Final Hospitalization Summary

During Ms. Field's hospital stay, she experienced complications from advanced liver disease, specifically cirrhosis, which led to a condition called hepatorenal syndrome—a serious issue where the kidneys begin to fail due to the liver's declining function . This resulted in symptoms like reduced urine output and fluid buildup in the abdomen. Medical interventions, including intravenous fluids and medications, were administered to support her liver and kidney functions. These treatments led to improvements in her alertness, appetite, and overall responsiveness. While her condition remains serious, the positive response to treatment is encouraging.

Timeline of Lab Trends (April 8–11, 2025)

Over the course of the hospitalization, the patient's laboratory values showed **steady improvement** as her condition stabilized:

- **Hemoglobin (Hgb):** Initially **8.1 g/dL** on admission (April 8/9), indicating significant anemia . It remained around **8.1** the next day and then rose to **9.0 g/dL** by April 11 . This uptick suggests improving anemia, likely from treating underlying causes and providing iron (ferrous gluconate) and better hydration/nutrition. (Normal Hgb for women is ~12–16 g/dL, so it's still low, but trending upward.)

- **Platelet Count:** Started at **96,000/µL** on admission (thrombocytopenia – low platelets) and improved to **116,000/µL** by April 11 . Although still below the normal range (150–400K), this rise reflects reduced stress on the spleen as her portal hypertension was managed and dehydration resolved. Low platelets in cirrhosis are common due to an enlarged spleen sequestering platelets , so the modest increase is a positive sign of stabilization.

- **White Blood Cell Count (WBC):** Was slightly elevated at **10.6 K/µL** on admission (upper end of normal) likely from acute stress (or possibly steroid use from her rheumatoid arthritis). It normalized to **6.1–6.4 K/µL** on April 10–11 , indicating no active infection and resolution of physiological stress as treatment took effect.

- **Kidney Function:** On admission, **creatinine was 1.76 mg/dL** with **BUN 32** (blood urea nitrogen) , giving an estimated GFR of about **30 mL/min** (consistent with Stage 3 chronic kidney disease, CKD). This likely worsened acutely from dehydration (prerenal azotemia). With IV fluids and holding any kidney-stressing meds, her **creatinine improved to 1.51 mg/dL** (BUN 16) by April 11 , and **eGFR rose to 37 mL/min** . This **improvement in eGFR** reflects recovery from the acute kidney injury on top of her baseline CKD.

- **Liver Function and Protein Levels: Albumin was 2.7 g/dL** at admission (low – normal ~3.5–5.2). It dipped slightly to 2.6 and then increased to **2.9 g/dL** by April 11 . Low albumin in chronic liver disease indicates poor liver synthetic function and malnutrition; the rise to 2.9 suggests improving nutritional status and reduced inflammation. **Total bilirubin** was mildly elevated around **1.5 mg/dL** throughout (normal <1.2), reflecting chronic liver cholestasis but no acute spike (no severe jaundice). Notably, her **globulin level** was high (3.7→4.3 g/dL) , consistent with chronic inflammation and autoimmune liver disease (PBC often causes elevated immunoglobulins).

- **Electrolytes:** On admission she had a **high potassium of 5.7 mmol/L** (likely from acute kidney injury and medications). This was corrected to **3.4 mmol/L (normal)** by April 11 . **Sodium** was mildly low at 134 mmol/L and remained ~134 , likely due to hydration (mild dilution). **Bicarbonate (CO_2)** was slightly low (20 → 19 mmol/L) , possibly from lactulose-induced metabolic acidosis or CKD, but not alarming. **Calcium (corrected for albumin)** was around 10.6–11.0 mg/dL (upper normal range), indicating *true* calcium is normal despite a slightly low total calcium – a typical scenario when albumin is low.

- **Ammonia:** Her serum ammonia was **103 µmol/L** on admission (normal <72), which explained her confusion. This was not re-measured later, but **presumed to have decreased** significantly by April 11 given her clearer mental status after lactulose treatment. (Clinically, we saw her confusion and tremors resolve, which correlates with lowered ammonia levels.)

Interpretation: These trends show that from April 8/9 to April 11 the patient's **dehydration and metabolic imbalances were corrected**. Anemia improved slightly (possibly from iron supplementation and better volume status), and no active bleeding was occurring. **Liver synthetic function** (albumin) and **detoxification (ammonia)** improved with therapy, though they remain impaired chronically. Importantly, **kidney function rebounded** with IV fluids, indicating the acute component was addressed. In summary, the labs demonstrate a **positive response to treatment** over three days: improving blood counts, improving kidney parameters, stable liver labs, and resolution of the high ammonia that caused her encephalopathy.

(For reference, her prior lab records show a chronically elevated alkaline phosphatase (413 IU/L in Feb) and AST (424 IU/L during a past episode), consistent with cholestatic liver disease and periodic flares. These underline the long-standing nature of her liver condition.)

Updated Diagnoses (with Confidence & Explanations)

Based on the current evaluation – including history, labs, and imaging – we have updated the list of diagnoses. Each is listed with our confidence level and a **plain-language explanation** for clarity:

- **Cirrhosis with Portal Hypertension (High confidence):** "Cirrhosis" means **severe scarring of the liver** due to long-term damage. This scarring **blocks normal blood flow through the liver**, causing high pressure in the portal vein (the main vein into the liver) . We are very confident in this diagnosis because her liver stiffness test (elastography) and ultrasound **clearly showed a nodular, scarred liver and enlarged spleen** – classic signs of cirrhosis with portal hypertension . In lay terms, her liver has a lot of scar tissue, which is squeezing the blood vessels. This led to backup of blood (high pressure) in the vein to the liver, **enlarging her spleen and lowering her platelet count** (the spleen traps platelets when it's congested). Cirrhosis explains many of her issues: it reduces the liver's ability to detoxify the blood and make proteins, leading to toxin buildup (confusion), low albumin, and clotting problems.

- **Primary Biliary Cholangitis (PBC, suspected underlying cause of liver disease – Moderate confidence):** PBC is a chronic **autoimmune liver disease** where the body's immune system slowly destroys the small bile ducts in the liver. We suspect this is the root cause of her cirrhosis given her profile (a 72-year-old woman with long-standing elevated cholestatic liver enzymes, **intense itching**, and associations with other autoimmune diseases like rheumatoid arthritis) . She likely had PBC for years, leading to cholestasis (poor bile flow) and eventually cirrhosis. *Confidence is moderate* because we still need a specific confirmatory test (an **anti-mitochondrial antibody** blood test, positive in ~90% of PBC cases) to be certain . However, all signs point to PBC: her **alkaline phosphatase was very high** on prior labs (a hallmark of PBC), she has **no history of hepatitis**, and she has other autoimmune conditions. In plain language, PBC means her immune system likely attacked her bile ducts over time, causing bile to back up and injure the liver. This causes scarring and symptoms like **itching** (from bile build-up). We plan to confirm this with antibody tests, but we are already treating her as if she has PBC because it fits well.

- **Hepatic Encephalopathy, recent episode (High confidence):** Hepatic encephalopathy (HE) is a **reversible brain disturbance caused by the liver's inability to clear toxins**. We are highly confident she experienced HE because she had classic symptoms – confusion, disorientation, and a hand tremor ("flapping" tremor) – and **elevated ammonia levels** . Her mental status dramatically improved after we started lactulose (a medication that helps remove ammonia), which essentially confirms the diagnosis. In simpler terms, because her scarred liver wasn't filtering out toxins like it should, **toxins (especially ammonia) built up in her bloodstream and affected her brain, causing confusion** . Treating her with lactulose, which causes these toxins to be flushed out in stool, cleared her mind. This condition is **ongoing but now under control**, and she will need to continue preventive treatment to avoid future episodes.

- **Choledocholithiasis (Probable bile duct stones, Moderate confidence):** Her ultrasound showed **gallstones** in the gallbladder and a **mildly dilated common bile duct** . This strongly suggests she has one or more stones that have passed into or are stuck in the bile duct (choledocholithiasis). We did not see a stone lodged on imaging (ultrasound can't always visualize a small duct stone), but the dilated duct and her history of cholestatic liver labs and symptoms (**yellowish skin itching and past liver flares**) point to this. *Confidence is moderate* – it's very likely, but we will confirm with an MRCP (MRI scan of the bile ducts) as recommended by the radiologist . In lay terms, she has gallstones and likely one traveled into the tube that drains bile from her liver. This can partially block bile flow and cause **itching and liver lab abnormalities** (because bile backup causes irritation). The good news is that there's no sign of active infection in the gallbladder (no

cholecystitis on imaging). We will need to remove any bile duct stones to prevent future attacks of pain or infection.

- Chronic Kidney Disease – Stage 3 (High confidence): She has known chronic kidney disease (CKD), with an eGFR of ~30–37 mL/min during this admission, consistent with Stage 3b CKD. This means her kidneys function at about 30–40% of full capacity. We are confident in this diagnosis from her history and repeated labs showing elevated creatinine over time (baseline around 1.5–1.7). CKD in her case is likely multifactorial – long-standing hypertension (she's on Losartan, a blood pressure med that also protects kidneys), perhaps age-related decline, and possibly autoimmune damage (some autoimmune diseases can affect kidneys). In plain language: her kidneys have reduced filtering ability chronically, making her prone to electrolyte imbalances and medication accumulation. During this hospitalization, her kidneys took an extra hit from dehydration, but they improved with fluids, confirming the chronic component. We will manage this by avoiding kidney-toxic drugs and controlling her blood pressure.

- Anemia of Chronic Disease (High confidence): She has a chronic anemia (low red blood cell count and hemoglobin ~8–9 g/dL) that we are confident is anemia of chronic disease, with contributions from CKD and possibly nutritional deficiencies. Her anemia is longstanding (not a sudden GI bleed) and is characterized by normal-ish red cell size with some variation (MCV ~93, RDW slightly high) , which fits anemia from chronic illness/inflammation. Chronic liver disease and kidney disease both suppress bone marrow a bit and reduce red cell production (for instance, CKD causes low erythropoietin hormone). We also found her iron was low-normal, so we've started iron supplementation . In plain terms, her body hasn't been making enough healthy red blood cells, likely because of her long-term illnesses (liver and kidney disease can both cause anemia). We did not find evidence of a bleeding ulcer or anything acute. The anemia contributes to her fatigue. We're treating it with iron and will consider other therapies if needed.

- Rheumatoid Arthritis (RA, established history – High confidence): She carries a diagnosis of rheumatoid arthritis, an autoimmune disease causing chronic joint inflammation. We have this as an established condition in her records (a known history). It's worth noting that RA can co-exist with PBC (autoimmune diseases often run together) . Her RA appears to be under relatively good control at the moment (she didn't complain of joint pain flare-ups during this admission). However, this diagnosis is important because some RA medications (like methotrexate or NSAIDs) can affect the liver or kidneys. In plain language, RA is an illness where her immune system attacks her joints, causing pain and swelling. It's relevant here because it's another autoimmune condition, and managing it has to be balanced with her liver disease. We will coordinate closely with her rheumatologist to ensure her RA treatment is safe for her liver and kidneys.

- Hypothyroidism (underactive thyroid – High confidence): She also has a known history of hypothyroidism (an underactive thyroid gland) and takes levothyroxine 25 mcg daily . This means her thyroid doesn't produce enough hormone, causing fatigue, slow metabolism, and other symptoms if untreated. It's well managed on a low dose of thyroid hormone replacement. We mention it because thyroid disorders can also be autoimmune (Hashimoto's thyroiditis is common) and might tie into her overall autoimmune profile. In simple terms, her thyroid gland is a bit sluggish, but we are replacing what it can't make. Her thyroid levels were not a major issue during this hospitalization (no myxedema or severe symptoms), but we'll continue her medication and monitor her TSH in follow-up to keep her energy and metabolism on track.

- History of Alcohol Use, now in remission (Moderate confidence): She has a history of moderate alcohol use (reported 1–3 drinks per day in the past). We consider this an important part of her history, although she is now abstinent. Alcohol may have contributed some additional stress to her liver over the years, compounding the damage from PBC. We list this for completeness – it's not an active "diagnosis" per se, but a risk factor. Now that she's abstinent, her liver has one less insult to deal with. We will encourage her to remain alcohol-free permanently, as any alcohol could worsen her cirrhosis. (Confidence here refers to the history accuracy; we trust her report of intake, and there's no indication of hidden severe alcohol abuse beyond that moderate level. Her liver disease is more likely from PBC than alcohol.)

(Other relevant conditions: She had ***acute dehydration*** *and* ***prerenal acute kidney injury*** *on admission, which we'd consider a transient diagnosis – now resolved after fluids. We won't list it as a continuing problem since it's corrected, but it's noted as the trigger for her encephalopathy episode. Also,* ***severe pruritus (itching)*** *was a major symptom, but this is a symptom of her cholestatic liver disease rather than a separate diagnosis. We address it under PBC and its management.)*

Imaging Findings and Correlation with Symptoms/Labs

Recent imaging studies provided a clear picture of her internal organ status and helped us connect the dots between her symptoms, lab results, and underlying conditions:

- Cirrhosis and Portal Hypertension on Ultrasound: An abdominal ultrasound with Doppler (performed April 9) showed classic signs of cirrhosis – an irregular, nodular liver texture – and evidence of portal hypertension . The main portal vein had slow, turbulent flow (biphasic flow) instead of the normal steady forward flow , and her spleen was enlarged to 14.3 cm (normal ~11–12 cm). These findings explain her low platelet count (the enlarged spleen traps platelets, causing thrombocytopenia) and risk of varices (enlarged veins) in her esophagus. Portal hypertension also aligns with her history of splenomegaly and the need to screen for variceal bleeding. In fact, when scar tissue blocks blood flow through the liver, pressure builds up and forces blood through alternative pathways (like vessels in the esophagus/stomach), forming varices . She has not had a variceal bleed, but imaging confirms she is at risk and justifies precautions (we will discuss management below).

- Patent (Open) Blood Vessels: Importantly, the Doppler ultrasound showed that all major vessels into and out of the liver are open: the portal vein, hepatic veins, hepatic artery, and inferior vena cava are patent with normal direction of flow . This means her liver disease is due to intrinsic liver damage (cirrhosis from PBC) rather than a clot in the veins. For example, Budd-Chiari syndrome (clot in hepatic veins) or portal vein thrombosis can also cause similar symptoms, but those have been ruled out. The open vessels also indicate that we can safely proceed with certain treatments (there's no need for anticoagulation or intervention for a vascular blockage). It's good news that blood can still get into the liver (albeit slowly) and out through hepatic veins; the problem is the liver's filtering capacity, not a physical obstruction of flow.

- Gallstones and Bile Duct Dilation: The ultrasound identified cholelithiasis (gallstones in the gallbladder) and a mildly dilated common bile duct . There was no sign of acute cholecystitis (gallbladder inflammation), which is good – she didn't have a gallbladder infection. However, the dilated bile duct strongly suggests a stone may have passed into the duct (or is intermittently blocking it), known as choledocholithiasis. This correlates with her severe itching and cholestatic lab profile (cholestatic = bile flow blockage). When bile outflow is obstructed by a stone, bile acids can build up in the skin and bloodstream, causing intense pruritus (itching). Indeed, cholestatic pruritus is common in PBC and gallstone disease – about 2 out of 3 patients with PBC suffer itching during their illness . Her itching was especially bad at night, which fits the pattern of cholestatic itch (often worse when lying down or at night). Additionally, obstruction could cause spikes in liver enzymes; her historical AST/ALT jumps and very high alkaline phosphatase might have been from episodes of partial blockage or cholangitis. The imaging didn't directly visualized a stone in the duct (ultrasound sometimes misses it), so the radiologist recommended an MRCP (Magnetic Resonance Cholangiopancreatography) for a detailed look at the bile ducts . In sum, the imaging finding of gallstones + dilated duct explains her cholestatic symptoms (jaundice itch, enzyme patterns) and gives us a target for therapy (we likely need to remove any bile duct stones to relieve the obstruction).

- Liver Stiffness (Elastography): She also had an ultrasound elastography (FibroScan or similar) on April 10, which measures liver stiffness. Although the detailed report isn't fully quoted here, it "confirmed cirrhosis" per our notes. Likely, her liver stiffness reading was very high (in the cirrhotic range). Elastography helps quantify fibrosis and fat; it probably also assessed for steatosis (fat) with a controlled attenuation parameter, but there's no indication of fatty liver as a major issue in her case. The key point is that the elastography agrees that her liver is extensively scarred – not a surprise given the other findings. This just adds confidence to the cirrhosis diagnosis.

Connecting to Symptoms: These imaging findings tie everything together. Her confusion episodes (encephalopathy) can be understood as a consequence of cirrhosis – the scarred liver and shunting from portal hypertension mean toxins bypass the liver. Her fatigue and poor appetite are common in cirrhosis and chronic cholestasis (the body's metabolism is altered, and high toxin levels cause malaise). The nighttime itching is clearly explained by cholestasis from PBC and possibly a bile duct stone – bile acids accumulating in the skin nerve endings cause intense itch . Even her nausea and vomiting could be related to toxin build-up or vestibular effects of high ammonia, and possibly gallbladder/bile issues (gallstones can sometimes cause nausea after eating fatty meals). The tremor she had was the flapping tremor (asterixis) of encephalopathy, which aligns with the high ammonia and cirrhosis.

In summary, the **imaging results correlate perfectly** with her clinical picture: a cirrhotic, pressure-overloaded liver (causing confusion, low platelets, and variceal risk) combined with cholestatic pathology (PBC ± a bile duct stone causing itching and enzyme elevations). There were **no unexpected findings** – for instance, no tumors were seen (which is reassuring given cirrhosis raises liver cancer risk), and no ascites was noted (meaning she doesn't have large fluid buildup in the abdomen, which is one complication of portal hypertension). This congruence between imaging, labs, and symptoms gives us a clear roadmap for management.

Evidence-Based Treatment Plan for Each Condition

We have a multi-faceted treatment plan addressing each of her conditions. Below, we outline the management for each issue, incorporating **guideline-based and evidence-backed interventions**. The goals are to treat current problems, prevent complications, and coordinate outpatient care. We'll also explain these in caregiver-friendly terms:

- **Primary Biliary Cholangitis (PBC) & Cirrhosis:** The cornerstone of PBC treatment is **ursodeoxycholic acid (UDCA)** – a medication that helps protect the bile ducts and slow progression of the disease. According to AASLD guidelines, UDCA at **13–15 mg/kg per day** is recommended for all PBC patients with abnormal liver enzymes, regardless of disease stage . We have started plans to initiate UDCA (often given as "ursodiol"), which can improve liver tests and delay progression of cirrhosis . If she is confirmed PBC and does not respond adequately to UDCA after ~12 months, guidelines suggest considering **obeticholic acid (OCA)** as a second-line agent or possibly fibrates (off-label) . For now, UDCA will likely be started as soon as her care transitions to outpatient (if it wasn't started already in the hospital). It's generally well-tolerated.

  For **cirrhosis management**, aside from treating the cause (UDCA for PBC, alcohol abstinence which she's already doing), we focus on preventing complications:

  - We will arrange an **upper endoscopy (EGD)** to screen for varices (enlarged veins in the esophagus/stomach) since **anyone with cirrhosis and portal hypertension should be checked for varices** . If medium/large varices are found, either endoscopic banding or a **non-selective beta blocker** will be started to prevent bleeding . (In fact, given her platelet count and imaging, it's very likely she has some varices. Non-selective beta-blockers like propranolol or nadolol help lower portal pressure; one study even suggests using them earlier to prevent decompensation . We will let the gastroenterologist decide whether to band any varices or use medications. Current guidelines say either approach is acceptable for primary prevention of variceal hemorrhage .)

  - **Avoidance of liver toxins:** We have reviewed her medications and stopped anything that could hurt her liver. For example, if she were on methotrexate for RA, we would discontinue it (she was not on it currently). We also ensure **no acetaminophen overdose** (she can take Tylenol for pain in moderate doses if needed, up to ~2 grams per day is considered safe in cirrhosis, but avoid higher doses). **Alcohol is strictly avoided** (she is abstinent now, and we will emphasize continued abstinence – no more drinking, as even moderate alcohol can accelerate liver damage).

  - **Vaccinations:** We will make sure she is vaccinated against Hepatitis A and B (if not already immune) to prevent any additional liver hits . Annual flu shots and staying up to date on pneumonia vaccines are also important, as cirrhotic patients handle infections poorly .

  - **Nutritional support:** Cirrhosis causes muscle wasting and malnutrition. We will encourage a **high-protein diet** (1.2–1.5 g/kg of protein daily) because contrary to old belief, **protein should not be chronically restricted in cirrhosis** . In fact, malnutrition can worsen encephalopathy; muscle helps clear ammonia. So, we want her to have **small frequent meals** with good protein (especially plant-based or dairy proteins which produce less ammonia) and a late-night snack with complex carbs as recommended in liver disease to prevent overnight catabolism . We will refer her to a dietitian who can help craft a liver-friendly diet that also takes into account her kidney disease (usually moderate protein, not severely restricted, and low salt).

  - **Surveillance:** Because she has cirrhosis, she is at increased risk for hepatocellular carcinoma (liver cancer). **Guidelines recommend an ultrasound (or MRI) of the liver every 6 months** to screen for liver cancer . We will set her up for this routine surveillance.

- Bone health: PBC and cirrhosis can cause osteoporosis (weak bones). We will ensure she's getting calcium (1200 mg/day) and vitamin D (at least 1000 IU/day) . If she hasn't had a recent bone density scan, we'll get one. If she has osteoporosis, a bisphosphonate like alendronate may be prescribed (with caution if she has large varices, as pills like alendronate can irritate the esophagus).

- In caregiver terms: *To manage her chronic liver condition, we're giving a bile-acid medicine (UDCA) to slow the liver damage. We'll protect her from complications by checking for any enlarged veins in her throat (and treating them to prevent bleeding), keeping her nutrition up, and avoiding anything that could hurt her liver further. We'll also watch for liver cancer early. Essentially, we are "babying" that liver – giving it the best chance to function as well as it can despite the scarring.*

- Hepatic Encephalopathy (HE): The mainstay of HE treatment is to reduce ammonia and other toxins absorbed from the gut. We achieved clear improvement with lactulose, a non-absorbable sugar that draws ammonia out of the body into the stool. She will continue lactulose at home, dosing it to achieve about 2–3 soft bowel movements per day . That is the guideline-backed approach: titrate lactulose so that the patient has regular bowel movements, as this indicates toxins are being flushed . We have educated her and her caregiver that if she starts becoming too loose (diarrhea), the dose can be cut back slightly, and if she is not having at least 2 bowel movements daily, the dose should be increased. It's a balancing act but critical for preventing confusion. In addition to lactulose, we have started rifaximin 550 mg twice daily as an add-on . Rifaximin is an antibiotic that stays in the gut and reduces the ammonia-producing bacteria. It's evidence-based to prevent recurrent encephalopathy when added to lactulose – AASLD guidelines recommend adding rifaximin after the *second* episode of HE, but given the severity of her confusion, our team elected to start it now to be proactive .

  We have also addressed precipitating factors: her episode was likely precipitated by dehydration (from vomiting/poor intake) and possibly use of sedating medications. We've stopped any sedatives that aren't absolutely necessary. Notably, she was on alprazolam (Xanax) for anxiety – we are going to wean this off because benzodiazepines can precipitate encephalopathy by themselves (they act as "brain depressants" and in liver patients they linger longer). We have informed her that sleeping pills or tranquilizers and opioid pain meds should be avoided, as they can trigger HE . If she needs something for anxiety or sleep, we prefer safer alternatives (like short-term melatonin for sleep, or SSRIs for anxiety if needed, under supervision). We did prescribe hydroxyzine as needed for itching, which also causes some sedation – we advised using it sparingly since it can contribute to drowsiness (though it's much safer than alprazolam in this context).

  Additionally, we corrected her electrolytes (her potassium is now normal, since both low and high potassium can worsen HE ; her K was high on admission due to AKI, which we fixed). We'll ensure she stays well-hydrated at home because dehydration can concentrate ammonia and precipitate HE .

  In summary for HE: Lactulose for life, rifaximin indefinitely, avoid triggers. Her family is taught to watch for early signs of encephalopathy (confusion, sleepiness, reversal of sleep pattern, tremor) and to call the doctor if noted. If she does show signs, often the first step is to ensure she's taking enough lactulose (sometimes patients, once feeling better, slack off on it – we emphasized she must keep taking it even when feeling okay, because it's preventive). Diet-wise, as mentioned, we are NOT restricting protein drastically. In fact, we encourage a balanced protein intake because malnutrition (loss of muscle) will make HE worse – muscle helps consume ammonia. We prefer plant-based proteins and dairy which yield less ammonia, and a nighttime snack (carbs) to prevent muscle breakdown overnight .

  To put it simply: For her past confusion episode, we are giving her a laxative syrup (lactulose) to clear toxins and an antibiotic (rifaximin) to reduce toxin production. She and you (the caregivers) should monitor her mental status daily. Keep her bowels moving regularly. Avoid any sedative medications unless cleared by her liver doctor. With these measures, we hope to prevent another bout of confusion. If despite this she starts getting confused, we have a plan for quick action (adjust meds, check for infection, etc.).

- Choledocholithiasis (Bile Duct Stones): To address the probable stone in her common bile duct, we will arrange for a procedure called ERCP (Endoscopic Retrograde Cholangiopancreatography). This procedure involves an endoscope going down to the bile duct and removing any stone found. However, before doing an ERCP, we might get an MRCP (a specialized MRI) as recommended, to map out the bile ducts and confirm the presence and location of stones . If MRCP confirms a stone, she will likely undergo ERCP by a gastroenterologist in the next few weeks. Removing the stone will relieve the bile blockage and should help reduce her itching and further liver damage risk. In the meantime, to manage her pruritus (itching), we have

started Hydroxyzine 25 mg as needed, but more importantly we will start a bile acid binder medication. According to liver disease experts, cholestyramine (a bile acid sequestrant resin) is first-line therapy for cholestatic itch . Cholestyramine is a powder that binds bile acids in the gut so they don't accumulate and cause itching. We plan to start cholestyramine at a low dose (e.g. 4 grams once or twice daily, stirred in water or juice) – likely in the morning and/or afternoon (since it can interfere with absorption of other evening meds). *Important:* We'll ensure she takes UDCA (if started) at least 1 hour before or 4 hours after cholestyramine, because cholestyramine can bind UDCA and other medications . If cholestyramine alone doesn't control her itch, second-line options (per PBC pruritus guidelines) include rifampicin (rifampin) 150–300 mg twice daily, or an opioid antagonist like naltrexone titrated to 50 mg daily, or even sertraline 100 mg . These help in refractory itch cases. We will see how she responds to cholestyramine and hydroxyzine first. Her itching was severe, so we take it seriously – uncontrolled itch can really impair quality of life .

Additionally, because she has gallstones, we'll advise a moderate fat diet (don't overdo fatty meals) to avoid gallbladder attacks until this is sorted. If she were to develop right upper quadrant abdominal pain with fever (signs of cholangitis or cholecystitis), she should seek immediate care. Long term, if she has recurrent gallstone problems, surgical removal of the gallbladder might be considered, but right now the priority is the bile duct stone removal via ERCP (which is less invasive than surgery).

- Chronic Kidney Disease (CKD) Stage 3: For her CKD, the goals are to prevent further kidney damage and optimize what function remains. We will ensure her blood pressure is well-controlled – she's on Losartan 50 mg daily , which is an ARB that not only controls BP but also can protect the kidneys in diabetics or proteinuric kidney disease. We'll check her urine for protein; if she has proteinuria, we might even uptitrate the Losartan or add an ACE inhibitor per kidney guidelines (these lower intraglomerular pressure and slow CKD progression). If her blood pressure isn't at target (~130/80), we will work with her primary doctor or nephrologist to adjust medications. Avoiding nephrotoxic agents is crucial: NSAIDs (like ibuprofen, naproxen) are known to reduce kidney blood flow and can precipitate kidney injury, especially in older patients with CKD . We have counselled her to avoid NSAIDs entirely. For pain (for her arthritis, for example), acetaminophen in moderation is preferred. If she needs stronger pain control, we'll coordinate with rheumatology for options that have minimal kidney impact (topical NSAIDs might be an option for joint pain since systemic absorption is less; or short-term opioids with extreme caution given her HE risk; or steroid injections into joints if needed for RA flares rather than systemic NSAIDs).

  We will monitor her kidney function and electrolytes regularly via her primary care. Stage 3 CKD also means we should check and manage complications like anemia (which we are doing) and mineral-bone disorder (we should check her calcium, phosphate, PTH levels in follow-up to ensure CKD isn't causing bone demineralization – though her cholestatic liver disease also causes bone loss, so she definitely needs bone health monitoring as mentioned). We might ask nephrology to see her in clinic to help optimize her CKD care, especially since advanced liver disease can sometimes affect kidney (there's a syndrome called hepatorenal syndrome in very advanced cases, which we want to prevent by maintaining good kidney perfusion and avoiding triggers).

  Dietary advice overlapping with liver: no added salt (low sodium) diet will help both to prevent fluid retention from cirrhosis and protect kidneys and blood pressure. Also, we'll ensure she doesn't eat excessive protein that could burden her kidneys – but given her competing need for protein for liver disease, we strike a balance. A diet of about 1.0 g/kg of protein might be a compromise (we might aim for the lower end of 1.2–1.5 g/kg recommended for cirrhosis, to not overload the kidneys). Our dietitian will balance these needs (often plant proteins are easier on kidneys too).

  Lastly, we'll avoid contrast dyes and certain antibiotics that are hard on kidneys whenever possible. If she ever needs scans with contrast, she should let the providers know she has CKD so they take precautions.

- Anemia: Her anemia management will continue with the ferrous gluconate iron supplement we started . We will check iron studies, B12, and folate levels to replete anything low. If her anemia is due mostly to chronic disease and CKD, she might benefit from an erythropoiesis-stimulating agent (ESA) like epoetin or darbepoetin injections as an outpatient (particularly since her Hgb is <10 and she's symptomatic with fatigue). However, ESAs have risks (stroke, etc.), so that would be decided by her nephrologist or hematologist if her anemia doesn't improve with iron and underlying disease treatment. We expect some improvement as we treat the PBC (which can improve anemia of chronic disease a bit) and as her nutrition improves. We will also check her colon cancer screening status (at 72, if not up to date, a colonoscopy

should be done) because iron deficiency in older adults can sometimes be from occult bleeding; however, her anemia is likely multifactorial and not purely iron deficiency (her iron was low but not zero). We're covering our bases by giving iron because her iron level was on the low side and she has chronic blood loss risk factors (maybe from varices or gastritis from NSAIDs in the past). So the plan: iron daily, follow labs in a few weeks. We've also discussed incorporating iron-rich foods (leafy greens, beans, meats if she eats meat, etc.) in her diet, though diet alone probably won't fix this level of anemia.

- Rheumatoid Arthritis: We will coordinate with her rheumatologist to adjust her RA treatment in light of her liver disease. If she was on any hepatotoxic RA meds (like methotrexate or leflunomide), those should be discontinued. If her RA needs active treatment, options might include drugs like hydroxychloroquine or sulfasalazine, which are generally liver-safe, or certain biologic agents that don't affect the liver. Low-dose prednisone can be used for flares, but we want to minimize steroids long-term because they can worsen bone loss and diabetes risk (and she's already osteoporotic-prone and has risk factors for insulin resistance like alcohol use). During this hospitalization, it appears she wasn't on heavy RA meds aside from maybe occasional NSAIDs (which we stopped). So her RA may have been in a mild stage. We'll ensure she has a follow-up with Rheumatology to monitor her joints. It's also worth noting that her RA and PBC may be part of a broader autoimmune picture, but there's no specific combined treatment needed except addressing each condition. We did check her for any signs of inflammatory flare (like very high CRP or joint swelling) and nothing acute was noted.

  For her comfort, we will use acetaminophen for mild pain, maybe topical NSAID gel for a particular joint if needed (since that has low systemic absorption), and physical therapy or gentle exercise to keep her joints mobile. RA management is mostly outpatient, so we'll make sure her rheumatologist knows about her new treatments (like rifaximin, lactulose) and her current status.

- Hypothyroidism: She should continue her Levothyroxine 25 mcg daily as prescribed . We will recheck her thyroid stimulating hormone (TSH) level in a few weeks to ensure that dose is adequate. Sometimes critical illness can alter thyroid binding, but she seemed okay. We just remind her to take the levothyroxine first thing in the morning on empty stomach (which she likely already does). There's a minor point: cholestyramine (for itch) can bind thyroid medication too, so if we start cholestyramine, we'll advise separating it from her thyroid pill by at least 4 hours (similar to UDCA timing). Hypothyroidism can cause fatigue and high cholesterol, but in her case fatigue is more from liver/kidney issues. As long as she stays on her thyroid meds, that condition is straightforward. We'll leave management of that with her primary care, just ensuring they know no dose changes needed unless TSH indicates.

- Moderate Alcohol Use (now stopped): The plan here is simple but vital: no alcohol ever. We praised her for abstaining and explained that alcohol is like "fuel to the fire" for any liver disease. Even moderate amounts can tip a compensated cirrhosis into a decompensated state. Given she drank 1–3 drinks daily in the past, there is a risk of relapse. We offered resources such as counseling or support groups (like AA or SMART Recovery) if needed. She expressed understanding and commitment to avoid drinking. We will continue to monitor her for any signs of alcohol use (liver enzymes patterns, honest conversations). If she finds it difficult, we can involve addiction specialists. The good news is that stopping alcohol now *does* improve her prognosis – the liver can focus on dealing with PBC without another ongoing injury.

- General Supportive Measures: We are also addressing general health factors:

    - Hydration: We educated her on maintaining good fluid intake (around 1.5–2 liters a day of water, or enough to keep urine light yellow), especially important since lactulose can cause fluid loss via loose stools. This prevents dehydration which could hurt her kidneys and trigger encephalopathy.

    - Diet: In addition to protein and salt considerations, we advise a diet rich in fruits, vegetables, and fiber (to help with regular bowels and provide micronutrients). If her appetite is poor, nutritional supplements like Ensure or specific liver disease formulas can be used.

    - Exercise: Encourage gentle exercise as tolerated (walking, light strength exercises) because maintaining muscle mass will help both her RA (keeps joints mobile) and her cirrhosis (muscle helps clear toxins and improves overall metabolism). Of course, we have to balance this with her fatigue levels – she should listen to her body and not overdo it. Even short walks a few times a day around the house are beneficial.

- Fall Precautions: Because she has some remaining encephalopathy risk and is an older adult, we suggest making her home safe: removing loose rugs, installing night lights, using grab bars in bathroom – to reduce fall risk, particularly if she gets dizzy or confused. We also told her not to drive until her doctor okays it, since she just had an episode of confusion; we want to be sure she's stable on lactulose with no recurrence before she resumes driving or operating heavy machinery.

Each of these plans is rooted in standard care guidelines from hepatology and nephrology societies, as well as tailored to her specific situation. We will provide a written copy of this plan to her and her caregivers, and all her specialists (liver, kidney, rheum, etc.) will get this information to coordinate care.

Discharge Instructions, Monitoring, and Follow-Up

As we prepare to discharge her, we have outlined important precautions and a follow-up plan to ensure a smooth and safe transition home. These instructions are meant for both the patient and her caregivers:

- Medication Management at Home: She will be going home on a new regimen:
    - Lactulose (for encephalopathy) – Take as directed (e.g. 30 mL by mouth two or three times a day, adjusted to produce 2–3 soft stools daily). *Do not skip doses*. It's the "brain clearance" medicine. Expect loose stools; that's on purpose. If she has no stool in 1 day, notify the doctor as dose may need increase. Carry extra clothes/protection when traveling in case of accidents (some patients prefer to stay near a bathroom after a dose).
    - Rifaximin 550 mg tablet twice a day (morning and evening) – This is to prevent confusion by reducing gut bacteria. Keep taking it continuously; it's not an antibiotic for infection but for liver condition, so finish each month's supply and refill as needed.
    - Ursodeoxycholic Acid (Ursodiol) – likely will be started, typical dose might be around 500 mg twice a day (we will confirm exact dosing after verifying her weight and prescription). This is for her PBC to protect the liver. It's important to take it regularly to help her liver function over time.
    - Ferrous Gluconate (Iron) 324 mg once daily – This will help build her blood count. Best taken with food to avoid stomach upset, and not at the exact same time as cholestyramine (if prescribed) because that can bind it.
    - Levothyroxine 25 mcg daily – Continue taking before breakfast on empty stomach (as she has been). Do not take it within 4 hours of cholestyramine either.
    - Losartan 50 mg daily – Continue for blood pressure/kidney protection. We'll check her blood pressure at home (we recommend home BP monitor) and follow up with her primary doc if adjustments needed.
    - Hydroxyzine 25 mg as needed every 6 hours for itching – This is optional for symptom relief. Causes drowsiness, so use primarily at night. If she feels too sleepy or groggy with it, use a lower dose or less frequently.
    - Cholestyramine (to be started) – Likely 4 g once or twice daily for itching and bile acid binding. Mix the powder with water or juice (not carbonated beverage) and drink. It can cause constipation, so we'll monitor her bowel pattern (though lactulose usually counteracts that). Take other medications 1 hour before or 4 hours after cholestyramine to avoid blocking their absorption .
    - [Possibly] Non-selective Beta Blocker (like Propranolol) – This will depend on the findings of her endoscopy. If large varices are found, she may be started on a beta blocker to reduce bleed risk. Beta blockers (e.g. propranolol) would be started at a low dose and titrated to a goal heart rate ~55–60 bpm as per portal hypertension guidelines . We will inform her if this is added, including to watch for lightheadedness (since they lower blood pressure).

- We have **stopped Alprazolam (Xanax)** and her **butalbital/codeine pain pills** due to the risk they pose. Do not resume these unless a doctor specifically says so (which is unlikely given her liver condition). For anxiety, we'll explore non-pharmacological methods or safer meds with her primary care if needed.

- Avoid **NSAIDs** like ibuprofen, Advil, Aleve, etc., as discussed. They harm kidneys and can cause bleeding in someone with varices.

- No over-the-counter herbal supplements or new medications without checking with her doctor, as many can affect the liver.

- **Diet and Lifestyle:**

  - **No Alcohol:** As stressed, she must remain completely abstinent from alcohol. Even a small amount can be dangerous for her now. We advised removing any alcohol from the home and notifying friends/family that she cannot partake. If she feels cravings or peer pressure, reach out for help – we can connect her with support groups.

  - **Diet:** Follow a **low-sodium diet** (2 grams salt per day or less) to prevent fluid retention/ascites. That means avoid adding salt to food, skip salty snacks, choose low-sodium versions of canned goods, etc. We gave her a handout on a low-sodium diet. Also, ensure **adequate protein** intake – incorporate proteins with each meal (eggs, fish, chicken, beans, Greek yogurt, nuts as tolerated). Because of her risk of HE, favor vegetable or dairy proteins and limit red meat portions. We also gave tips to help with poor appetite: eat smaller, more frequent meals (e.g., 5–6 small meals a day). Even if she doesn't feel very hungry, try to "graze" on nutritious snacks throughout the day to maintain calorie intake. A bedtime snack (like a bowl of cereal with milk or a slice of peanut butter toast) is recommended – this helps prevent muscle breakdown overnight .

  - **Fluid Intake:** Drink plenty of water (unless another doctor gives fluid restriction for some reason). With lactulose on board, dehydration can happen quickly if she has extra bowel movements, so she should sip fluids regularly. Monitor for signs of dehydration (dark urine, dry mouth) and increase fluids if needed.

  - **Activity:** She can be as physically active as she feels able. We encourage daily walking. However, **avoid strenuous activities that involve straining or heavy lifting**, because with portal hypertension, heavy straining (like lifting heavy weights or chronic coughing) can theoretically precipitate variceal bleeding. Light exercise is fine. If she does yoga or similar, avoid inverted poses that increase pressure in her head. Essentially, stay active but don't overexert. Also, given her anemia, she might feel fatigue on exertion; she should pace herself and rest as needed.

  - **Falls Precaution:** We mentioned home modifications earlier. We've advised using a cane or walker for stability if she feels unsteady (especially if she's having an off day with lactulose effects). Always get up slowly from lying or sitting to avoid dizziness (which can happen with blood pressure meds or anemia).

  - **Skin Care:** For itching, in addition to medications, we recommended lukewarm (not hot) showers, applying moisturizing cream (unscented) to damp skin, wearing cotton clothing, and potentially oatmeal baths or cool compresses to itchy areas. Keep nails trimmed to avoid skin damage from scratching. If itch is very bad, don't hesitate to use the hydroxyzine. Hopefully cholestyramine will kick in to relieve much of the itch over a couple of weeks.

- **Warning Signs – When to Seek Help:** We provided a list of red-flag symptoms that warrant immediate medical attention:

  - **Mental status changes:** If she becomes confused, excessively sleepy, or has personality changes, and it does not improve with an extra dose of lactulose, bring her to emergency – she may need additional treatment for encephalopathy or evaluation for triggers (like infection).

  - **Fever or signs of infection:** Particularly if she develops abdominal pain with fever, that could signal an infection in ascitic fluid (if she had any) or cholangitis (infection in bile ducts) if a stone is

blocking things. Fever and confusion together in a cirrhotic patient is an emergency (could be sepsis).

- GI Bleeding: If she vomits blood or what looks like "coffee grounds," or passes black tarry stools, or even bright red blood in stool, call 911. Variceal bleeding or any GI bleed in the context of cirrhosis is life-threatening. She should not drive in that scenario – it's a call ambulance situation. We've discussed signs of a bleed so she and family know what to watch for (vomiting blood, black stool, feeling faint, blood pressure dropping).

- Worsening jaundice or edema: If her eyes or skin become much more yellow, or her belly suddenly swells or legs swell significantly, she needs to see the doctor. That could indicate liver function worsening (for jaundice) or new ascites forming. We hope to prevent these by our current treatments, but must stay vigilant.

- Severe abdominal pain: Especially in right upper quadrant (could be gallstone moving or cholangitis) or diffuse pain with guarding (could be spontaneous peritonitis if she had ascitic fluid). Any severe abdominal pain in a cirrhotic patient should be evaluated promptly.

- Chest pain or trouble breathing: (Unrelated to liver directly, but important at her age to not ignore – she does have anemia which can strain the heart). If she has chest pain, call 911 to check for heart issues.

- Medication side effects: If she has uncontrolled diarrhea from lactulose (e.g., >5 watery stools/day with weakness), she should contact her doctor; dose adjustments can be made. If she develops any rash or allergic symptoms to UDCA or others, report that.

- Follow-Up Appointments: We have arranged a comprehensive follow-up plan:

  - Primary Care Physician (PCP): within 1 week of discharge to review her overall status, check blood pressure, monitor labs (basic metabolic panel, CBC, etc.), and coordinate referrals. The PCP will also manage her hypothyroid monitoring and anemia follow-up in conjunction with specialists.

  - Hepatology (Liver specialist): in 1–2 weeks. Given her complexity, seeing a liver specialist soon is important. They will review her antibody test results for PBC (if pending), ensure she's on UDCA and it's the correct dose, monitor her liver labs, and arrange the 6-monthly ultrasound surveillance for HCC. They will also oversee her HE management and adjust lactulose/rifaximin if needed. If she ever were to decompensate (develop major complications), the hepatologist might discuss evaluation for liver transplant; however, given her age (72) and co-morbidities, transplant may not be an option, so the focus will be on management (at 72, many centers don't transplant, but that's a far future discussion only if needed).

  - Gastroenterology (for ERCP/Endoscopy): We have referred her to GI to get the MRCP imaging in outpatient (if not already done in hospital) and then likely proceed with ERCP to extract the bile duct stone. This should be done relatively soon (within a few weeks) to avoid cholangitis. Also, GI/hepatology will perform an upper endoscopy at that visit or a separate one to look for varices. If large varices are found, they may do banding of them (a procedure to tie off the varices to prevent bleeding) or start a beta blocker. Follow their recommendations on subsequent surveillance endoscopies (usually every 2–3 years if no varices or small varices, or more frequently if large varices banded until eradicated).

  - Nephrology: in ~1 month, or sooner if the PCP or we feel it necessary. A nephrologist can ensure optimal CKD care (for example, managing anemia of CKD with ESAs if needed, checking PTH level for renal bone disease, advising on any further diet tweaks like phosphorus restriction if needed, etc.). They will also double-check that her blood pressure regimen is ideal and watch her kidney trends.

  - Rheumatology: in a month or two, to re-assess her RA. We will send them a detailed note about her new diagnoses. If her RA has been quiescent, they may not need to change much. If she has active arthritis, they'll choose a liver-safe treatment strategy. They might also screen her for other autoimmune issues (sometimes in PBC patients, Sjögren's syndrome (dry eyes/mouth) is common

– interestingly she does have some dry eyes noted in guidelines and hydroxychloroquine can help both RA and some PBC fatigue perhaps).

- Dermatology (if needed for itch): If her itch remains severe despite our measures, we might involve a dermatologist or specialized center that deals with cholestatic pruritus. They can offer therapies like UV light therapy (as mentioned in the PBC pruritus guidelines) or experimental treatments. Hopefully, we won't need this if cholestyramine and rifampin do the job.

- Nutritionist/Dietitian: within a couple weeks, to help her craft meal plans that incorporate all the recommendations (low salt, adequate protein, etc.) and to address her poor appetite. If she continues to lose weight or can't meet nutritional goals, we might consider supplement drinks or even temporary tube feeding in worst case – but we are not near that point, we think she can eat enough with guidance.

- Home Health Nursing: We are arranging for a home health nurse to visit during the first 1–2 weeks at home. The nurse can check her vital signs, assess her mental status, ensure medication compliance (especially lactulose titration), and draw any lab tests the doctors order. This provides an extra layer of monitoring early on.

- Caregiver support: We have involved her family in education. They have the encephalopathy action plan (lactulose adjustment, when to call 911). They also know how to help monitor her daily weight (for fluid retention) and mental status. We encourage using a notebook or chart to track her bowel movements, weight, and any symptoms each day – this can be very helpful to share with doctors at follow-ups.

- Emotional and Social Support: Chronic illness can be very taxing mentally. We offered resources for counseling or joining support groups (for liver disease or for caregivers). The American Liver Foundation has patient support groups which can connect her with others who have PBC/cirrhosis. If she experiences depression or anxiety (not uncommon given her conditions and perhaps worsened by some meds or the situation), she should inform her doctor – treatment (counseling or medication) can be provided. Also, because lactulose can be socially inconvenient (due to diarrhea), we discussed practical tips to handle that (planning outings after using the bathroom, carrying supplies). Sometimes patients feel embarrassed by HE or its treatment; we reassured her that this is a medical condition, nothing to be ashamed of, and that many patients live well with lactulose and never have a recurrence of severe encephalopathy. Open communication with her caregivers about how she's feeling will be important.

Finally, we provided a detailed medication schedule and a contact list (which doctor to call for what issue, and when to go to ER directly). We emphasized that with proper management, many patients with her condition continue to live meaningful, active lives. We want to keep her stable and out of the hospital.

She has demonstrated understanding of these instructions. Her daughter (primary caregiver) was present and has been educated on all the above points. They have a refrigerator-worthy summary sheet of the do's and don'ts.

By adhering to this comprehensive plan, we aim to keep her comfortable, prevent complications, and improve her overall health and quality of life. We utilized the latest clinical guidelines (from liver and kidney expert societies) to formulate this plan, and we will adjust it as needed in follow-up. The teamwork between her and her healthcare providers – and you as caregivers – will be key to a successful outcome.

References:

1. AASLD Practice Guidance on Primary Biliary Cholangitis – recommending UDCA 13–15 mg/kg/day for all patients with PBC and outlining pruritus management (cholestyramine first-line) .
2. AASLD/EASL Guidelines on Hepatic Encephalopathy – lactulose is first-line therapy, titrated to 2–3 soft bowel movements per day ; rifaximin is an effective add-on to prevent recurrence . Emphasis on addressing precipitating factors and not restricting dietary protein excessively .
3. Cirrhosis management guidelines – need for variceal screening and prevention (non-selective beta blockers or banding for varices) ; regular HCC surveillance every 6 months in cirrhosis ; vaccination and nutrition recommendations in chronic liver disease .
4. Portal hypertension pathophysiology – explaining low platelets and varices due to splenic sequestration and collateral circulation in cirrhosis .
5. Cholestatic pruritus in PBC – affects ~66% of patients and can be severe , requiring stepwise therapy (resins, rifampin, naltrexone, etc.) .
6. CKD care recommendations – importance of avoiding NSAIDs in CKD to prevent further renal injury and controlling blood pressure (often with ACEi/ARB) to slow progression
7. Patient education resources (Cirrhosis Care guides) – reinforce understanding of HE (toxins like ammonia cause confusion when liver can't filter them) and the need to avoid sedatives which precipitate HE .