The Management of Gout & CV Risk

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Identification and Management of People with MASLD and MASH in Primary Care

Medscape UK X Guidelines
Primary Care Hacks

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What Is MASLD?[1–7]

- There has been recent international consensus to rename nonalcoholic fatty liver disease (NAFLD) to improve awareness and patient identification and reduce stigma;[1] using this terminology, the EASL, the EASD, and the EASO produced an updated guideline in 2024[2]
- NAFLD is now termed **metabolic dysfunction-associated steatotic liver disease (MASLD)**[1]
  - MASLD encompasses individuals who have **hepatic steatosis and at least one cardiometabolic risk factor**[3]
- **Metabolic dysfunction-associated steatohepatitis (MASH)** replaces nonalcoholic steatohepatitis (NASH).[3] MASH is defined by inflammation of hepatocytes and carries a risk of progression to fibrosis, cirrhosis, and HCC[3,4]
- **MetALD** describes individuals with MASLD who consume more than recommended amounts of alcohol per week (defined as 3.75–7.50 units/day [30–60 g/day] in men and 2.50–6.25 units/day [20–50 g/day] in women)[2]
  - for all adults in the UK, the recommended alcohol intake is ≤14 units/week (i.e. 112 g/week, or 2.0 units/day [16 g/day]), best spread evenly over ≥3 days[5]
- **MASLD is primarily a metabolic disease heavily influenced by lifestyle factors, and is the liver's manifestation of the MetS alongside hypertension, insulin resistance and dysglycaemia, dyslipidaemia, and obesity/increased** waist circumference.[6,7]

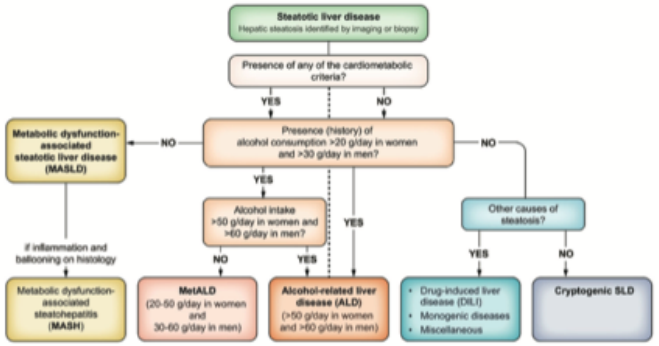
Cardiometabolic Risk Factors[2,8,9]

- BMI ≥25 kg/m² (23 kg/m² if high-risk ethnic minority) or waist circumference ≥94 cm (37.0 inches) for men (≥90 cm [35.4 inches] in men of South Asian or Chinese ethnicity, or ≥85 cm [33.5 inches] in men of Japanese ethnicity) or ≥80 cm (31.5 inches) for women of all ethnicities
- HbA_{1c} 39–47 mmol/mol, fasting plasma glucose of 5.6–6.9 mmol/l (100–125 mg/dl), or 2-hour plasma glucose during OGTT of 7.8–11 mmol/l (140–199 mg/dl), or established T2D
- BP ≥130/85 mmHg or antihypertensive drug treatment
- Plasma triglycerides ≥1.70 mmol/l or lipid-lowering treatment
- Plasma HDL-cholesterol ≤1.0 mmol/l in men, ≤1.30 mmol/l in women, or lipid-lowering treatment.

Secondary Causes of Hepatic Steatosis[2,8,10]

- Drug-induced liver injury, e.g. amiodarone, methotrexate, tamoxifen, and corticosteroids
- Endocrine disorders, such as hypothyroidism, PCOS, panhypopituitarism, or growth hormone deficiency
- Nutrient deficiency or malnutrition, such as from acute weight loss due to bariatric surgery or fasting, total parenteral nutrition, or small intestinal bacterial overgrowth
- Chronic hepatitis C virus infection.

Flowchart for SLD and Its Subcategories[2]



Note: 50 g of alcohol equates to 6.25 units, and 60 g equates to 7.5 units.

How Common and Serious Is MASLD?[2,11–13]

- MASLD is now the most common liver disorder in Western countries, and has been estimated to affect up to 30% of adults in the UK[11,12]
  - MASH has been estimated to affect up to 5% of the UK population[11]
- MASLD (specifically progressive MASH) is the fastest growing indication for liver transplantation in Western countries[13]
- MASLD is also associated with an increased prevalence and incidence of CVD[2]
  - CVD is a more common cause of death than liver disease in MASLD[2]
- MASLD is highly prevalent in people living with T2D.[2]

Screening for MASLD in Primary Care[2,14]

- The appearance of steatosis on abdominal USS is operator-dependent and a normal USS does not rule out MASLD[14]
- Consider case-finding strategies for MASLD with liver fibrosis in those who have abnormal liver enzymes, cardiometabolic risk factors, and/or incidental radiological signs of hepatic steatosis[2]
- The EASL, the EASD, and the EASO recommend looking for MASLD with liver fibrosis in individuals with one or more of the following:[2]
  - T2D
  - abdominal obesity and ≥1 additional metabolic risk factor
  - abnormal liver blood test results.

Useful Resources

- The British Liver Trust:
  - *Coffee consumption and the liver—the potential health benefits*
  - *MASLD, NAFLD, and fatty liver disease*
- The EASL:
  - *Non-alcoholic fatty liver disease (NAFLD): how you can reduce the risk for your liver and for other health issues?*

Interpreting Liver Blood Tests in Primary Care

Medscape UK X Guidelines
Primary Care Hacks

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LBTs have traditionally been referred to as liver function tests (LFTs). However, typical LBTs include measurement of both hepatobiliary enzymes (e.g. ALT and AST) and markers of liver function (e.g. albumin and clotting factors).[1,2] Additionally, many individuals with abnormal LBTs have normal liver function.[1,2] Therefore, to avoid confusion and over-investigation, 'LBT' is now the preferred terminology.[1,2]

Key Messages

1. **Standard LBTs usually comprise ALT, AST, ALP, GGT, total bilirubin, and serum albumin**
2. **Abnormal LBTs are very common in primary care!**
3. **LBTs should only be checked when specifically indicated by the clinical situation**
4. **Interpretation of abnormal LBTs should be individualised and in clinical context**
5. **Abnormal LBTs are likely to remain abnormal on repeat testing**
6. **Common patterns of abnormal LBTs are often more helpful than individual markers**
7. **GGT is useful in determining whether raised ALP is of bone or liver origin**
8. **Isolated raised bilirubin is commonly caused by Gilbert's syndrome, but haemolysis should be excluded**
9. **Always consider a possible pharmacological cause for abnormal LBTs**
10. **Liver enzymes are a poor guide to the development of ALD.**

1. Commonly Requested LBTs[1,3–9]

Test	Notes
ALT	• ALT level varies with age, gender, ethnicity, BMI, illness, and exercise • Recent consensus suggests that the current ULN for ALT is too low; updated EASL–EASD–EASO guidance on MASLD[11] suggests that an individual has elevated ALT if >33 U/l in males and >25 U/l in females (usual normal range 10–50 U/l in both men and women).
AST	• Not as liver-specific as ALT; however, in ALD, AST is a more sensitive marker of liver injury than ALT • May be elevated in MI or myositis • The **De Ritis/AST:ALT ratio** may be useful in elevated aminotransferase levels, as most causes of liver injury are associated with a greater increase in ALT than AST. As a general guide: o **AST:ALT <1** (i.e. AST<ALT) is suggestive of MASLD, chronic viral hepatitis B or C, or acute hepatocellular injury o **AST:ALT ≥2** is associated with ALD, cirrhosis (e.g. in MASH), drug-induced liver injury, and primary liver malignancy o **AST:ALT ≥5** warrants suspicion of possible extrahepatic causes (e.g. MI, myositis), particularly if ALT levels are normal.
ALP	• ALP is elevated in **cholestatic liver disease** (e.g. PBC, drug-induced liver injury), **extrahepatic biliary obstruction** (e.g. gallstones, pancreatic cancer), **bone disease** (e.g. bony metastases, vitamin D deficiency, Paget's disease, bone fractures), **renal osteodystrophy**, and **hepatic congestion caused by right-sided HF** • ALP is also higher in pregnancy because of placental production, and in adolescence because of increased bone turnover.
GGT	• **Raised by multiple factors**, particularly excessive alcohol consumption, obesity, and various drugs (e.g. paracetamol. phenytoin, sodium valproate) • Mild elevations are nonspecific, and isolated increases rarely indicate liver disease • Can also be raised in a range of nonhepatic conditions (e.g. COPD or CKD), and for several weeks after acute MI • Despite its low specificity for liver disease, **GGT is one of the best predictors of mortality in those with established liver disease** • **GGT is useful in determining whether raised ALP is of bone or liver origin** (see *7. Interpreting Raised ALP*).
Bilirubin	• Initial testing usually reports **total bilirubin** (including both unconjugated and conjugated fractions) • **Unconjugated hyperbilirubinaemia** is increased primarily in **Gilbert's syndrome and RBC breakdown (i.e. haemolysis)** (see *8. Isolated Raised Bilirubin*) • **Conjugated hyperbilirubinaemia** is usually caused by **impaired liver processing or bile flow**, e.g. from hepatitis, drug-induced cholestasis, or biliary obstruction (e.g. by gallstones or malignancy).
Albumin	• Serum albumin is a **marker of synthetic liver function**; levels are usually reduced in liver failure but may still be normal in severe acute liver damage, as the half-life of albumin in plasma is around 20 days • Levels are also reduced in sepsis, malnutrition, systemic inflammatory disorders, malabsorption, nephrotic syndrome, GI protein loss, acute infection, and HF (all subtypes) • In the absence of other abnormal LBTs, low serum albumin is unlikely to be of liver origin.
PT and INR	• In the presence of significant liver injury (usually >70% loss of synthetic function), production is reduced and PT prolonged/INR raised • Prolonged PT/raised INR can also be caused by warfarin therapy, or by vitamin K deficiency in fat malabsorption or chronic cholestasis • In the presence of otherwise normal LBTs, prolonged PT is unlikely to be of liver origin.

Platelet reduction is also an indicator of advanced liver disease; this is the result of a multifactorial mechanism involving bone marrow suppression, hypersplenism (secondary to portal hypertension), and subsequent splenic sequestration.

Note: the standard set of LBTs differs between areas. The BSG recommends assessing bilirubin, albumin, ALT, ALP, and GGT when first investigating potential liver disease, with an FBC *'if not already performed within the previous 12 months'*.[1]

Abbreviations
AAC=Accelerated Access Collaborative; **AIH**=autoimmune hepatitis; **ALD**=alcohol-related liver disease; **ALP**=alkaline phosphatase; **ALT**=alanine aminotransferase; **AMA**=antimitochondrial antibody; **ANA**=antinuclear antibody; **AST**=aspartate aminotransferase; **AUDIT**=Alcohol Use Disorders Identification Test; **BALLETS**=Birmingham and Lambeth Liver Evaluation Testing Strategies; **BMI**=body mass index; **BSG**=British Society of Gastroenterology; **CKD**=chronic kidney disease; **CMV**=cytomegalovirus; **COPD**=chronic obstructive pulmonary disease; **CVD**=cardiovascular disease; **DMARD**=disease-modifying antirheumatic drug; **EASD**=European Association for the Study of Diabetes; **EASL**=European Association for the Study of the Liver; **EASO**=European Association for the Study of Obesity; **EBV**=Epstein–Barr virus; **FBC**=full blood count; **FIB-4**=Fibrosis-4; **GGT**=gamma-glutamyl transferase; **GI**=gastrointestinal; **HAV**=hepatitis A virus; **HbA_{1c}**=glycated haemoglobin; **HBV**=hepatitis B virus; **HCV**=hepatitis C virus; **HEV**=hepatitis E virus; **HF**=heart failure; **IBD**=inflammatory bowel disease; **INR**=international normalised ratio; **LBT**=liver blood test; **LDH**=lactate dehydrogenase; **LFT**=liver function test; **LKM**=liver kidney microsomal; **MASH**=metabolic dysfunction-associated steatohepatitis; **MASLD**=metabolic dysfunction-associated steatotic liver disease; **MI**=myocardial infarction; **NG**=NICE Guideline; **NSAID**=nonsteroidal anti-inflammatory drug; **OTC**=over-the-counter; **PBC**=primary biliary cholangitis; **PSC**=primary sclerosing cholangitis; **PMH**=past medical history; **PT**=prothrombin time; **PTH**=parathyroid hormone; **RA**=rheumatoid arthritis; **RBC**=red blood cell; **RCGP**=Royal College of General Practitioners; **SMA**=smooth muscle antibody; **T2DM**=type 2 diabetes mellitus; **UGT**=uridine diphosphate glucuronosyltransferase; **ULN**=upper limit of normal; **USS**=ultrasound scan; **U&E**=urea and electrolytes; **WBC**=white blood cell.

2. Contextual Interpretation of Results[1,4,5,10]

- LBTs are regularly checked for unexplained or nonspecific symptoms, almost as a measure of wellbeing
  - o in this context, >20% will have abnormal LBTs, and most of these individuals will not have significant liver disease[10]
- Moreover, the degree of LBT abnormality does not always correlate with disease severity
  - o LBTs may be normal even in advanced liver disease, and are often abnormal in the absence of significant underlying liver disease
- Therefore, interpreting LBTs in isolation is not helpful when diagnosing or ruling out liver disease, and additional history-taking, examination, and/or investigation is usually required (see *4. History, Examination, and Screening* and *5. Further Investigations and Repeat Testing*)
- Review previous LBTs (if available) and assess trends, e.g. mild fluctuation or progressive increase.

3. Indications to Check LBTs[1,4,9,11–17]

- Indiscriminate testing of LBTs in response to nonspecific symptoms that are **not** suggestive of liver disease is likely to lead to unnecessary patient concern, further testing, and investigation
- Opportunistic testing of LBTs is not recommended for asymptomatic people without risk factors for liver disease.

Main Indications for Checking LBTs

- **Nonspecific symptoms suggestive of liver disease**, e.g. fatigue, nausea, or loss of appetite
- **Evidence of chronic liver disease**, e.g. symptoms or signs of portal hypertension, cirrhosis, or liver failure (including ascites, peripheral oedema, hepatosplenomegaly, and spider naevi)
- **Conditions associated with an increased risk of developing liver disease**—including coexisting autoimmune disease, e.g. RA and coeliac disease (increased risk of AIH), and IBD (around 10% risk of comorbid PSC)
- **Monitoring of potentially hepatotoxic drugs** (see *9. Pharmacological Causes*)—various drugs are associated with liver disease and may require LBT monitoring. Notably:
  - o **statin monitoring—statins can cause a transient rise in liver aminotransferases but do not cause liver disease;** they are likely to be beneficial in MASLD (note: **CVD is a more common cause of death than liver disease in MASLD**—see the Primary Care Hack on MASLD/MASH) and are associated with reduced primary liver cancer
    - – current monitoring of LBTs for statins is unnecessary and costly
    - – a single baseline ALT is all that is required—if this is <3x ULN, commence the statin and repeat ALT only if clinically indicated (note: NICE[18] and NHS AAC[19] guidelines do recommend further measurements of ALT/AST as part of early statin monitoring)
  - o **DMARD monitoring:**
    - – discussion with the specialist team and withholding of therapy may be warranted if ALT and/or AST >100 U/l, or an unexplained reduction in albumin <30 g/l
- **Family history of liver diseases**, e.g. haemochromatosis or Wilson's disease (both autosomal recessive disorders); this may warrant more specific, relevant tests
- **Suspected alcohol misuse and dependence**—to identify physical health complications, e.g. liver inflammation and injury (elevated ALT/AST) and portal hypertension (platelet reduction), and to assess liver synthetic function (PT/INR); see *10. Alcohol-related Liver Disease*
- **Suspected acute or chronic viral hepatitis** (e.g. HBV, HCV, HAV, CMV, EBV)—standard LBTs in addition to hepatitis serology
- **Suspected primary or secondary liver malignancy on examination**, in addition to an urgent, direct-access USS
- As part of **screening for MASLD** in the presence of its cardiometabolic risk factors and/or features of the metabolic syndrome, and/or when hepatic steatosis is found incidentally on USS (see the Primary Care Hack on MASLD/MASH).

4. History, Examination, and Screening[1,3,4,14,18,19]

- Review current clinical status, PMH and comorbidities, family history, travel history, sexual history, occupation, tattoos, prescribed and/or OTC medications, herbal remedies, illicit drug use, and alcohol intake
- Ask about specific signs and symptoms (e.g. jaundice, arthralgia, weight loss, abdominal pain, pruritis, skin hyperpigmentation), as well as tick bites and muscle injury
- Consider assessing BMI and waist-to-height ratio[18], and examining for jaundice, anaemia, ascites, hepatosplenomegaly, and other signs of chronic liver disease (e.g. spider naevi, palmar erythema, gynaecomastia, asterixis)
- Screen for MASLD and features of the metabolic syndrome (see the Primary Care Hack on MASLD/MASH).

5. Further Investigations and Repeat Testing[1,4,9,20,21]

- **Continually repeating LBTs to see whether they normalise is usually an inefficient strategy in primary care**, and is generally only appropriate if transient causes are suspected in the clinical context (e.g. simple viral illness or suspected drug-induced cause)
  - o the BALLETS study (2013)[21] found that 84% of abnormal LBTs in primary care remained abnormal on retesting 1 month later, and 75% were still abnormal after 2 years
- Early additional investigation should be considered, informed by LBT results (see Figure 1)
- **Consider an early targeted liver screen, irrespective of level or duration of abnormal LBTs**. Tests to consider are listed to the right.

Tests to consider in a targeted liver screen include:

- HbA_{1c}
- lipid profile
- USS
- coeliac screen
- iron studies
- autoimmune profile (anti-SMA, AMA, anti-LKM antibodies, ANA)
- immunoglobulins
- HBV/HCV serology
- HIV
- TFTs
- alpha-1 antitrypsin
- FIB-4, if MASLD is suspected
- serum or urine copper/caeruloplasmin (if family history of Wilson's disease, and/or aged <45 years)
- if acute hepatitis is suspected, also consider HAV, HEV, CMV, and EBV serology.

6. Common Patterns of Abnormal LBTs[1,3–5,20]

- The pattern of abnormal LBTs is often more informative than individual LBT changes, especially if there is no obvious underlying cause
- The magnitude of LBT abnormality does not necessarily correlate with clinical significance, but as a general guide LBTs <2–3x ULN are considered borderline/mild
- Look for the predominant pattern of enzyme alteration (see table, right).

Pattern	ALT/AST	ALP/GGT	Bilirubin	Notes
Hepatocellular	↑	↔	↑ or ↔	• Causes include MASLD, ALD, viral hepatitis, AIH, and drug-induced liver injury (e.g. paracetamol overdose) • Consider targeted further investigation and USS (see Figure 1).
Cholestatic/ obstructive	↔	↑	↑ or ↔	• Causes include PBC, PSC, biliary obstruction (e.g. from gallstones, strictures, malignancy), drug-induced liver injury, and hepatic congestion (e.g. secondary to HF) • Consider USS and targeted further investigation (see Figure 1).
Mixed hepatocellular/ cholestatic	↑	↑	↑ or ↔	• Often seen in the context of medication use (see *9. Pharmacological Causes*).
Isolated raised bilirubin	↔	↔	↑	• Often suggestive of Gilbert's syndrome, but haemolysis should also be excluded (see *8. Isolated Raised Bilirubin*).
Failure of liver synthetic function	• Jaundice, low albumin, and prolonged PT/INR • See Figure 1.			

↑ = Raised ↓ = Lowered ↔ = Unchanged

7. Interpreting Raised ALP[1,3,4,22]

- **If ALP is raised, check GGT:**
  - o if GGT is normal, think 'bone' origin and consider investigations such as calcium, phosphate, magnesium, PTH, U&E, and vitamin D levels (see the Primary Care Hack on abnormal calcium levels)
    - – if there is no obvious cause, consider bony metastases
  - o if GGT is high, think 'liver' origin and consider a targeted liver screen and USS (see Figure 1)
- **ALP isoenzyme electrophoresis** can also be requested to determine the source of a raised ALP, if unclear
- Consider checking **AMA** and **ANA** if raised ALP of liver origin persists, to exclude PBC
  - o PBC is much more common in women and in those aged >50 years, and often presents with intense, widespread itch.

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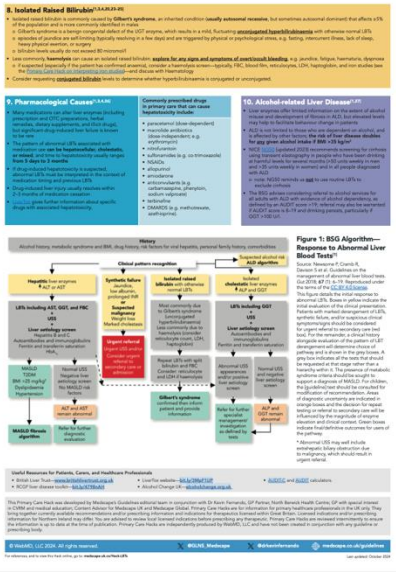

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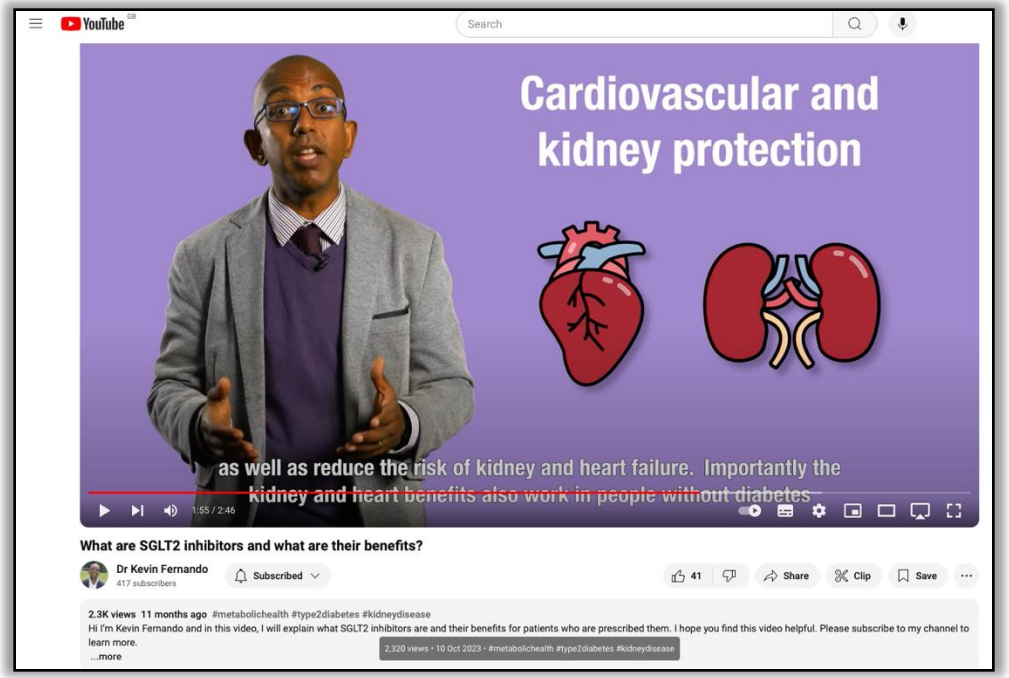
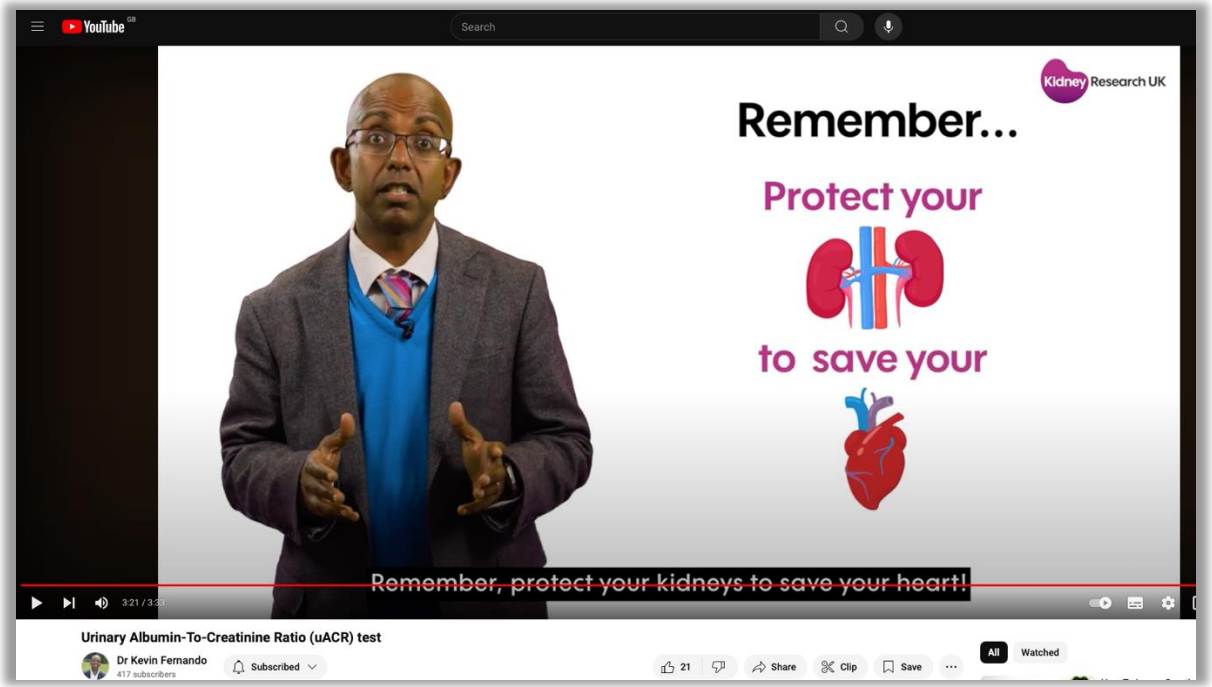
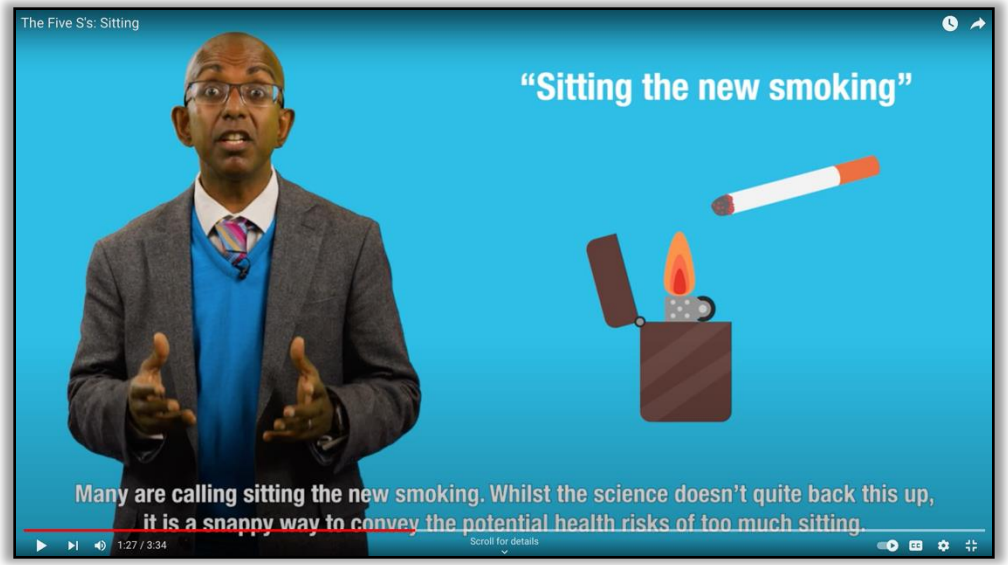






YouTube @DrKevinFernando

Henry

Age	58
History	Presents to duty surgery with a very sore & red left big toe. He's never had this before
PMH	Hypertension
Examination	Clear evidence of podagra
BP	137/84
eGFR	>60 mL/min/1.73m²
Current Medication	Lisinopril 20mg od & Bendroflumethiazide 2.5mg od
Social history	Marketing. Non-smoker but enjoys alcohol regularly

🎯What do we offer Henry for his acute attack of gout?

- What lifestyle advice should we offer Henry?
- When do we offer him urate-lowering therapy and what options are available?
- What target serum uric acid should we aim for?
- Should we review his medications?
- What are the implications for Henry's future CV risk?

Management of Gout

NICE NG219 2022, BMJ 2022

- Gout is increasing in prevalence, incidence and severity yet remains under-diagnosed and under-managed by both HCPs and patients

- Commonest inflammatory arthritis

- A change in thinking…
  - From troublesome recurrent condition to chronic inflammatory arthritis
  - Ongoing crystal deposition leads to joint destruction and long-term pain & disability

🎯 What do we offer Henry for his acute attack of gout?

- **Clinical diagnosis** - exclude septic arthritis, pseudogout (wrist & knee)
  - Uric acid often falls during an acute attack so of no diagnostic value
  - Consider joint aspiration & microscopy if diagnosis uncertain

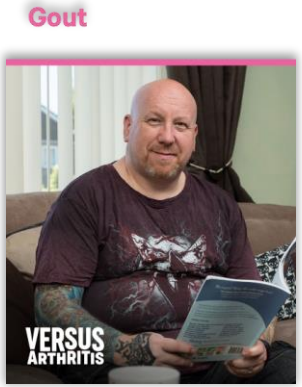
- Treat acute attacks early
  - Advise rest, ice pack & elevation

- Treatment choice should be guided by co-morbidities, current therapies, renal function & patient preference

  - **1st line**: Full strength NSAID+/-PPI **OR** colchicine 500mcg bd-qds maximum dose 6mg (12 doses) (reduce dose if eGFR<50 or short course) **OR** oral steroids (prednisolone 30-35mg od for 3-5 days)

  - Consider intra-articular or intramuscular steroids if NSAIDs and colchicine are contraindicated, not tolerated or ineffective e.g. IM methylprednisolone 80-120mg

🎯 What lifestyle advice should we offer Henry?



- **All patients with gout should be given verbal & written information**
- https://versusarthritis.org/about-arthritis/conditions/gout/
- UK Gout Society www.ukgoutsociety.org/
- Weight management
- DASH diet

- Reduce intake of high purine food e.g. red meat, game (venison), offal (liver & kidney) seafood, oily fish & shellfish, foods rich in yeast extract (e.g. Marmite)
- Vitamin C, skimmed milk & low-fat yoghurt can help. Cherries & cherry extract also helpful particularly alongside allopurinol

- Avoid excess alcohol especially beers and spirits

- Main adequate hydration

🎯 When do we offer him ULT and what options are available?

- **Offer ULT using a treat-to-target strategy** to people with gout (**1st or subsequent flare**) who have:

  – Recurrent or troublesome flares
  – CKD stages G3-5
  – Diuretic therapy
  – Tophi
  – Chronic gouty arthritis

- ULT often life-long treatment

🎯 When do we offer him ULT and what options are available?

- ULT is best delayed until acute inflammation & pain has settled (usually at least 2-4 weeks). If more frequent flares can be started during a flare
  - Check SUA after 4-6 weeks
  - **Do not stop ULT during acute attacks**

- **1st line: Allopurinol** or **feboxustat** considering co-morbidities & preferences
  - Allopurinol 1st line in people with gout & CVD
  - Consider switching if SUA target not achieved or 1st line rx not tolerated

- Allopurinol: 100-900mg daily (lower if eGFR<60)
  - Increase 4-weekly by 100mg
- Feboxustat: 80-120mg daily
  - Safe in renal impairment
  - in patients with pre-existing major cardiovascular diseases, febuxostat therapy should be used cautiously, particularly in those with evidence of high urate crystal and tophi burden or those initiating urate-lowering therapy (MHRA 2023)
- Offer **gout prophylaxis** for up to 6 months to prevent acute gout
  - Colchicine 500mcg od-bd or low dose NSAID with PPI cover or low dose steroid if colchicine CI, not tolerated or ineffective

🎯 What target serum uric acid should we aim for?

- **Treat to a SUA target of <360µmol/l** to prevent further crystal formation & to dissolve existing crystals

- **Consider a lower target < 300µmol/l if:**
  - Have tophi or chronic gouty arthritis
  - Continue to have ongoing frequent flares despite SUA < 360µmol/l

- Annual assessment of SUA

🎯 Should we review his medications?

- Yes!
  - Switch diuretic to an alternative class of antihypertensive
  - **Losartan and CCBs are better options as they have uricosuric properties**
  - Also, fenofibrate and statins
  - Continue low dose aspirin

- SGLT2i's reduce SUA in people living with T2D

- Bempedoic acid can increase SUA and gout incidence

🎯 What are the implications for Henry's future CV risk?

- **Gout is strongly associated with an increased risk of a wide range of CVD**
  - This association persists even after adjusting for traditional cardiovascular risk factors
  - Temporal association with flares: gout flares may signal a short-term increased risk for heart attack or stroke, especially in the 2m following an acute episode

- **Monitor & manage CV risk factors aggressively in people with gout**

- **Assess CV risk e.g. using QRISK3-2018**
  - Discuss life story & consider lifestyle choices
  - Review CV risk factors, BP, pulse etc.
  - Consider other bloods e.g. HbA1c, lipids, FIB-4

Thank-you for listening. Any questions?

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