear lung fields without rhonchi or wheezing. Abdominal examination showed normal bowel sounds (5–6/min), no organomegaly, and a liver span of 8 cm without shifting dullness.

Based on clinical findings, the primary working diagnosis was compressive myelopathy, most likely due to suspected tuberculous spondylitis (Pott's disease), with a differential of a spinal cord space-occupying lesion. The presence of incontinence raised suspicion for neurogenic bladder. Additional diagnoses included long-standing diabetes mellitus (possible Type 1 or Type 2) with suspected diabetic neuropathy, and acute on chronic kidney disease most likely prerenal AKI on background CKD with differentials including diabetic kidney disease or post-infectious nephropathy. The patient was also noted to have mild anemia, possibly due to iron deficiency or anemia of chronic disease. Electrolyte imbalance, including isosmolar normovolemic hyponatremia and hypokalemia, was suspected due to inadequate oral intake. Lastly, a complicated urinary tract infection was considered as a potential concurrent condition

Laboratory investigations revealed significant abnormalities across hematological, biochemical, and urinalysis profiles. The complete blood count (CBC) showed leukocytosis with a white blood cell count of 12.05 $\times 10^9$/L and marked neutrophilia (NE# 9.60 $\times 10^9$/L), indicating an active inflammatory or infectious process. Hemoglobin was notably decreased at 8.6 g/dL, accompanied by low hematocrit (25.3%) and microcytic indices (MCV 74.9 fL, MCH 25.4 pg), suggestive of a normocytic-hypochromic anemia, possibly related to chronic disease or iron deficiency.

Urinalysis showed cloudy urine with strongly positive leukocyte esterase (++++) and marked glucosuria (4+), along with the presence of 91 leukocytes and a high bacterial load (5,505.40 per μL), confirming a complicated urinary tract infection. The urine pH was acidic (5.00), specific gravity was within normal limits (1.018), and nitrite was negative. Electrolyte studies demonstrated profound hyponatremia (Na 114 mmol/L, corrected Na 125 mmol/L) and hypokalemia (K 3.2 mmol/L), indicating normovolemic isosmolar hyponatremia likely due to low intake, as well as potassium loss or redistribution. Renal function tests were impaired, with elevated BUN (30.6 mg/dL) and serum creatinine (2.95 mg/dL), resulting in a significantly decreased estimated glomerular filtration rate (GFR) of 28.96 mL/min/1.73m², consistent with stage 3 to 4 chronic kidney disease, most likely secondary to longstanding uncontrolled diabetes mellitus.

Blood glucose was critically high at over 800 mg/dL, indicating severe hyperglycemia with likely risk of hyperosmolar state. Liver function tests were within normal range (SGOT 9 U/L, SGPT 4 U/L), and serum albumin was at the lower normal limit (3.4 g/dL), which may reflect chronic illness or mild malnutrition. Arterial blood gas analysis showed a normal pH (7.35), with slightly decreased bicarbonate (HCO_3^- 20.4 mmol/L) and negative base excess (BEecf -5.2),

suggesting mild metabolic acidosis in the setting of preserved respiratory compensation. Oxygenation was adequate with pO_2 of 133 mmHg and SO_2c of 99%.

Chest X-ray (AP view) demonstrated no abnormalities in the soft tissue, bony thorax, or pulmonary fields. The heart size and shape were normal with a cardiothoracic ratio of 48%, and the trachea was midline. No signs of consolidation or nodular lesions were seen. The impression was a normal thoracic radiograph. An abdominal X-ray was also provided, showing preserved skeletal anatomy and bowel gas pattern, but further interpretation would require radiological expertise.

The patient was initiated on a comprehensive therapeutic regimen addressing multiple clinical issues, including suspected spinal cord compression, poorly controlled diabetes mellitus, electrolyte imbalance, and complicated urinary tract infection. For infection management, Ceftriaxone 2 grams IV once daily was administered as empiric antibiotic therapy. Pain management included Paracetamol 1000 mg IV every 8 hours, complemented by Pregabalin 75 mg IV once daily and Mecobalamin 500 mcg IV every 12 hours to address neuropathic symptoms and support nerve repair.

Given the significant anemia (Hb 8.6 g/dL), the patient was scheduled for 1–2 units of blood transfusion per day, targeting a hemoglobin level of at least 10 g/dL. For glycemic control, a bolus of Insulin Glulisine (4 units) was initiated, followed by a continuous insulin infusion starting at 4 units/hour. The insulin drip rate was titrated according to blood sugar response: increased by 1 unit/hour if the glucose drop was 0–49 mg/dL, maintained if the decrease was 50–75 mg/dL, and reduced by 1 unit/hour if the reduction exceeded 75 mg/dL.

Electrolyte correction was performed with 25 mEq of potassium chloride diluted in 500 mL of NaCl 0.9%, administered over 8 hours at a rate of 20 drops per minute. Fluid management involved IV normal saline (NaCl 0.9%) at 20 drops per minute, with a target daily fluid intake of 1000–1500 mL. The patient was also prescribed a 1,900 kcal diabetic diet. The treatment plan included close monitoring and follow-up of vital signs, blood glucose levels (fasting and 2-hour postprandial), serum sodium and potassium, and 24-hour urine studies. Planned laboratory investigations included HbA1c (postponed due to anemia), C-peptide, urine albumin-to-creatinine ratio (ACR), serum iron studies (iron, TIBC, ferritin), and urine culture. A neurology consult was requested to evaluate suspected spinal tuberculosis or an extramedullary spinal lesion, with a thoracolumbar MRI as a key diagnostic tool.

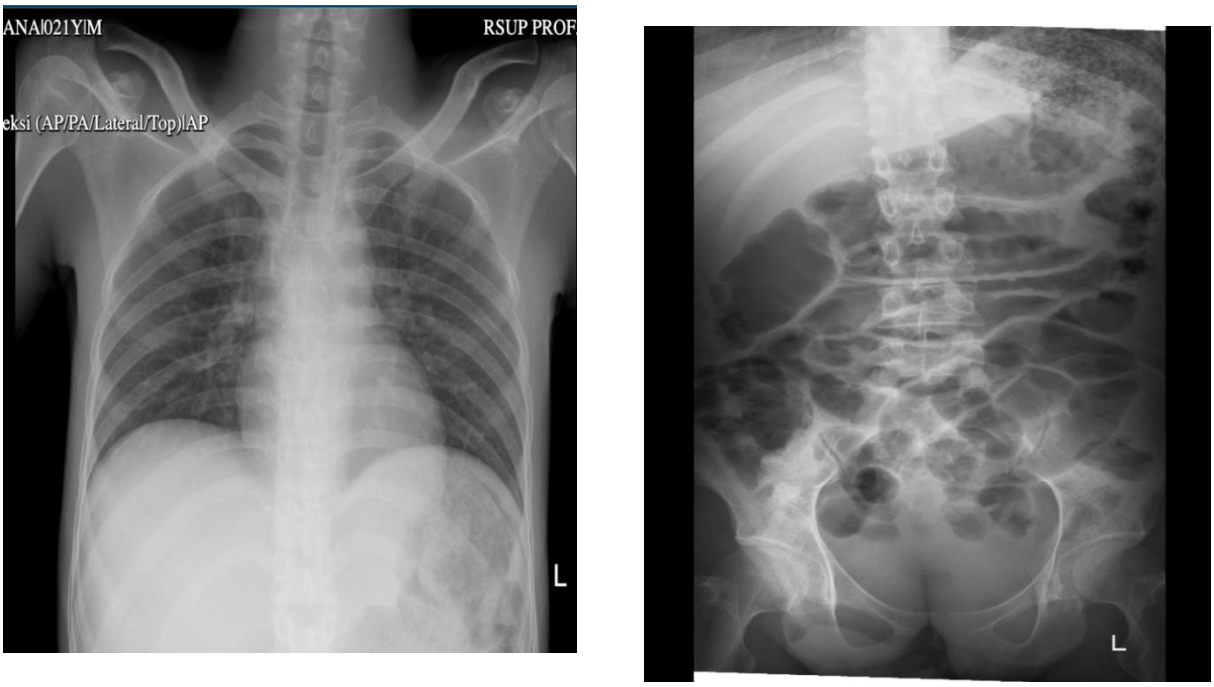

Figure 1. Rontgen thorax and BOF of the patient

RESULT AND DISCUSSION

Compressive myelopathy is characterized by a constellation of neurological deficits resulting from spinal cord compression. Patients often present with progressive motor weakness, sensory disturbances, and autonomic dysfunctions such as bladder and bowel incontinence. These symptoms reflect the involvement of various spinal cord tracts and are indicative of the severity and progression of the underlying pathology. (4)

In regions with high tuberculosis prevalence, Pott's disease (spinal tuberculosis) is a significant cause of compressive myelopathy. It typically involves the anterior vertebral bodies, leading to collapse and kyphotic deformity, which in turn compresses the spinal cord. Alternatively, spinal cord tumors, such as intradural extramedullary neoplasms, can also present with similar compressive symptoms. Differentiating between these etiologies is crucial, as it dictates distinct therapeutic approaches. (5)

The pathophysiological mechanisms underlying compressive myelopathy involve both mechanical and ischemic factors. Mechanical compression leads to direct neuronal injury, demyelination, and disruption of axonal transport. Concurrently, vascular compromise results in ischemia, exacerbating neuronal loss and gliosis. These changes culminate in irreversible spinal cord damage if not promptly addressed. (6)

Magnetic Resonance Imaging (MRI) stands as the gold standard for diagnosing compressive myelopathy. It provides detailed visualization of spinal cord anatomy, enabling the identification of intramedullary signal changes, degree of compression, and the presence of pathological lesions such as abscesses or tumors. MRI not only aids in diagnosis but also plays a pivotal role in surgical planning and prognostication. (7)

In the presented case, the patient exhibits significant renal impairment, evidenced by an estimated glomerular filtration rate (eGFR) of 28.96 mL/min/1.73 m^2, elevated serum creatinine at 2.95 mg/dL, and increased blood urea nitrogen (BUN) levels. These findings are indicative of

advanced diabetic kidney disease (DKD), a common microvascular complication of diabetes mellitus. DKD is characterized by progressive renal function decline, often leading to end-stage renal disease (ESRD) if not adequately managed. Early pathological changes in DKD include glomerular hyperfiltration, glomerular basement membrane (GBM) thickening, and mesangial expansion, which collectively contribute to the deterioration of renal function. (8)

Glomerular hyperfiltration is an early functional alteration in DKD, resulting from hemodynamic changes such as afferent arteriolar dilation and efferent arteriolar constriction. These alterations increase intraglomerular pressure, leading to mechanical stress on the glomerular capillaries and subsequent damage. Persistent hyperfiltration accelerates the progression of nephropathy by promoting proteinuria and further structural damage to the glomeruli. (9)

Structural changes in DKD, notably GBM thickening and mesangial matrix expansion, are hallmarks of disease progression. GBM thickening impairs the selective permeability of the glomerular filtration barrier, facilitating the leakage of proteins into the urine. Mesangial expansion, driven by the accumulation of extracellular matrix proteins, disrupts normal glomerular architecture and function. These morphological alterations are exacerbated by chronic hyperglycemia, which induces oxidative stress and the formation of advanced glycation end-products, further damaging renal tissues. (10)

Effective glycemic control is paramount in slowing the progression of DKD. Studies have demonstrated that intensive glucose management can reduce the onset and progression of albuminuria, a key marker of DKD, and preserve renal function . Additionally, early detection of albuminuria through regular screening enables timely intervention, potentially delaying the transition to ESRD. Therefore, integrating stringent glycemic control and routine albuminuria monitoring into the management plan is essential for patients with diabetes to mitigate the risk of DKD progression. (11)

The patient presents with classical manifestations of diabetic neuropathy, including tingling sensations, numbness, and electric shock-like pain predominantly in the lower extremities. These symptoms are characteristic of diabetic peripheral neuropathy (DPN), a common complication affecting up to 50% of diabetic individuals. Chronic hyperglycemia leads to metabolic and vascular alterations that induce oxidative stress, impair nerve blood flow, and result in axonal degeneration, thereby manifesting as sensory deficits and neuropathic pain. Over time, this peripheral nerve damage progresses proximally in a "stocking-glove" distribution, significantly impacting the patient's functional mobility and quality of life. (12)

In addition to peripheral neuropathy, autonomic nervous system involvement is evident in this patient, particularly in the form of neurogenic bladder dysfunction. Diabetic autonomic neuropathy affects the sympathetic and parasympathetic innervation of the bladder, leading to impaired sensation of bladder fullness and weakened detrusor contractility. As a result, patients develop urinary retention, overflow incontinence, and are predisposed to recurrent urinary tract infections. These complications further burden the patient's systemic condition and can exacerbate the risk of progressive renal damage. (13)

Management of diabetic neuropathy in this context aims at both symptomatic relief and prevention of further nerve damage. Pregabalin, a gabapentinoid anticonvulsant, has demonstrated significant efficacy in randomized clinical trials by reducing neuropathic pain severity and improving sleep disturbances associated with diabetic neuropathy. It works by modulating calcium channel activity at the nerve terminals, thus decreasing the release of excitatory neurotransmitters that perpetuate neuropathic pain. (14)

Furthermore, the administration of neurotropic vitamins, particularly thiamine (vitamin B1), pyridoxine (vitamin B6), and cobalamin (vitamin B12), serves an important supportive role. These vitamins are crucial cofactors in neuronal metabolism and repair mechanisms, and their supplementation has been associated with symptomatic improvement in diabetic patients suffering from neuropathy. Studies have shown that combined vitamin therapy can enhance nerve regeneration, reduce oxidative stress, and improve overall nerve conduction in affected individuals. Therefore, a multimodal approach incorporating tight glycemic control, pregabalin administration, and neurotropic vitamin supplementation forms the cornerstone of managing neuropathic and autonomic complications in this patient. (15)

CONCLUSION

In conclusion, this case illustrates the severe multisystemic complications arising from long-standing, poorly controlled diabetes mellitus, leading to compressive myelopathy, diabetic kidney disease, diabetic neuropathy, and autonomic dysfunction. The patient presented with progressive neurological deficits, urinary and fecal incontinence, and evidence of chronic kidney impairment, highlighting the interplay between metabolic, vascular, and infectious complications in uncontrolled diabetes. Early recognition and aggressive multidisciplinary management including infection control, metabolic stabilization, neurologic evaluation, and renal protection are crucial to prevent irreversible damage and to optimize patient outcomes. This case emphasizes the urgent need for continuous glycemic control and routine complication screening in diabetic patients to mitigate the profound impact of chronic hyperglycemia on multiple organ systems.

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