



CASE STUDY 29:

Fatty liver disease diagnosed from incidental finding of mildly abnormal liver function tests

James, 55, is a retired accountant who has booked a short appointment at his regular general practice for a review of his type 2 diabetes.

His GP had organised for the routine blood tests required to be performed a week before the appointment. By inadvertently ticking E/LFT on the request form instead of U and E, she included liver function tests among these.

The GP is running half an hour behind schedule but still makes time to follow her usual routine of checking the patient notes, including those from the last consultation, plus recent investigations and correspondence, before calling James to the room.

From the diabetes perspective, James is stable and remains at or near targets for renal function, HBA1c and lipids. This would usually indicate that the consultation is likely to be quite straightforward and involve mostly congratulating the patient on his efforts to control the diabetes and general encouragement to keep up the good work and so on.

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But there are raised values for AST of 88 U/L (normal range for lab 5-40 U/L) and ALT of 65 U/L (normal range for lab 7-56 U/L) among otherwise normal LFT values. The GP notices that there were similar levels on the test at the last review. These appear not to have been acted on.

Sadly, what might have been a quick and straightforward routine diabetes review and a chance to get the session back on time is now going to be more complicated. But preparing as much as possible before calling the patient into the room will help make covering the extra agenda more efficiently and with more GP confidence.

Before calling James into her room, the GP opens up HealthPathways Melbourne, writes "abnormal LFTs" in the search bar and immediately spots and opens the suggested pathway in the hit list: the [Abnormal Liver Function Tests pathway](#).

She quickly scans it to revise risk factors, symptoms and signs of the main serious liver disease scenarios of relevance here – acute hepatitis, cirrhosis, and acute liver failure.

Having done so, she is ready to assess the patient and rule out immediate dangerous disease from clinical history and arrange diagnostic investigations to identify the cause of the abnormality. Checking the pathway further she notes point three which details initial investigations advised.

3. Order investigations:

- Obtain historic LFTs from multiple points in time or order serial LFTs over 3 to 6 months (this is important to show progression in abnormal LFTs over time).
- Arrange further tests:
 - FBE – low platelets in portal hypertension, pancytopenia when severe
 - Electrolytes, urea, and creatinine – low albumin indicates impaired synthetic function
 - HbA1c
 - Lipid profile
 - INR and PT – indicates impaired synthetic function

At management point four, she notices details of further possible causes and investigations for the specific abnormal LFT picture.

4. Arrange further investigations based on pattern of raised LFTs:

- [Isolated raised GGT](#)
- [Isolated raised ALP](#)
- [Isolated raised bilirubin](#)
- [Cholestatic pattern](#) – indicated by increase in ALP and GGT compared to ALT and AST
- [Hepatocellular pattern](#)[^] – indicated by increase in ALT and AST (transaminases) compared to ALP and GGT

Hepatocellular pattern

- Consider [hepatic causes](#).
- Arrange [investigations for liver disorders](#).

She opens the two drop-down panels in the hepatocellular pattern section.

Hepatic causes

- Common causes:
 - [Metabolic-associated fatty liver disease \(MAFLD\)](#) including non-alcoholic steatohepatitis (NASH)
 - [Type 2 diabetes](#)
 - Alcohol-related liver disease
 - [Coeliac disease](#)
 - Viral causes – hepatitis A, B, C, cytomegalovirus, Epstein-Barr virus
 - Medications
 - [Haemochromatosis](#)
- Non-liver:
 - Hypothyroidism
 - [Congestive hepatopathy](#)

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Investigations for liver disorders

Arrange:

- abdominal ultrasound if not already done.
- hepatitis serology:
 - hepatitis B surface antigen (HBsAg).
 - hepatitis B surface antibody (anti-HBs)
 - hepatitis B core antibody (anti-HBc).
 - hepatitis C virus antibody (anti-HCV).
- iron studies screening for [haemochromatosis](#)

From these her strong suspicion is that James will have a diagnosis of metabolic-associated fatty liver disease or diabetes as the cause of the LFT changes. She plans to share her thinking with him and organise a USS and further blood tests.

She calls him in from the waiting room to make a start. Some of the required blood tests have already been done for the diabetic review. However, she decides from the pathway details and James's negative history for other causes listed to also check hepatitis b and c serology, iron studies and coeliac serology.

These are conditions that might have important interventions and be difficult to exclude on history and examination only.

She completes the routine diabetes check, then asks James to get the blood tests and USS done in the next few days.

She reviews the results as they arrive in her inbox.

All the blood test are negative. However, the USS shows steatosis, suggesting the diagnosis of [metabolic dysfunction - associated fatty liver disease \(MAFLD\)](#).

She looks at the [Fatty Liver pathway](#) and notes that the assessment checklist on the pathway for this disease is already substantially complete because it echoes many of the recommendations she has followed on the abnormal liver function pathway.

But at assessment point six she is encouraged to assess the stage by using the Fib-4 score calculated from age, LFTs and platelet count.

The drop-down panel here contains a link to an online calculator for determining the Fib-4 score. Subsequent dot points outline its use to decide who should go forward for formal fibrosis assessment.

Typing James's age, AST, ALT and platelet count (which is 330) into the appropriate fields yields a result of 1.82. This places him in the 1.3 to 2.7 range – for which the pathway suggests liver assessment by FibroScan is to confirm if cirrhosis is present.

The GP telephones James to discuss the results and explain the rationale for a FibroScan. Information on how to organise this (or alternative imaging) is easily obtained by following the link from the fatty liver pathway to the [Transient Elastography \(FibroScan\) Referrals pathway](#).

Fortunately, the scan identifies minimal fibrosis only, so the main ongoing care identified on the pathway should be aggressive cardiovascular risk and additional monitoring of LFTs and platelets and Fib-4 assessments.

Had higher levels of fibrosis been found, or cirrhosis identified, referral to a liver assessment clinic would have been advised. In the latter case, management advice is available on the [Cirrhosis pathway](#).

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