Pan Birmingham NHS
Cancer Network

Guideline for the Management of Diabetes Mellitus in Palliative Medicine

Date Approved by Network Governance	**July 2012**

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Changes between versions 1 and 2
Page 7 – last bullet point and reference to Appendix 3 removed
Appendix 3 removed and not replaced
Flow charts added to the end of document

ENDORSED BY GOVERNANCE COMMITTEE

1. Scope of the guideline

1.1 This guideline has been produced to support the management of diabetes mellitus by specialist palliative care teams, but may be useful to any clinicians dealing with patients with diabetes who are dying.

2. Guideline background

2.1 The prevalence of diabetes mellitus (DM) is 3-5% population of the UK[1,2,3]. It is a disease which results in significant morbidity and mortality and management may require significant lifestyle changes[1,2,3].

2.2 DM is a disease characterized by persistent hyperglycaemia (high blood sugar levels), resulting either from inadequate secretion of the hormone insulin, an inadequate response of target cells to insulin, or a combination of these factors[1,2,3,4]. Insulin is crucial to the balance of glucose metabolism[1,2,3,4]. In people with type 1 diabetes, there are two reasons for hyperglycaemia. The total absence of insulin results in overproduction of hepatic glucose leading to hyperglycaemia. There is also a marked defect in peripheral glucose utilization. As the body is unable to utilize the glucose as energy, the body changes from metabolism based on carbohydrate, to fat oxidation. Fatty acids are broken down to form ketone bodies[1,2,3,4]. Ketones are acidic and their build-up can result in metabolic acidosis and nausea[1,2,3,4]. If left untreated, coma and death will occur[1,2,3,4]. For type 2 diabetes, there is not usually a total absence of insulin although in extreme cases hyperglycaemia can lead to hyperosmolar non ketotic coma which has a high mortality.

2.3 Types of diabetes that are likely to encountered within the hospice setting are Type 1, Type 2 and steroid induced diabetes. It is extremely important to understand the differences between these forms of diabetes and ascertain which type the patient has. Most patients will have managed their diabetes for many years and any attempts at altering or adjusting regimens need to be sensitively addressed. For more information on diabetes see appendix 1.

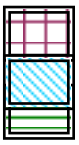
Guideline statements

3 Aims – All patients

3.1 The management of diabetes will differ for patients, depending predominantly on their prognosis. The difficulties of prognostication are well recognised and hence the guidance below should be adjusted to each patient and their changing condition e.g. reduced dietary intake[7]. There has been little formal research into the management of diabetes during the terminal phase.

3.2 Outlined below are general aims for all diabetic patients and then more specific treatment aims based on prognosis. The guideline is background coded according to prognosis for ease of use.

YEARS =
MONTHS =
WEEKS OR LESS =



GENERAL [6, 9, 10, 11]

- Always consider checking the blood sugar in any patient with diabetes whose condition has suddenly changed or in whom symptomatic hyper/hypoglycaemia is suspected.
- Avoid unnecessarily frequent monitoring of blood sugars if at all possible.
- Always consider benefit versus burden of steroids in patients with diabetes as they will worsen blood sugar control.
- For hospice inpatients - if any patient develops a diabetic emergency as outlined in appendices 2 + 3, transfer to an acute medical unit for active management should be considered. Discussion with the patient and/or relatives should be considered to ensure an informed decision on the patients' behalf given the limited resources for active management within the hospice setting [8].

PROGNOSIS – YEARS [6, 9, 10, 11]

- Maximising glycaemic control according to national guidelines to prevent long-term complications, HbA1c <7%.
- Blood pressure <140/80 mmHg
- If under the care of Diabetes team, liaise with them re: diabetes management.
- In patients with Type 2 – always use short acting sulphonylureas to reduce the risk of hypoglycaemia especially in elderly patients.

PROGNOSIS – MONTHS [6, 9, 10, 11]

- Relax BG targets aiming for BG levels 8-15mmol/L range.
- Note that some patients may feel unwell if control too relaxed. Main issue is avoidance of hypoglycaemia. Range of say 7-12 mmol/L may be better.
- Diabetes symptom free.
- In Type 2 - minimise burden/benefit of treatment e.g. Consider stopping oral antidiabetes agents and use once daily long-acting insulin etc.

TERMINAL PHASE – DAYS OR WEEKS [6, 9, 10, 11]

- Avoid hypoglycaemia.
- Try to limit symptomatic hyperglycaemia– polyuria, thirst, nausea and vomiting, drowsiness, blurred vision etc.
- Avoid unnecessary invasive monitoring of blood sugars. It is well recognised that the burden of repeated blood sugar monitoring is distressing for patients.

3.3 It can be hard for patients to adjust their thinking towards their diabetes since usually, achieving good control in order to minimise complications in the longer term has until now been the message sent out to them. Clearly, if they develop a life-limiting condition, we can help to allow them to balance maintaining reasonable control of blood sugars with maximising enjoyment of life and minimising the burden of treatment. Any changes should only be made after discussion with the patient/relatives.

4 Treatment Guidelines

4.1 These are general guidelines and it is important to consider checking blood glucose (BG) if the patient's condition changes. Particularly for patients in the terminal phase, it can be difficult to differentiate between symptoms caused by their cancer and those that may be related to altered blood sugars. Clinical awareness and judgement should be used but carrying out a BG test (with blood glucose monitoring strip/meter) should always be considered.

4.2 Hypoglycaemia (BG<4 mmol/L) may be suggested by sweating, tremor, anxiety, palpitations, confusion, aggressive/inappropriate behaviour, seizures or coma (1, 2, 3, 6).

4.3 Hyperglycaemia (BG>15 mmol/L) may be suggested by thirst, polyuria, blurred vision and fatigue (1, 2, 3, 6, 9).

PROGNOSIS – YEARS

Type1 [1, 2, 3]

- Follow patients' usual BG monitoring and insulin regimen.
- If acutely unwell, check BG to exclude hyper/hypoglycaemia. If either present, treat as diabetic emergency. (See appendix 2)
- If BGs high or low but patient not acutely unwell, adjust regular insulin accordingly usually increase or decrease by 10%dose. Consider liaising with diabetes team for advice.

Type2 [1, 2, 3]

- ***DIET CONTROLLED***
  - Monitoring at practice as usual.
  - No routine patient monitoring recommended.
  - If found to be persistently hyperglycaemic, consider commencing oral antidiabetes agent such as metformin or short acting sulphonylurea such as gliclazide.

- ***ORAL Anti-diabetes agents***.
  - No routine monitoring required unless suspect symptomatic hyperglycaemia or hypoglycaemia.
  - If found to be persistently hyperglycaemic, consider increasing oral antidiabetes agents. Liaise with diabetes team for advice if not responding.
  - Usually no need for sliding scale insulin.

- ***CONTROLLED ON INSULIN***
  - Monitor BGs as per patient's usual regimen.
  - Many patients are confident to adjust insulin dose.
  - If BG levels remain high ask diabetes team for advice.

PATIENTS ON CORTICOSTEROIDS [6, 12]

- Patients on Dexamethasone >4mg (or equivalent) and not previously diagnosed with diabetes should have their BG levels checked twice weekly for the first month of treatment and if any symptoms occur thereafter. If on Dexamethasone <4mg (or equivalent) check BG if symptomatic e.g. polydipsia, polyuria.
- Hyperglycaemia is directly linked to steroid dose and so reducing the dose should reduce blood sugars.
- Consider checking BG if suspect symptomatic hyperglycaemia or patients condition suddenly changes.
- Steroids can effect fasting pre-meal and post-meal BG due to their mechanism of action
- If persistent hyperglycaemia, follow treatment for type2 diabetes according to symptoms and degree of hyperglycaemia. Remember reducing steroids is likely to improve hyperglycaemia.
- Consult Diabetes Team for advice if in doubt.

PROGNOSIS - MONTHS

Type1 [1, 2, 3, 6, 9, 10, 11]

- Follow patient's usual BG monitoring and insulin regimen.
- If acutely unwell, check BG to exclude hyper/hypoglycaemia. If either present, treat as diabetic emergency, (see appendix 2 & 3).
- If oral intake decreasing, consider altering insulin regimen to minimise invasive monitoring and hypoglycaemic episodes aiming for BG 8-15mmol/L.

Type2 [1, 2, 3, 6, 9, 10, 11]

- ***DIET CONTROLLED***
  - No need for routine monitoring.
  - Consider checking BG only if thought to have symptomatic hyperglycaemia.
  - If persistently hyperglycaemic, consider commencing treatment taking into account burden/benefit.

- ***ORAL Antidiabetes Agents***
  - No need for routine monitoring.
  - If acutely unwell, check BG to exclude hypoglycaemia. If present, treat as diabetic emergency. (See appendix 2)
  - Consider checking BG if thought to have symptomatic hyperglycaemia.
  - If oral intake decreasing/variable, consider reducing or stopping oral antidiabetes agent to minimise risk of hypoglycaemia and burden of therapy. Monitoring should be considered during the adjustment period.
  - If persistent symptomatic hyperglycaemia consider adjusting treatment taking into account burden/benefit. Consult Diabetes Team.

- ***CONTROLLED ON INSULIN***
  - Whilst remains on insulin, follow patients usual blood monitoring regimen aiming for BG levels 8-12 mmol/L.
  - If acutely unwell, check BG to exclude hypoglycaemia. If present, treat as diabetic emergency. (See appendix 2.
  - Consider checking BM if thought to have symptomatic hyperglycaemia, (if>35mmol/L see appendix 3)
  - If oral intake decreasing or variable, consider stopping or reducing insulin to minimise risk of hypoglycaemia and burden of therapy.
  - If continuing insulin, consider once daily long-acting insulin to minimise injections. (See appendix 4)

PATIENTS ON CORTICOSTEROIDS [6, 12]

- Consider checking BG if thought to have symptomatic hyperglycaemia.
- If persistent symptomatic hyperglycaemia, consider treatment as per type2 diabetes taking into account burden/benefit. Remember reducing steroids is likely to improve hyperglycaemia.
- Consult Diabetes Team if in doubt.

PROGNOSIS – DAYS OR WEEKS

The well recognised difficulty of prognostication means it can be hard to ascertain when a patient has entered the terminal phase. Always consider the possibility of reversible diabetic emergencies when assessing an apparently dying patient-if in doubt refer to appendices and consult with Specialist Palliative Care/Diabetes Team. The guidance below must be adapted to each patient.

Type1 [1, 2, 3, 6, 9, 10, 11]

Routine BG should be checked at admission (or on first assessment for community patients) manage as table below.

BG	ACTION
<4 mmol/L	Manage as hypoglycaemia (see appendix 2)
<10mmol/L	Reduce long acting or intermediate acting insulin dose by ½.
>15mmol/L	PATIENT CONSCIOUS - Continue regular long acting or intermediate acting insulin dose and continue daily BGs, reducing when BG<10mmol/L as above. If remain persistently high and patient symptomatic, consider increasing insulin dose PATIENT IN DYING PHASE AND UNCONSCIOUS – Consider reducing long acting or intermediate acting insulin dose by ½.

There is not enough evidence to support or refute stopping insulin in this group of patients. Patients and their families often find the thought of stopping insulin distressing since they have always been told that they must never stop their insulin.

The main aim should be to avoid hypoglycaemia and symptomatic hyperglycaemia. Degrees of reduction of insulin will need to be adjusted according to each patient. Simply because a type 1 diabetic patient is not eating, it does not mean they don't need any insulin.

Once-daily BGs can be performed or discontinued if stable, at the discretion of the medical staff.

In the clearly imminently dying patient, where the burden of injections and monitoring will likely outweigh any benefit, insulin can be discontinued after discussion with the patient (if possible) and the family.

Type 2[1, 2, 3, 6, 9, 10, 11]

- ***DIET CONTROLLED***
  - No routine checks required.

- ***ORAL HYPOGLYCAEMICS***
  - Check random blood sugar on admission to ensure the patient is not hypoglycaemic, particularly if deterioration has been rapid. (see appendix 2 for treatment of hypoglycaemia).
  - General caution of metformin use in dehydrated patients, and patients with severe liver and renal impairment.
  - Particular caution required if patient on sulphonylureas because of danger of hypoglycaemia.
  - Reduce or stop oral hypoglycaemics and observe for symptoms of altered blood sugars.
  - If persistent symptomatic hyperglycaemia consider adjusting treatment taking into account burden/benefit.

- ***CONTROLLED ON INSULIN***

  Check random blood sugar on admission/first assessment and follow table below.

BG	ACTION
<4 mmol/L	Manage as hypoglycaemia (see appendix 2)
<10mmol/L	PATIENT CONSCIOUS – Stop insulin or reduce long acting/intermediate acting insulin dose by ½. PATIENT IN DYING PHASE AND UNCONSCIOUS – Stop insulin and BGs.
>15mmol/L	PATIENT CONSCIOUS - Continue regular long acting or intermediate acting insulin dose and continue daily BGs, reducing when BG<10mmol/L as above. If remain persistently high and patient symptomatic, consider increasing insulin dose by ½ or addition of sliding scale. (See appendix 3) PATIENT IN DYING PHASE AND UNCONSCIOUS – Stop insulin and BGs

STEROID INDUCED [6, 12]

It is likely that steroids will be reduced or discontinued in the terminal phase and hence blood sugars should normalise.

No need for routine BG monitoring unless the patient is already receiving treatments for hyperglycaemia in which case, treat as Type 2 diabetes above.

Monitoring of the Guideline

Adherence to the Network guidelines may from time to time be formally monitored.

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Approval Signatures

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Appendices

Appendix 1 Further Information on Diabetes Mellitus
Appendix 2 Hypoglycaemia
Appendix 3 Ketoacidosis and Honk
Appendix 4 Conversion to Once-Daily Long Acting Insulin

Appendix 1

Further Information on Diabetes Mellitus

Type 1 Diabetes Mellitus (Previously Insulin Dependent Diabetes)

This is characterised by total loss of insulin production by the beta cells within the pancreas. It usually presents in childhood or early adolescence but can present at any age[1,2,3]. Insulin is required for survival and the principal treatment of type 1 diabetes from the time of diagnosis is replacement of insulin[1,2,3]. The main emergencies associated with Type 1 are hypoglycaemia and diabetic ketoacidosis, see Appendix 2[1,2,3,4].

Type 2 Diabetes Mellitus (Previously Non-Insulin Dependent Diabetes)

This form is due to a combination of defective insulin secretion and defective responsiveness to insulin (often termed reduced insulin sensitivity) [1, 2, 3]. Type 2 diabetes is common, comprising 90% or more of cases of diabetes in many populations [1, 2, and 3]. Since it is a progressive disease, insulin may be required for adequate control although the patient is not *usually* insulin dependent[1,2,3].

In the early stages, hyperglycemia can be reversed by lifestyle measures and oral medications that increase insulin production and/or improve insulin sensitivity or reduce glucose production by the liver. As the disease progresses the impairment of insulin secretion worsens, and therapeutic replacement by insulin often becomes necessary[1, 2, 3].

At present there are three main forms of oral antidiabetes agents in use in the UK - sulphonylureas such as Gliclazide or Glibenclamide, biguanides such as Metformin, and glitazones [1, 2, 3, and 5] such as Pioglitazone and Rosiglitazone. Newer drugs such as Exanetide and Sitagliptin or Vildagliptin have reached the UK market in the last year. Their role in the treatment algorithm is presently being discussed by NICE and will not be considered further here.

Sulphonylureas stimulate insulin production whereas Metformin and glitazones tend to decrease or counter insulin resistance. Their respective mechanisms of action allow one to understand why only sulphonylureas can induce hypoglycaemia, since they can provoke excessive insulin production[1,2,3,5]. When insulin is required and used after a varying time period it is aimed at minimising symptoms and maximising glycaemic control to reduce risk of long-term complications of hyperglycaemia such as retinopathy, neuropathy and other vascular complications [1, 2, and 3].

The main emergencies associated with Type 2 are hypoglycaemia but usually only with sulphonylureas and insulin [1, 3, and 4]. Patients with Type 2 diabetes may rarely get diabetic ketoacidosis but are more likely to develop HONK - hyperglycaemic hyperosmolar non-ketotic diabetic coma, see Appendix 1[1, 3, and 4].

Steroid Induced Diabetes

Corticosteroids have a direct metabolic hyperglycaemic effect and can increase appetite [(6).] The effect is dose dependent and approximately one in five patients on high dose steroids will develop steroid induced diabetes[(6).]

NB: Patients with pancreatic cancer are particularly prone to develop diabetes due to destruction or altered function of pancreatic tissue[(6).] Sometimes the development of diabetes can be the first manifestation of pancreatic cancer.

Appendix 2

HYPOGLYCAEMIA [1, 2, 3, 4, 6, 8]

BM<4.0mmol/L For safety reasons action level probably less than 4mmol/L rather than 3mmol/L.
Symptoms - sweating, tremor, palpitations, confusion, aggressive/inappropriate behaviour, seizures or coma.

MANAGEMENT WHEN PROGNOSIS MONTHS/YEARS

PATIENT CONSCIOUS

- Give oral glucose in form of 4 teaspoons sugar in water, 120mls lucozade, or hypostop gel.
- Follow this with a longer acting carbohydrate such as toast, biscuits.

PATIENT UNCONSCIOUS

- IV glucose, 25-50mls 50% dextrose – response in 4-6 mins.

Or

- 1mg Glucagon IM/IV – response within 10mins.

NB: patients with marked cachexia may not respond to glucagon.

- It may be necessary to commence on-going 5%dextrose infusion.

Cause of the hypoglycaemia should be ascertained and adjusted e.g.; overdose of medications, altered requirement of medications, worsening renal function.

Remember that hypoglycaemia caused by sulphonylureas may last up to 72hrs and may require acute admission for management.

BGs should be monitored regularly if BGs remain low.

MANAGEMENT WHEN PROGNOSIS DAYS OR WEEKS

PATIENT CONSCIOUS

- Give oral glucose in form of 4 teaspoons sugar in water, 120mls lucozade, or hypostop gel.
- Follow this with a longer acting carbohydrate such as toast, biscuits.

PATIENT UNCONSCIOUS

- IV glucose, 25-50mls 50% dextrose – response in 4-6 mins.

Or

- 1mg Glucagon IM/IV – response within 10mins.

NB: patients with marked cachexia may not respond to glucagon.

Monitor BGs thereafter and further reduce or discontinue insulin according to patient's clinical state.

Ketoacidosis and honk

1. Diabetic Ketoacidosis [1, 2, 3, 4, 6, 8]

If any palliative patient is suspected of suffering from this condition then referral to acute medical services should be considered and if patients are conscious, discussion with them and/or relatives, regarding transfer should take place.

Usually presents following 2-3 day history of decline with polyuria, polydipsia, lethargy, anorexia, hyperventilation, ketotic breath, vomiting, and coma. But presentation can be even quicker than that. Precipitants include infection, non-compliance, and incorrect insulin dose.

Diagnosis requires ketosis and acidosis and hence, we would be only able to presume such a diagnosis in the hospice setting. BG usually >20mmol/L, but not always.

This is a medical emergency and full active treatment would include IV rehydration with close monitoring and replacement of potassium. An IV insulin sliding scale would also be routine.

If it is felt inappropriate to transfer the patient or the patient/relatives make an informed decision not to transfer them to the hospital, an adapted regimen for rehydration (according to individual patient) and administration of a subcutaneous sliding scale of insulin should be commenced, according to the needs of each patient (Appendix 3).

2. Hyperglycaemic Hyperosmolar Non-Ketotic Coma (honk) [1, 2, 3, 4, 6, 8]

If any palliative patient is suspected of suffering from this condition, then referral to acute medical services should be considered and if patients are conscious, discussion with them and/or relative, regarding their transfer should take place.

Usually a 5-7day history of decline with decreasing consciousness, focal neurological signs, marked dehydration and BG >35mmol/L. Blood osmolality would be high on testing >340mmol/L.

This is a medical emergency and full active treatment would consist of IV rehydration and anticoagulation due to high risk DVT. Insulin is usually needed.

If it is felt inappropriate to transfer the patient or the patient/relatives make an informed decision not to transfer them to the hospital, an adapted regimen of rehydration should be commenced, according to the needs of each patient.

Appendix 4 [13, 14]

1. Conversion to once-daily long acting insulin

Recommended conversion to long acting insulin preparations, e.g. Glargine or Detemir, is 80% of total NPH insulin in 24hrs. Clearly this may need to be less in the presence of hypoglycaemia.

e.g. Human Mixtard 30 contains 70% NPH insulin and 30% soluble insulin.

For a patient taking 24 i.u. in the morning and 16 i.u. in the evening. Total NPH insulin in 24hrs = (24+16) x 0.7 = 28 i.u.

Conversion to Glargine = 28 x 0.8 = 22 i.u.

General principles of diabetes management in hospice in-patients

- See also Pan Birmingham Palliative care Network guidelines available at www.birminghamcancer.nhs.uk under important documents, network agreed guidelines, palliative care
- Aims of control are predominantly avoiding hypoglycaemia and symptomatic hyperglycaemia
- Management should reflect the stage of the disease and always follow careful discussion with the patient and/or their carers
- Management should aim to minimise frequency of testing as this is often the most distressing intervention for patients
- Avoid using metformin and glitazones in patients who are rapidly deteriorating or not eating/drinking (unlikely to be effective and risk of lactic acidosis/liver impairment)
- Avoid using bd mixed insulins – risk of lunchtime and overnight hypos
- Avoid using qds regimes which involve multiple testing and injections
- Avoid giving prn doses of actrapid – rarely achieves control, necessitates frequent testing and may cause evening hypos
- Steroids given as a single dose in the morning cause lunchtime and evening hyperglycaemia
- Steroids given bd cause marked continuous hyperglycaemia
- Long acting insulins given in patients with steroid induced diabetes may cause overnight/fasting hypos

References and further reading

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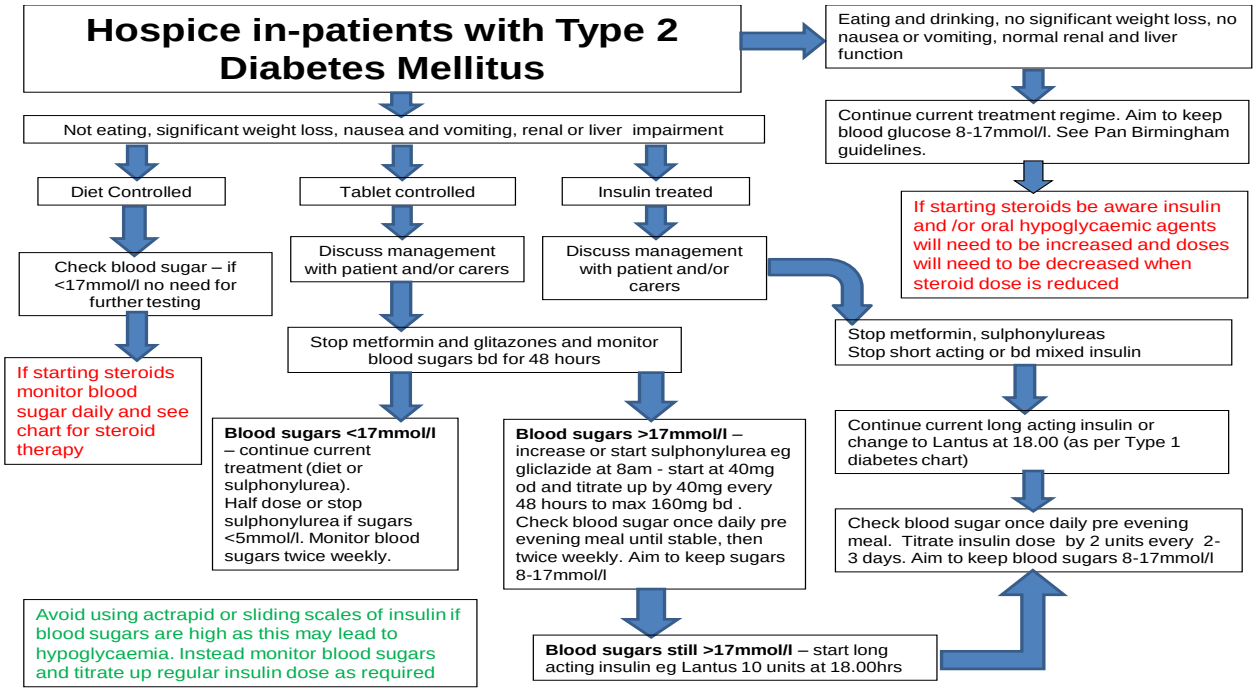

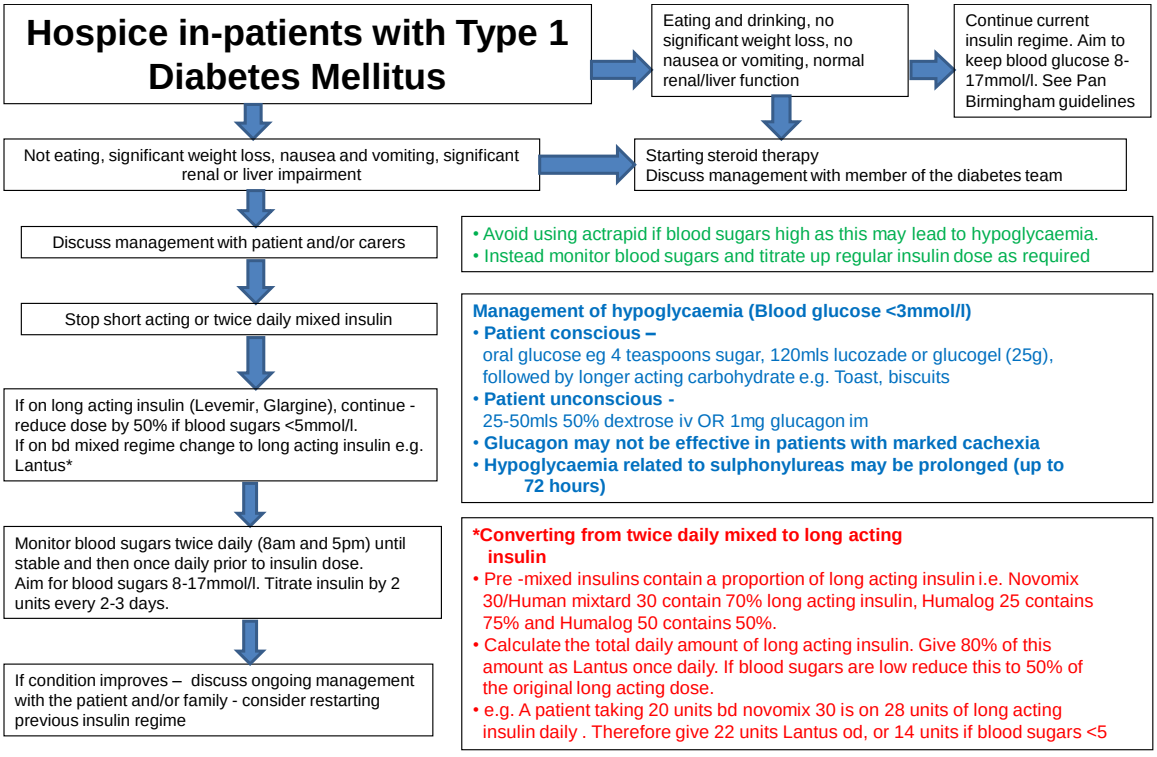

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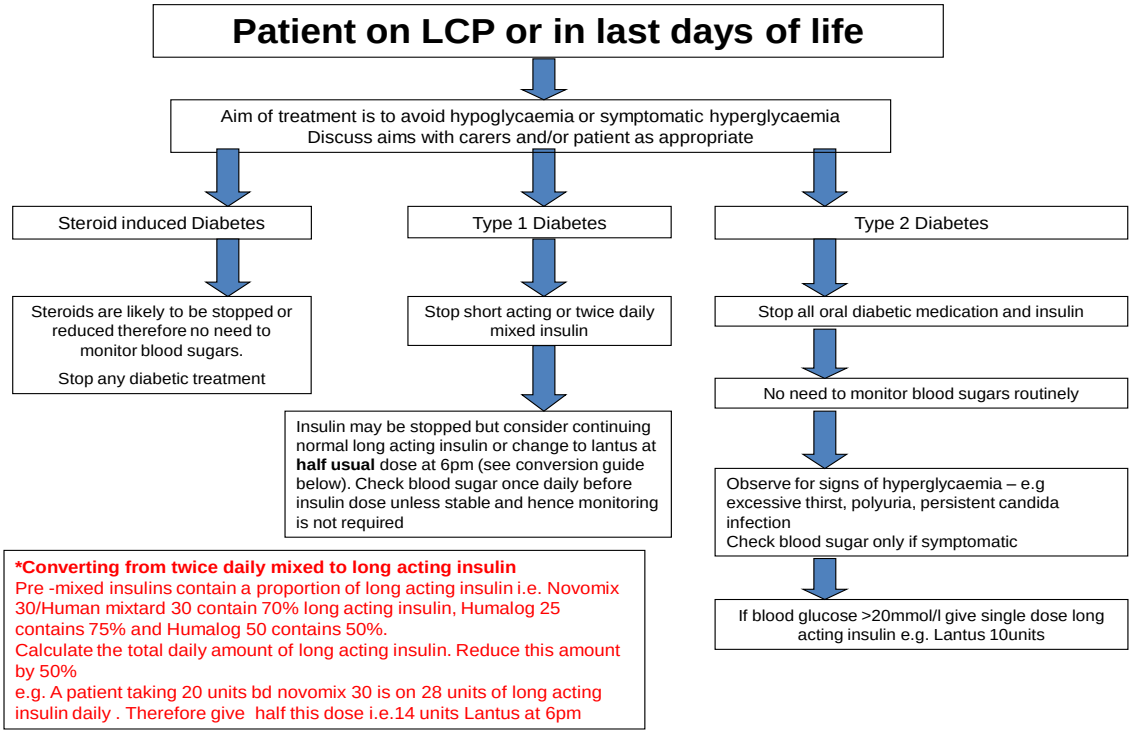

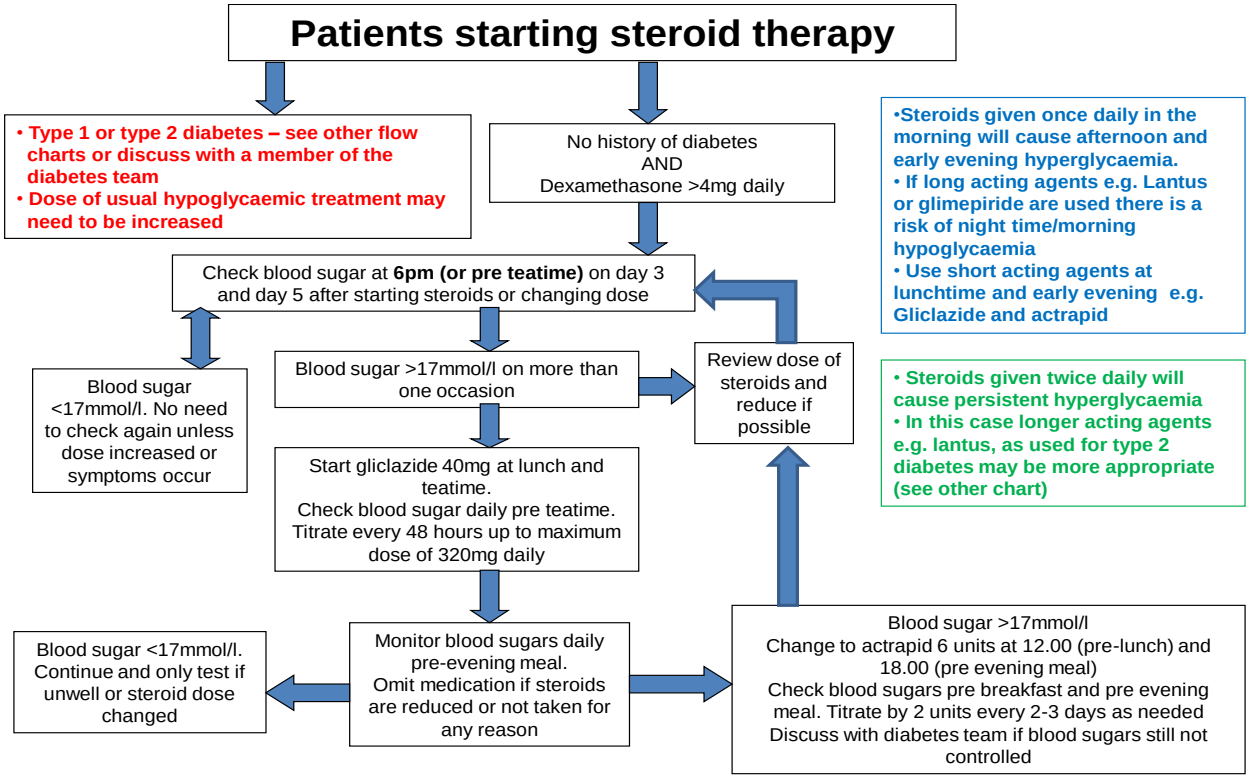

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