Elevated prevalence of clinically significant chronic liver disease among older, community-dwelling type 2 diabetes patients (Morling JR et al)

Data over 6 years from 939 participants in the Edinburgh Type 2 Diabetes Study showed that 36 patients had new or existing chronic liver disease, including 35 (3.7% overall) with cirrhosis, 9 (1.0%) with hepatocellular carcinoma and 11 (1.2%) with oesophageal varices. Only 7/15 subjects with incident chronic liver disease during the follow-up were identified by an extensive liver assessment.

Abnormal liver enzymes predicted chronic liver disease (IRR 5.7, 95% CI 2.0 to 16.0, p=0.001), but hepatic steatosis did not. Systemic inflammation, steatohepatitis and hepatic fibrosis were also associated with developing incident chronic liver disease. The incidence of chronic liver disease in this type 2 diabetic population was higher than for the general population and we need to identify patients at risk to facilitate timely intervention and follow-up.

The proteomic classifier CKD273 is distinguished between different severities of renal dysfunction in type 2 diabetes albuminuria (Currie G et al)

This study involved 45 type 2 diabetes patients (15 each with normoalbuminuria, microalbuminuria and diabetic nephropathy). The groups were similar for age and BP and were similarly at high cardiovascular risk, based on Framingham scores, ASSIGN scores, Alx@75 and carotid intima-media thickness (the last two were independent of eGFR). Nevertheless, mean CKD273 differed (p=0.002) between normoalbuminuria (−0.17 ± 0.37), microalbuminuria (0.42 ± 0.37) and diabetic nephropathy (0.77 ± 0.43), without significant correlation with measures of vascular damage. Thus, CKD273 distinguished between categories of diabetic renal dysfunction independently of vascular phenotype.

A case of devastating myopathy in a statin-treated type 2 diabetes patient (Whincup C et al)

A type 2 diabetes patient presented to Accident and Emergency with a 3-month history of worsening myalgia, generalised weakness, fatigue, fever, night sweats, dysphagia and 4 kg weight loss, previously treated as a viral illness but starting soon after commencing simvastatin. He had markedly reduced power in the proximal muscles bilaterally with normal power in peripheral muscle groups; sensation, reflexes and tone were normal. Creatinine kinase was grossly elevated (24,514 iu). Electromyography, MRI and biopsy revealed myopathic changes with an acute inflammatory response in the proximal muscles, oedema and severe acute myopathy with necrosis and myophagocytosis. He was positive for anti-HMG-Co-A antibody. A full recovery followed treatment with i.v. methyprednisolone and later high dose prednisolone followed by i.v. immunoglob-
ulin. Statin cessation may not result in symptom relief if the patient has developed autoantibodies to HMG-Co-A reductase. Patients and clinicians should be aware of the side effects of statins and weigh their risks and benefits.

How to improve your insulin prescription chart (Dashora U et al)\textsuperscript{7}
Insulin related prescription errors are common. The Joint British Diabetes Societies for Inpatient care (JBDS) organised a national competition for the ‘best in class’ insulin prescription chart (41 trusts entered). Strengths of the best charts included a ‘very practical three page fold-out’, ‘uncluttered, ‘easy to understand’, ‘inclusion of sc insulin in main drug chart’, ‘separate charts for the various IV regimes’, ‘inclusion of pre admission regimen’, ‘storage advice on chart’, ‘advice on moving insulin WITH patients and self-administration’, ‘advise on non return valves for iv insulin infusion’ and ‘instruction on making up IV infusion.’ Suggestions for improving charts included ‘prescribing by brand name’, ‘integrated blood glucose monitoring’, ‘chart for hyperkalaemia’, ‘colour coding for different charts’, ‘better guidance on when to start which regimen’, ‘better reference to units (rather than U)’ and ‘adequate space for monitoring and dose changes.’ Look out for an upcoming article on this subject in BJDVD.

Mixed early impressions of degludec (Crane J et al)\textsuperscript{8}
Twenty-four patients received degludec over 15 months (18 with type 1 diabetes, 3 with type 2 diabetes and 3 with pancreatic failure; age 19–75 y and diabetes duration 1–48 y). The intention was to address issues of adherence and hyperglycaemia (n=17), recurrent hypos (n=6; hypo frequency reduced in two patients and increased in one patient) or to improve flexibility of injection timing (n=1). Mean HbA1c was unchanged between initiation and 10 months (11.1%/98 mmol/mol at each time). For 5 patients with recurrent DKA, the frequency decreased for one and increased for another, with insufficient follow-up for the remaining three. There was one admission for severe, prolonged hypoglycaemia (suggesting a need for caution with ultra-long acting insulin in patients at increased hypo risk). Overall, there was no change in extremes of glycaemia.

Using degludec instead of U500 in a patient with severe insulin resistance due to antibodies (Acharya et al)\textsuperscript{9}
This was a case report of an Asian male, age 56 y, with diabetes for 30 y. In 2011, his U100 insulin requirement increased to 3.8 U/kg (300 U/day) and insulin antibodies were positive. He was changed to Insulin U500, titrated to 35/20/30 U/day (insulin 425 U/day). In 2013, he was changed to degludec U200 (total 62 U QD). HbA1c fell from 11% to 9%, antibody levels normalised and weight remained stable. Degludec cost £120/month vs. £200/month for U500. Our case report suggests that degludec U100/200 could be considered in patients with severe insulin resistance.

Consider primary aldosteronism as a possible cause of refractory hypertension (Hussain S et al)\textsuperscript{10}
A 61 year old, previously healthy Caucasian man presented with abdominal pain, general tiredness and refractory hypertension, despite an ACE inhibitor, calcium channel blocker and diuretics. Local gastroenterologists reported a normal oesophagogastro-duodenoscopy and colonoscopy, but a CT scan and further investigation found a 1.5cm right-sided adrenal adenoma and a raised plasma aldosterone/renin ratio, consistent with primary hyperaldosteronism (Conn’s Syndrome). Adrenal vein sampling revealed raised contralateral aldosterone levels, ruling out surgery. Spironolactone alone has maintained normotensive blood pressure, and the patient is delighted to be free of polypharmacy despite a small degree of drug-related gynaecomastia. This case demonstrates the need to further investigate the causes of hypertension, in particular unremitting hypertension despite antihypertensive polypharmacy.

Abstract titles and authors
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