# THE ILL DIABETIC PATIENT

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*Introduction*

*Diabetes mellitus is one of the commonest diseases that afflicts mankind; in Malta there is a particularly high prevalence. Diabetes mellitus is therefore one of the diseases which we encounter most frequently in our professional lives. A diabetic patient can become ill either because of a reason directly related to his diabetes, or due to other diseases that also afflict the non-diabetic population. We will consider each in turn.*

## *ACUTE METABOLIC DISORDERS*

### *Hypoglycaemia*

Although hypoglycaemia can also occur spontaneously in liver failure, Addison's disease, hypopituitarism and insulinoma, it is largely a side effect of anti-diabetic treatment. Both insulin and sulphonamides can cause hypoglycaemia; metformin rarely, if ever, does so.

The usual causes of hypoglycaemia in a diabetic patient are an excessive dose of insulin or sulphonamides, missed or ill-timed meals or unusually strenuous exercise.

### *Prevention*

A most important aspect of hypoglycaemia is its prevention.

This includes:

i Patient education regarding the importance of proper dietary regime. It cannot be overemphasised that insulin and sulphonamides should always be taken 30-45 minutes before meals. It is also important to stress the importance of the mid-morning and late evening snacks.

ii Long-acting sulphonamides such as chlorpropamide and glibenclamide, should be avoided in the elderly or in those with renal failure as these drugs would accumulate with an increased risk of hypoglycaemia. Shorter-acting sulphonamides such as tolbutamide, gliclazide and glipizide are preferable in these circumstances.

iii Avoid excessive dosage of sulphonamides and insulin.

iv Special care should be exercised in those with liver disease in view of their increased risk of developing hypoglycaemia.

v There is usually little point in aiming for very tight glycaemic control in the elderly, who are more at risk from the effects of hypoglycaemia than from long term complications of diabetes.

### *TREATMENT*

Hypoglycaemia requires prompt treatment if permanent neurological deficits are to be avoided. Treatment modalities include oral glucose (in those who can swallow), glucagon and intravenous dextrose. Glucagon has the advantage that it can be given by the intramuscular or subcutaneous routes. It can therefore be given by the patient's relatives even if the patient is vomiting or is unconscious. It works by mobilising liver glycogen; it is therefore unlikely to be effective in long-standing or recurrent hypoglycaemia as liver glycogen would be depleted under these circumstances. Intravenous glucose is the fastest and most effective way of relieving hypoglycaemia; it can be used in the patient who is unconscious or is vomiting and does not depend on an adequate store of liver glycogen. It is, however not always available (eg. at the patient's home) and requires medical supervision.

It is important to realise that hypoglycaemia can recur if it is due a long-acting sulphonamide or insulin; hence such patients need admission for continuous intravenous infusion of glucose.

Once hypoglycaemia has been treated one should try to identify its cause and take corrective action; this might include patient education on dietary regime or decreasing the dose of insulin or sulphonamide.

## *HYPERGLYCAEMIC STATES*

**Diabetic ketoacidosis** is a medical emergency occurring due to a severe deficiency of insulin. It can be the mode of presentation of type 1 diabetes, or occur in a previously diagnosed type 1 diabetic patient as a result of missed insulin therapy, gross dietary indiscretion, the presence of an acute stressful condition (eg. acute myocardial infarction, an acute abdomen or an infection) or a combination of these factors.

The hallmarks of the condition are hyperglycaemia, systemic metabolic acidosis with consequent hyperventilation, glycosuria and ketonuria.

**Hyperosmolar non-ketotic syndrome** is thought to occur due to a lesser degree of insulin deficiency so that there is hyperglycaemia (often severe) but no ketosis. There is an increased thrombotic tendency in this condition which carries a relatively poor prognosis.

**Lactic acidosis** is not specific to diabetes but can occur whenever there is tissue hypoxia, such as heart failure and shock. Lactic acid is a product of anaerobic combustion of glucose. Biguanides can precipitate lactic acidosis; phenformin is particularly dangerous in this regard and therefore should no longer be used. Metformin is safer and is the biguanide of choice. Biguanides should not be used in those with renal or hepatic impairment, heart failure, coronary artery disease, a history of alcohol abuse or previous an episode of lactic acidosis.

Diabetic ketoacidosis, lactic acidosis and hyperosmolar non-ketotic syndrome all require hospitalisation and intensive management including intravenous insulin, meticulous attention to fluid and electrolyte balance and frequent monitoring. Prophylactic heparin is often used in the hyperosmolar non-ketotic syndrome in view of its thrombotic tendency.

## *CONDITIONS NOT DIRECTLY RELATED TO DIABETES*

Diabetic patients can suffer from any acute illness that a non-diabetic can. Indeed diabetes predisposes to a number of other diseases, including coronary artery disease and infections.

Any acute illness such as an infection or an acute coronary event can precipitate loss of glycaemic control as insulin demands are increased in these circumstances. Diabetes can, in turn, inhibit resolution of an infection.

Glycaemic control is therefore of paramount importance in an ill diabetic patient. It can be summarised thus:

- If possible maintain an adequate calorie intake and continue on the patient's usual treatment regime.
- If necessary top up anti-diabetic treatment by increasing the insulin dose.
- If necessary change over to insulin (often requires hospitalisation) until the acute episode has resolved.
- Stop biguanides in situations at risk of lactic acidosis (such as dehydration, shock, acute coronary events).
- If a patient or insulin treatment is not eating or is vomiting, he requires hospitalisation for intravenous glucose and insulin.
- **NEVER STOP INSULIN.**

## *DIABETES AND INFECTION*

Diabetes mellitus and infection interact in a number of ways:

- Diabetic patients are more prone to infection (eg. urinary tract infection, pyothorax and abscesses). It has, for example, been shown that neutrophils from diabetic subjects exhibit impaired bactericidal activity.
- Signs of infection may be modified.
- Infection worsens diabetic control (there are increased insulin demands as a result as increased counter-regulatory hormones such as cathecolamines and glucocorticoids).
- Poor glycaemic control inhibits resolution infection.

It is therefore of paramount importance to:

- Treat bacterial infection promptly.
- Achieve good glycaemic control.

If infection does not respond adequately to treatment consider:

i Changing antibiotic regime. This can mean using a different antibiotic or a different route. Enteral absorption of an antibiotic can be impaired in any ill patient; diabetes can be associated with further impairment, possibly as a result of autonomic neuropathy. If an infection is not responding to treatment, this may signify the need to change over to intravenous antibiotics.

ii Presence of an abscess/ collection of pus that needs drainage.

iii Presence of necrotic tissue (eg. in the diabetic foot). Antibiotics do not penetrate necrotic tissue; this requires surgical debridement.

iv Alternative diagnosis. Fever can be due to a non-infective cause, such as antibiotic-induced fever.