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	T1D	LADA	T2D	Monogenic Diabetes	GDM	T3cD (Pancreatogenic)
Pathophysiology	<p>Autoimmune destruction of pancreatic beta cells</p> <p>Clinical diagnosis ± PG and ketone levels. Urgent specialist discussion required</p> <p>It is increasingly challenging to differentiate T1D from T2D, partly due to the obesity epidemic. Often the safest strategy is to presume T1D until proven otherwise</p>	<p>LADA is essentially ‘slow-onset’ T1D</p> <p>Gradual autoimmune destruction of pancreatic beta cells. Diagnosis and management similar to T1D</p> <p>See <a href="#">Diabetes UK’s Latent autoimmune diabetes in adults</a></p>	<p>IR with relative insulin deficiency</p> <p>T2D is usually diagnosed when HbA<sub>1c</sub> ≥48 mmol/mol. If use of HbA<sub>1c</sub> is inappropriate (e.g. pregnant women, genetic variants [HbS or HbC trait], acute or chronic blood loss, end-stage kidney disease) then T2D is diagnosed by an FPG ≥7 mmol/l</p> <p>If asymptomatic, the diagnosis should never be based on a single abnormal HbA<sub>1c</sub> or PG level; at least one additional abnormal test is essential</p>	<p>Genetic mutation leading to diabetes. Most common is MODY</p> <p>See <a href="#">diabetesgenes.org</a> for diagnosis guidance</p>	<p>Impaired glucose tolerance in pregnancy due to pancreatic beta-cell dysfunction on background of IR</p> <p>NICE NG3<sup>1</sup> diagnostic criteria: FPG ≥5.6 mmol/l or 2-hour PG post 75 g OGTT ≥7.8 mmol/l, i.e. much lower than the diagnostic criteria for non-pregnant individuals</p> <p>Some areas use FPG levels ≥5.1 mmol/l, as any degree of hyperglycaemia in pregnancy increases the risk of both adverse fetal and maternal outcomes</p>	<p>Diabetes associated with disease, trauma or surgery of the exocrine pancreas</p> <p>Causes include acute and chronic pancreatitis, pancreatic surgery, cystic fibrosis, haemochromatosis and pancreatic cancer</p> <p>See <a href="#">Pancreatic Cancer Action’s information on T3cD</a></p> <p>Often misdiagnosed as T2D</p>
Age at Diagnosis	Usually <25 years but can occur at any age	<p>Can occur at any adult age</p> <p>Often initially mistaken for T2D</p>	Both adults and children at any age	MODY onset often during 2nd to 5th decades and usually <45 years	<p>Can occur in any women of child-bearing age</p> <p>Women with GDM have a nearly 10-fold higher risk of developing TD2<sup>2</sup></p> <p>Follow up after delivery: women require lifelong annual HbA<sub>1c</sub> (NICE NG3)<sup>1</sup></p>	<p>Both adults and children at any age</p> <p>Exclude pancreatic cancer in those &gt;60 years (NICE NG12)<sup>3</sup> or &gt;55 years (<i>Scottish referral guidelines for suspected cancer</i>)<sup>4</sup> with new-onset diabetes and unexplained weight loss</p>
Weight at Diagnosis	<p>Usually underweight but can occur at any weight</p> <p>Marked weight loss common</p>	Variable	Usually overweight	Variable	RF for GDM include overweight/obesity but baseline weight can be variable	Variable
Family History of Diabetes	Infrequent (5–10%)	Variable	Frequent (75–90%)	<p>Multi-generational</p> <p>MODY is AD</p> <p>Strong FH of diabetes (any type) involving two or three consecutive generations may point towards a diagnosis of MODY</p>	FH of diabetes is an important RF for GDM	<p>Variable</p> <p>Haemochromatosis and CF are AR</p>
History of Autoimmune Disease	Often personal or FH, e.g. thyroid and coeliac disease	Variable	Variable	Variable	Variable	<p>Variable but often PEI present, e.g. diarrhoea and steatorrhoea, abdominal discomfort, flatulence and bloating</p> <p>Check stool sample for faecal elastase-1. Low levels suggestive of PEI</p>
Pancreatic Autoantibodies	Present	Present	Absent	Absent	Absent	Absent
C-peptide Levels	Low/absent	Initially normal then low/absent	Normal to high	Normal	Normal to high	Low
Insulin Sensitivity	Normal when treated	Normal when treated	Reduced	Normal (maybe reduced if obese)	Reduced	Compensatory increase in peripheral insulin sensitivity
Insulin Requirements	Immediate; specialist input urgently required	Latent; months to years	Variable	Variable	Variable	<p>Variable</p> <p>Much more likely to need insulin within 5 years of diagnosis</p>
Risk of DKA	High	Low initially but high once insulin-deficient	Low but euglycaemic DKA is a rare side-effect of SGLT2i. See the <i>Guidelines Primary Care Hack, What Next After Metformin?</i>	Low	Low	Low but hypoglycaemia is common and can be prolonged

### Commonly Used Drugs That Can Induce Hyperglycaemia or Cause Diabetes

- Corticosteroids e.g. prednisolone, dexamethasone (see Useful Resources for more information)
- Thiazide diuretics e.g. bendroflumethiazide, indapamide
- Beta-blockers e.g. atenolol, propranolol
- Antipsychotics e.g. olanzapine, quetiapine, risperidone
- Statins—especially higher potency statins.

### Useful Resources

- Barker et al: [Practical guide to glucocorticoid induced hyperglycaemia and diabetes](#)
- Joint British Diabetes Societies for Inpatient Care: [Management of hyperglycaemia and steroid \(glucocorticoid\) therapy](#)
- Diabetes UK: [Steroid-induced diabetes](#)

**Abbreviations**  
AD=autosomal dominant; AR=autosomal recessive; CF=cystic fibrosis; DKA=diabetic ketoacidosis; FH=family history; FPG=fasting plasma glucose; GDM=gestational diabetes mellitus; HbA<sub>1c</sub>=haemoglobin A<sub>1c</sub>; HbC=haemoglobin C; HbS=haemoglobin S; IR=insulin resistance; LADA=latent autoimmune diabetes in adults; MODY=maturity onset diabetes of the young; NG=NICE Guideline; OGTT=oral glucose tolerance test; PEI=pancreatic exocrine insufficiency; PG=plasma glucose; RF=risk factor(s); SGLT2i=sodium-glucose cotransporter-2 inhibitors; T1D=type 1 diabetes; T2D=type 2 diabetes; T3cD=type 3c diabetes.