



12-31-2006

### Acute Neuropathy Mimicking Guillain-Barré Syndrome after Diabetic Ketoacidosis: A casereport

Po-Hong Chen

Hsiu-Fang Hsueh

Chang-Zern Hong

Follow this and additional works at: <https://rps.researchcommons.org/journal>



Part of the [Rehabilitation and Therapy Commons](#)

#### Recommended Citation

Chen, Po-Hong; Hsueh, Hsiu-Fang; and Hong, Chang-Zern (2006) "Acute Neuropathy Mimicking Guillain-Barré Syndrome after Diabetic Ketoacidosis: A casereport," *Rehabilitation Practice and Science*: Vol. 34: Iss. 1, Article 6.

DOI: [https://doi.org/10.6315/2006.34\(1\)06](https://doi.org/10.6315/2006.34(1)06)

Available at: <https://rps.researchcommons.org/journal/vol34/iss1/6>

This Case Report is brought to you for free and open access by Rehabilitation Practice and Science. It has been accepted for inclusion in Rehabilitation Practice and Science by an authorized editor of Rehabilitation Practice and Science. For more information, please contact [twpmrscore@gmail.com](mailto:twpmrscore@gmail.com).

# Acute Neuropathy Mimicking Guillain-Barré Syndrome after Diabetic Ketoacidosis: A Case Report

Po-Hong Chen, Hsiu-Fang Hsueh,<sup>1</sup> Chang-Zern Hong<sup>2</sup>

Department of Physical Medicine and Rehabilitation, Kuo General Hospital, Tainan;

<sup>1</sup>Department of Physical Medicine and Rehabilitation, Yung-Ho Hospital, Tainan;

<sup>2</sup>Department of Physical Therapy, Hung-Kuang University, Taichung.

A 42 year-old woman was admitted in a comatose state after exhibiting polydipsia and progressive body weight loss of 4-week duration. Diabetic ketoacidosis was diagnosed due to an increased blood glucose level (956 mg/dl), metabolic acidosis, and positive urinary ketone bodies. On the fourth hospital day, despite recovery from the critical state of ketoacidosis, the patient suffered from progressively ascending paresthesia and weakness in the lower limbs. On the basis of clinical and electrophysiological findings, axonal Guillain-Barré syndrome presenting as a paraparesis variant, a very unusual neuropathy related to diabetic ketoacidosis, was diagnosed. The patient reached satisfactory functional outcome after eight weeks of outpatient rehabilitation program. This article discusses the differential diagnoses of acute onset of weakness in critically ill patients. Correlation of pathogenesis between Guillain-Barré syndrome and diabetes mellitus is briefly described. Management policies of Guillain-Barré syndrome, including rehabilitative treatment, are also delineated. (Tw J Phys Med Rehabil 2006; 34(1): 41 - 47)

**Key words:** diabetic ketoacidosis, Guillain-Barré syndrome

## INTRODUCTION

Guillain-Barré Syndrome (GBS) is clinically defined as a disorder of rapidly ascending paresthesia, weakness and areflexia. It is one of the most common peripheral neuropathies, in which admission to intensive care unit (ICU) is necessary for respiratory monitoring and impending ventilatory failure.<sup>[1]</sup> However, GBS is a rarely identified neuropathic complication in the critically ill, although neuromuscular disorders, without respect to neuropathies, neuromuscular junction disorders, or

myopathies, are not an uncommon complication in the ICU.<sup>[2-10]</sup> The clinical examination of the peripheral nervous system is often unreliable in critically ill patients because of their impaired consciousness, and the presence of an endotracheal tube and intravascular lines. Comprehensive electrophysiological studies, therefore, are the best method to establish a definite diagnosis. In this article, we report a patient with diabetic ketoacidosis (DKA), which was complicated by an acute onset of paraplegia after admission to the ICU. Differential diagnoses are discussed on the basis of clinical and electrophysiological findings. Management policies, including

Submitted date: 14 September 2005.

Revised date: 28 November 2005.

Accepted date: 7 December 2005.

Address correspondence to: Dr. Po-Hong Chen, Department of Physical Medicine and Rehabilitation, Kuo General Hospital, No. 22, Second section, Ming-Sheng Road, Tainan 703, Taiwan.

Tel : (06) 2221111 ext 1260 e-mail : rpmnr70@pchome.com.tw

rehabilitative treatment, are also described.

## CASE REPORT

A 42-year-old woman was in good health, except for receiving a laparotomy due to endometriosis in March 2000. She had no history of major trauma, neuromuscular disorders or systemic diseases. Over the four weeks prior to admission, polydipsia along with progressive body weight loss manifested and became more and more prominent. Two weeks later, she began to suffer from vague tenderness around the peri-umbilical area, and one soft mass emerged progressively over the lower abdominal wall. However, she did not search for any medical assistance for the above-mentioned symptoms. In the early morning on September 30, 2001, she developed generalized weakness. Four hours later, she lost consciousness. On arrival at the emergency department, Kussmaul's breathing, body temperature of 36.4°C, blood pressure of 86/50 mmHg, and a heart rate of 142/min were observed. DKA was diagnosed on the basis of an increased blood glucose level (956 mg/dl), severe metabolic acidosis (arterial blood gas analysis: pH 6.8, PCO<sub>2</sub> 17 mmHg, PO<sub>2</sub> 137 mmHg, HCO<sub>3</sub> 6.5 µmol/l, BE -20.5 µmol/l) and urinary ketone bodies. Other laboratory findings were an electrolyte imbalance (Na 164 mEq/l, K 2.9 mEq/l), leucocytosis of 33600/mm<sup>3</sup> and an elevated C-reactive protein level of 113.9 mg/l. Serum ketone bodies and the hemoglobin A<sub>1C</sub> level (14.8%) were also increased. Thyroid function was within normal limits and autoantibodies were not detectable. The electrocardiogram revealed sinus tachycardia. The chest X-ray and computed tomogram of the brain were normal. After treating with insulin and hypotonic saline infusion for 4 hours, consciousness was regained and the ketoacidosis gradually improved. She was then admitted to the ICU.

On the third hospital day, the results of the blood gas analysis and the electrolyte levels were within normal limits. Meanwhile, neurological abnormalities were not observed. Unfortunately, the patient suddenly complained of numbness and paresthesia in bilateral lower extremities on the fourth hospital day (October 3). The symptoms exacerbated and progressively ascended proximally to the inguinal area on the following day. On physical examination, she had no obvious cranial nerve abnormalities.

Motor examination revealed normal muscle power in the upper limbs, whereas apparent weakness graded at 4/5 proximally and 0-1/5 distally in the lower limbs [Medical Research Council (MRC) scale]. Myotatic reflexes were characterized by absent bilateral ankle reflexes; 1/4 bilateral knee reflexes; and 2/4 bilateral biceps, brachioradialis and triceps reflexes. Sensory examination revealed decreased pin-prick, light touch, and temperature sensitivity in a circumferential and graded pattern over the feet to the midcalf level without the suggestion of a sensory level. There were also diminished vibration and joint position senses in the legs. Furthermore, she did not develop bladder and bowel dysfunction. An acute polyneuropathy with undetermined cause was diagnosed by a neurologist on the basis of clinical findings. However, only supportive treatment with close clinical observation was recommended. No analysis of cerebrospinal fluid (CSF) was performed on this patient. The first electrodiagnostic (EDX) evaluation, which was done on the tenth hospital day (October 9), revealed reduced amplitude and conduction velocity in tibial motor nerve conduction study (NCS), absent motor response in peroneal motor NCS, absent peroneal and tibial F waves, absent sensory response in sural sensory NCS, slightly reduced median and ulnar motor conduction velocities, and mildly prolonged median and ulnar F waves (tables 1 and 2). The EDX results were compatible with the early changes in an acute polyneuropathy. Fortunately, muscle weakness did not ascend to the upper limbs, and there were no clinical signs of respiratory compromise during the remaining hospital period.

Regarding the peri-umbilical mass, a fistulogram was performed under the impression of intestinal-abdominal fistula, in which only a subcutaneous cavity with a fistula to the skin was found. Besides, blood cultures sampled on the admission day did not reveal any pathogens, excluding a septic status.

A physiatrist was consulted because of inability to ambulate on the sixteenth day (October 15). The findings of this physical examination were similar to that one done on October 4, except for aggravated weakness graded at 3/5 proximally in lower limbs and ascending hypesthesia to the thigh level. However, the patient was almost wheelchair-bound, with impaired ability to transfer if unassisted. Minimal to moderate assistance was also

necessary in activities of daily living (ADL), especially in dressing of lower limbs, toileting, and heavy hygiene. She was referred to the outpatient department, where a rehabilitation program, including electrical stimulation, strengthening exercises of lower limbs, ambulation training, and ADL training, was prescribed. The second EDX evaluation was done on October 16, which demonstrated a complete absence of all motor, sensory and late responses in the NCS of lower limbs, but normal responses in upper limbs (tables 1 and 2). Needle electromyography (EMG) study was characterized by slightly increased insertion and spontaneous activities, normal motor unit configuration, and discrete motor unit recruitment in every examined muscle, including gastrocnemius, peroneus longus, tibialis anterior, vastus lateralis, and lower lumbar paraspinalis. Compared with the first EDX evaluation, these results suggested an acute progressing polyneuropathy. Magnetic resonance imaging (MRI) study of the lumbosacral spinal cord was also performed on October 23, and revealed nothing abnormal, suggesting the low probability of a spinal cord lesion. Thus, on the basis of the clinical and EDX findings, an axonal GBS

presenting as a paraparesis variant was diagnosed.

After eight weeks of outpatient rehabilitation, her functional status improved from being wheelchair-bound to being able to ambulate independently with a walker and bilateral ankle-foot orthoses. She was also independent in transfer and ADLs. The muscle strength based on manual muscle test was 4-5/5 in the proximal groups of lower limbs, and was 2-3/5 in the distal groups. Myotatic reflexes were 2/4 in bilateral knee and 1/4 in bilateral ankle. Sensory examination demonstrated normal sensitivity in all modalities. The third EDX studies on December 12 revealed reduced tibial motor amplitude and conduction velocity, absent peroneal motor responses, absent tibial and peroneal F waves, prolonged H reflexes, and absent sural sensory responses (tables 1 and 2). Needle EMG study revealed obvious spontaneous activity, increased neuropathic motor unit configuration, and moderately (in proximal muscles) to severely (in distal muscles) reduced motor unit recruitment in every examined muscle of the lower limbs. The above findings indicated poor correlation between clinical and electrophysiological recoveries.

Table 1. Summary of three sequential motor nerve conduction studies (symmetrical results bilaterally)

Nerve	Test #	Distal latency (msec)	Conduction velocity (m/sec)	Amplitude (mV) distal/proximal	Duration (msec) distal/proximal	F latency (msec)
Median	1	3.4	48.0	10.5/9.8	NA	30.1
	2	3.4	50.6	11.2/10.6	5.5/5.7	27.1
	3	3.4	55.1	10.0/10.0	5.5/5.8	26.4
Ulnar	1	2.4	46.0	6.3/5.9	NA	30.3
	2	2.9	53.7	5.9/5.8	5.7/6.0	26.8
	3	3.1	52.3	7.3/6.4	6.6/6.8	27.5
Tibial	1	4.9	32.0	2.6/2.1	NA	NR
	2	NR	NR	NR	NR	NR
	3	6.0	27.8	0.2/0.2	4.5/4.5	NR
Peroneal	1	NR	NR	NR	NR	NR
	2	NR	NR	NR	NR	NR
	3	NR	NR	NR	NR	NR
H reflex	1	NA				
	2	NR				
	3	41.0				

Test # 1, 2, and 3 = testing 7, 14, and 71 days following weakness onset respectively; F latency = minimal latency of F waves; NR, no response; NA, not available.

Table 2. Summary of three sequential sensory nerve conduction studies (symmetrical results bilaterally)

Nerve*	Test #	Peak latency (msec)	Amplitude ( $\mu$ V)
Median (wrist)	1	3.4	19.8
	2	3.6	26.6
	3	3.5	27.7
Ulnar (wrist)	1	3.4	20.6
	2	3.7	21.8
	3	3.3	25.1
Sural (calf)	1	NR	NR
	2	NR	NR
	3	NR	NR

Test # 1, 2, and 3 = testing 7, 14, and 71 days following weakness onset respectively. NR, no response.

\* Antidromic technique with stimulation locations shown.

## DISCUSSION

Weakness in the critically ill can lead to difficulty in weaning off a ventilator and contribute significantly to morbidity and mortality. A lot of neuromuscular disorders have been described in the literature, such as critical illness polyneuropathy (CIP), GBS, motor neuron disease, myasthenia gravis, Lambert-Eaton myasthenia syndrome, and cachectic myopathy, etc.<sup>[2-10]</sup> Differential diagnoses among these disorders are important clinically, not only for accurate prediction of the patient's prognosis, but also for prompt initiation of a specific treatment if available. In this article, we report a female patient admitted to the ICU with the diagnosis of DKA. On the fourth hospital day, the patient developed acute progressive paresthesia and weakness symmetrically in the lower limbs. An axonal GBS presenting as a paraparesis variant was diagnosed on the basis of clinical and electrophysiological findings. Although CSF analyses were lacking in our patient, her clinical presentations did meet the necessary diagnostic criteria of GBS. In fact, the diagnosis of GBS can usually be made on clinical characteristics, including the occurrence of symmetrical weakness, decrease or disappearance of the myotactic reflexes, a nadir within 4 weeks and exclusion of other diagnoses.<sup>[11,12]</sup> Another important supporting feature was the electrophysiological pattern of acute progressing polyneuropathy demonstrated in the serial EDX studies (tables 1 and 2).

Differential diagnoses rather than GBS responsible for the clinical presentations in this patient are diverse. Most of these differential diagnoses, including pure motor neuropathies, infectious neuropathies, neuromuscular junction disorders, intoxication, and myopathic disorders, were excluded based on history, clinical course and a detailed neurological examination on this patient. CIP, an axonal sensorimotor polyneuropathy, is the commonest neuropathy acquired in the ICU.<sup>[13]</sup> In a prospective study, it was present in 70% of patients with sepsis and multiple organ failure.<sup>[14]</sup> Sometimes, it is difficult to distinguish GBS from CIP, especially for the axonal variant of GBS as in this case. However, sepsis and multiple organ failure, the dominant components of CIP, were not found during the hospitalization. Besides, although CIP may occur as early as the fifth day of ICU stay, more frequently it occurs after two weeks with difficulty weaning from the ventilator and diffuse weakness.<sup>[13]</sup> In our patient, there was neither clinical sign of ventilatory compromise nor progressively generalized weakness. However, in this case, we lacked the useful data of CSF analysis to differentiate between GBS and CIP, which would show an albuminocytologic dissociation in GBS but not in CIP.<sup>[1,11,12]</sup> Nevertheless, the above clinical manifestations did not favor the diagnosis of CIP. Since this patient presented an acute onset of paraplegia, an acute myelopathy, such as transverse myelitis or vascular insult of spinal cord, was another consideration, which might account for the clinical presentations. On neurological

examination, however, there was no clear-cut sensory level, and the sphincter function was also preserved. Besides, the myotatic reflexes of lower limbs were reduced or absent throughout the whole clinical course. EDX studies revealed absent sensory responses of lower limbs, suggesting a post-ganglionic neuropathy, although a concomitant pre-ganglionic lesion could not be completely excluded. MRI of the lumbosacral spinal cord, demonstrating an absolutely normal result, provided the most important contrary evidence to a spinal cord lesion. Based on the above inferences, we also did not favor the diagnosis of acute myelopathy. As for the plateau of progression, in which the weakness was restricted clinically to her lower limbs, it could be considered as one variant of GBS. Ropper reported three patients presenting with areflexic paraparesis, which resembled a spinal cord lesion.<sup>[15]</sup> The clinical presentation of their patients was similar to that of ours. Their final diagnosis was an unusual clinical variant of GBS based on elevated CSF protein levels, normal radiographic studies, and the absence of signs of spinal cord damage. According to the above inference, GBS presenting as a paraparesis variant should be the most suitable diagnosis in this case.

Regarding the discrepancies between clinical and electrophysiological recoveries, we should take the “time-distance factor” into consideration. That is, reversible proximal conduction block often underlies rapid recovery,<sup>[16]</sup> as the better recruitment pattern in the proximal muscles demonstrated in the third EDX study. However, we performed the NCS in the distal nerve segments, which, of course, revealed a poorer electrophysiological recovery out of proportion to the clinical improvement.

GBS, the most common cause of acute nontraumatic neuromuscular paralysis,<sup>[17]</sup> is a very unusual acute neuropathy related to DKA. Only four cases of GBS have been reported as complications associated with DKA in the literature.<sup>[18,19]</sup> Up to seventy percent of patients with GBS give a history of an antecedent illness, most commonly described as an “influenza like” upper respiratory infection with fever. Ketoacidosis state, however, has never been documented as an antecedent event of GBS.<sup>[18,19]</sup> As generally accepted, GBS is an immune-mediated disorder, in which the autoimmune process is thought to be triggered by an infectious or environmental stimulus in the

genetically susceptible individual.<sup>[1,17]</sup> Similarly, type 1 DM is now considered a result of the synergistic effects of genetic, environmental, and immunologic factors that ultimately destroy the pancreatic beta cells.<sup>[20]</sup> In the past reports, two patients had had a history of DM prior to the presentation of GBS associated with DKA.<sup>[19]</sup> In those cases, the critical state of ketoacidosis seemed to be the antecedent event, which triggered the autoimmune process of GBS. In our patient, however, DM was disclosed by DKA, which was subsequently followed by GBS. The patients reported by Rouanet-Larriviere et al. had a similar situation.<sup>[18]</sup> It is likely that both autoimmune diseases were triggered by a common event. That is, GBS was not a complication of DKA in our patient; instead, they were both the result of the same autoimmune process. Nevertheless, more clinical evidence is required to address this issue.

Regarding the treatment of GBS, plasmapheresis and intravenous immunoglobulin (IVIg) are the accepted therapy, which will shorten the time required to achieve independent walking and the time a patient stays on respiratory support.<sup>[17]</sup> In our patient, fortunately, the clinical course was not so fulminant that the respiratory monitoring was necessary. Furthermore, the weakness was clinically restricted to the lower limbs at disease nadir. Therefore, aggressive treatment with plasmapheresis or IVIg was not recommended by the neurologist. In view of functional recovery, rehabilitative management should be commenced as soon as possible. In addition to the prevention of medical and musculoskeletal complications in the early stage, functional motor gain, as in our patient, and treatment of sensory dysfunction are the principal issues in the recovery stage. In brief, either medical or rehabilitative treatment is essential for the management of a patient with GBS.

## CONCLUSION

Although GBS is the most common cause of acute neuromuscular ventilatory failure requiring admission to ICU, it is an unusual complication in the critically ill patient, not to mention the patient with DKA. Sometimes, a small number of critically ill patients with weakness present quite a diagnostic challenge. In addition to a detailed neurological examination and electrophysiologic

studies, we recommend CSF analyses and MRI of spinal cord in such conditions, which would provide useful discriminating information. To our knowledge, the patient presented in this article is the first case of GBS following the critical state of ketoacidosis and being manifested as an unusual variant, i.e., the axonal paraparesis form. Although DKA may be the antecedent event of GBS, another assumption that type 1 DM and GBS are the results of a common autoimmune process is also reasonable. Of course, further study would be required to address this issue.

## REFERENCES

1. Fulgham JR, Wijdicks EFM. Guillain-Barré syndrome. *Crit Care Clin* 1997;13:1-15.
2. Jarrett SR, Mogelof JS. Critical illness neuropathy: diagnosis and management. *Arch Phys Med Rehabil* 1995;76:688-91.
3. Barohn RJ, Jackson CE, Rogers SJ, et al. Prolonged paralysis due to nondepolarizing neuromuscular blocking agents and corticosteroids. *Muscle Nerve* 1994;17:647-54.
4. Faragher MW, Day BJ, Dennett X. Critical care myopathy: an electrophysiological and histological study. *Muscle Nerve* 1996;19:516-8.
5. Gooch JL, Suchyta MR, Balbierz JM, et al. Prolonged paralysis after treatment with neuromuscular blocking drugs. *Crit Care Med* 1991;19:1125-31.
6. Hund E. Myopathy in critically ill patients. *Crit Care Med* 1999;27:2544-7.
7. Lacomis D, Petrella JT, Giuliani MJ. Causes of neuromuscular weakness in the intensive care unit: a study of ninety-two patients. *Muscle Nerve* 1998;21:610-7.
8. Lopez Messa JB, Garcia A. Acute polyneuropathy in critically ill patients. *Intensive Care Med* 1990;16:159-62.
9. Spitzer AR, Giancarlo T, Maher L, et al. Neuromuscular causes of prolonged ventilator dependency. *Muscle Nerve* 1992;15:682-6.
10. Zochodne DW, Ramsay DA, Saly V, et al. Acute necrotizing myopathy of intensive care: electrophysiological studies. *Muscle Nerve* 1994;17:285-92.
11. Asbury AK, Arnason BG, Karp HR, et al. Criteria for the diagnosis of Guillain-Barré syndrome. *Ann Neurol* 1978;3:565-6.
12. Van der Meché FGA, Van Doorn PA, Meulstee J, et al. Diagnostic and classification criteria for the Guillain-Barré syndrome. *Eur Neurol* 2001;45:133-9.
13. Chen R. Electrophysiological studies in the critical care unit: investigating polyneuropathies. *Can J Neurol Sci* 1998;25(1 Suppl):32-5.
14. Witt NJ, Zochodne DW, Bolton CF, et al. Peripheral nerve function in sepsis and multiple organ failure. *Chest* 1991;99:176-84.
15. Ropper AH. Unusual clinical variants and signs in Guillain-Barré syndrome. *Arch Neurol* 1986;43:1150-2.
16. Preston DC, Shapiro BE. Polyneuropathy. In: Preston DC, Shapiro BE, editors. *Electromyography and neuromuscular disorders*. 1st ed. Boston: Butterworth-Heinemann; 1998. p.355-89.
17. Meythaler JM. Rehabilitation of Guillain-Barré syndrome. *Arch Phys Med Rehabil* 1997;78:872-9.
18. Rouanet-Larriviere M, Vital C, Arne P, et al. Guillain-Barré syndrome occurring in two women after ketoacidotic comatose state disclosing an insulin-dependent diabetes mellitus. *J Peripher Nerv Syst* 2000;5:27-31.
19. Fujiwara S, Oshika H, Motoki K, et al. Diabetic ketoacidosis associated with Guillain-Barré syndrome with autonomic dysfunction. *Intern Med* 2000;39:495-8.
20. Foster DW. Diabetes mellitus. In: Braunwald E, Fauci AS, Kasper DL, et al, editors. *Harrison's principles of internal medicine*. 15th ed. New York: McGraw-Hill, inc.; 2001. p.2060-80.

# 糖尿病酮酸中毒併發類基蘭巴瑞症候群之急性神經病變：病例報告

陳柏宏 薛琇方<sup>1</sup> 洪章仁<sup>2</sup>

郭綜合醫院復健科 永和醫院復健科<sup>1</sup> 弘光科技大學物理治療系<sup>2</sup>

基蘭巴瑞症候群是週邊神經疾病中常見需要重症加護治療的診斷之一，甚至常常需要使用呼吸器治療。然而，在各類重症加護的疾病病患中，基蘭巴瑞症候群卻是極少見的週邊神經併發症，雖然其他神經肌肉疾病，譬如神經肌肉交接處病變(neuromuscular junction disorders)或各種肌肉病變(myopathy)，常常出現在重症加護疾患的身上。

本文描述一位四十二歲女性因呈現重度昏迷轉入本院加護病房，入院前四週患者開始出現多喝、多尿及體重快速減少的症狀。實驗室檢查顯示患者的血糖值高達 956 mg/dl，同時出現代謝性酸中毒以及尿中酮體陽性反應，因此診斷為糖尿病酮酸中毒。經過大量體液補充以及胰島素靜脈注射治療後患者意識逐漸恢復，而且實驗室檢查數據也趨於正常值。然而患者在入院後第四天下肢逐漸出現上行性肌肉無力與感覺異常(ascending weakness and paresthesia)，根據患者臨床症狀表現、脊椎磁共振攝影以及肌電生理檢查的結果，診斷為軸突性(axonal)基蘭巴瑞症候群，同時表現為截癱變異型(paraparesis variant)，此為糖尿病酮酸中毒極少見之相關合併症。患者出院後經過八週的門診復健治療，已可持助行器行走，同時日常生活活動可以完全獨立執行。本文將進一步討論加護重症病患發生急性肌肉無力的鑑別診斷，同時會針對基蘭巴瑞症候群與糖尿病的病生理機轉之相關性進行探討。對於基蘭巴瑞症候群的診斷和處理原則，以及相關的復健治療也將一併描述。(台灣復健醫誌 2006；34(1)：41 - 47)

**關鍵詞：**糖尿病酮酸中毒(diabetic ketoacidosis)，基蘭巴瑞症候群(Guillain-Barré syndrome)



