Simultaneous presentation of thyrotoxic crisis and diabetic ketoacidosis

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Summary

Two patients presented with co-existing development of diabetic ketoacidosis and thyrotoxic crisis. The difficulties of diagnosis and management of such cases are discussed.

Introduction

The combination of diabetes mellitus with thyrotoxicosis is well known and one condition usually precedes the other (Wilder, 1926) but the co-existence of diabetic ketoacidosis and thyrotoxic crisis is rare (Hanscom and Ryan, 1957). Two cases are described where these serious complications occurred simultaneously as the presenting features and provided difficulties in diagnosis and management.

Case reports

Case 1

An 18-year-old female presented with a 2-day history of generalized abdominal pain and vomiting. For one week she complained of polyuria and polydipsia. There was a pyrexia of 40°C, she was semi-comatose, dehydrated and ketoacidotic. The pulse was 180 sinus rhythm and the BP 80/60 mmHg. There was generalized guarding and tenderness of the abdomen with absent bowel sounds. The results of the serum electrolytes showed a sodium 135 mmol/l, potassium 2.6 mmol/l, chloride 75 mmol/l, bicarbonate 6 mmol/l, urea 5 mmol/l (30 mg/dl), and glucose 24 mmol/l (412 mg/dl). Treatment was commenced with i.v. fluids and insulin. After 12 hr the results of the serum electrolytes showed a sodium of 140 mmol/l, potassium 4.6 mmol/l, chloride 105 mmol/l, bicarbonate 14 mmol/l and glucose 12 mmol/l (216 mg/dl). However, there was no significant improvement in her general condition and the physical signs remained unchanged. At this stage a laparotomy was carried out.

At laparotomy fresh, clear, free fluid with enlargement of the mesenteric lymph nodes was found. Postoperatively she developed signs of meningism,

the CSF showed a slight increase in the lymphocyte count but was under normal pressure. She also developed a supraventricular tachycardia with a rate of 260/min which responded to i.v. practolol and digoxin reducing the rate to 120/min. The following day, a goitre and bilateral exophthalmos had become increasingly obvious. A diagnosis of thyrotoxic crisis was made and treatment commenced with propranolol, carbimazole and potassium iodide given *via* a nasogastric tube, in conjunction with continuous i.v. fluids and insulin. Within 24 hr her general condition had improved dramatically. Further investigations are illustrated in the table. After 6 months her thyrotoxicosis had gone into remission and her diabetes was well controlled on a twice daily combination of soluble and isophane insulin.

Case 2

A 31-year-old female, gave a 3-week history of weight loss, thirst and polyuria. For 2 days before admission to hospital she complained of persistent vomiting and generalized abdominal pain. On examination she was extremely restless, dehydrated, ketoacidotic with a pyrexia of 40°C. The BP was 100/60 mmHg and a pulse rate of 160 sinus rhythm. There was a well marked exophthalmos, an enlarged thyroid with an associated bruit. The blood glucose was 24 mmol/l (412 mg/dl) and the bicarbonate 4 mmol/l.

She was treated with i.v. fluids and insulin. Propranolol, carbimazole and potassium iodide were given *via* a nasogastric tube. Within 48 hr her general condition had improved significantly. Further investigations are shown in the table.

One year since her presentation she remains on conventional anti-thyroid drugs and a twice daily combination of soluble and isophane insulin. A Synacthen test of adrenal function remains normal.

Discussion

Case 1 suggested the possibility of a viral aetiology

TABLE 1. Results of investigations

	Case 1		Case 2	
Serum thyroxine (normal range 53–160 nmol/l)	205 nmol/l		210 nmol/l	
Free thyrotoxin index (normal range 45–150)	305		220	
Autoantibodies				
Thyroid – Thyroglobulin	–		+++	
Colloid	+		+++	
Cytoplasmic	+		+++	
Islet cell	–		+++	
Adrenal	–		+++	
Gastric parietal cell	–		+++	
HLA antigens	A2.AW26. B13.BW40.		A1.A2.B8.	
	<i>Sera</i>		<i>Sera</i>	
Virology	1.	2.	1.	2.
Coxsackie titres				
B2	40	80	32	64
B4	20	20	128	256
B5	40	80	12	12

because of the associated meningoencephalitis and mesenteric lymphadenitis, but it was not possible to confirm this assumption. However, Case 2 showed a rising titre to Coxsackie B2 and B4. A combination of an infection with Coxsackie B4 virus, the HLA type B8 and high titres of islet cell antibodies have been implicated in the aetiology of insulin-dependent diabetes (Gamble, 1974; Morris *et al.*, 1976) and these features were found in Case 2. Furthermore, there is an association of this particular HLA type with thyrotoxicosis (Grumet *et al.*, 1974) and in Case 2 there was also a striking autoantibody response to the thyroid, adrenal and stomach but these features were not apparent in Case 1. In these patients, it is not evident if one condition preceded or precipitated the other, because both had an acute fulminating presentation and furthermore the diabetic ketoacidosis may obscure the thyrotoxic crisis.

Thyrotoxicosis can produce vomiting (Rosenthal, Jones and Lewis, 1976) and diabetic ketoacidosis is a well known cause of abdominal pain and vomiting.

Clearly, although the surgeon may be well aware of the diabetic ketoacidosis, thyrotoxicosis must also be considered in the differential diagnosis, thus avoiding the difficulties in the management that were apparent in Case 1.

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