1 PATIENT INFORMATION

NAME
Rosalind K. Amankwah-Byrne

DATE OF SERVICE
December 3, 2026

DOB
06/29/1968 (58 yrs)

PROVIDER
Miriam Halvorsen, MD

AGE / SEX
58 / Female

CREDENTIALS
MD, Board-Certified Cardiologist

MRN / PATIENT ID
CAR-615092

VISIT TYPE
Cardiology Follow-Up — post-hospital discharge (12 days)

CARE SETTING
Outpatient

REFERRAL SOURCE
Mercy General Hospital cardiology service, discharged 11/21/2026

PRIMARY CARE PROVIDER, IF APPLICABLE
Dr. Yusuf Rahimi, Internal Medicine

CC CHIEF COMPLAINT

"I'm still winded going up the stairs at work, and my heart keeps doing that fluttering thing. And I had to stop the water pill — I can't be running to the bathroom on the floor." Twelve-day post-discharge follow-up after an admission for acute decompensated heart failure with newly diagnosed atrial fibrillation.

S SUBJECTIVE

3a REASON FOR CARDIOLOGY EVALUATION

Post-hospital follow-up after a 4-day admission (11/17–11/21/2026) for acute decompensated heart failure with preserved ejection fraction, during which new-onset atrial fibrillation with rapid ventricular response was identified. The visit must address residual symptoms, a self-discontinued diuretic, rate versus rhythm strategy, anticoagulation, and — as emerges below — an unexamined question about the underlying etiology of her heart failure.

3b CHEST PAIN / ANGINAL SYMPTOMS

No exertional chest pain, pressure, or tightness. Denies radiation to jaw, neck, or arms. No rest pain. She reports a vague "full" sensation in the chest when the palpitations occur, which she distinguishes clearly from pain. No nitroglycerin use — none prescribed. Coronary CTA during admission showed no obstructive disease, making ischemia an unlikely contributor.

3c DYSPNEA / HEART FAILURE SYMPTOMS

Improved from admission but not resolved. Currently NYHA class III — dyspnea climbing one flight of stairs at work, where she is a hospital ward clerk, versus class IV at admission. Two-pillow orthopnea persists, improved from four pillows. Paroxysmal nocturnal dyspnea occurred twice in the past week, down from nightly. Bilateral ankle edema present by mid-afternoon, better than the marked pitting edema at admission. **Weight has risen 2.7 kg since discharge**, from 94.1 kg at discharge to 96.8 kg today. Abdominal bloating and early satiety reported. Fatigue prominent and limiting. Diuretic response was good in hospital — she diuresed 6 kg — but as below she has not been taking it.

3d PALPITATIONS / ARRHYTHMIA SYMPTOMS

Reports irregular fluttering episodes lasting 20 minutes to several hours, occurring 3–4 times weekly, sometimes with lightheadedness but never syncope. In retrospect she describes similar but briefer episodes for at least 8 months before admission, which she attributed to anxiety and menopause and never reported. Atrial fibrillation was first documented on telemetry during admission with a rapid ventricular response of 140–150. No known flutter, SVT, or PVCs. No pacemaker or ICD. She was discharged in sinus rhythm after rate control, but her symptom description strongly suggests ongoing paroxysmal episodes.

3e SYNCOPE / PRESYNCOPE

No syncope, ever. Presyncope described during palpitation episodes — lightheadedness on standing quickly, lasting seconds, without loss of consciousness, no injury, no seizure-like activity, and rapid recovery on sitting. No exertional presyncope, which would be more concerning. Possible contributors include rate-related hypotension during atrial fibrillation and her antihypertensive regimen.

3f BLOOD PRESSURE HISTORY

Long-standing hypertension since her late thirties, historically poorly controlled. Home readings over the past week, which she began logging at the hospital's request, average 152–164 / 88–96 mmHg. She reports good adherence to amlodipine and losartan. Salt intake is high — she eats most meals from the hospital cafeteria and describes adding salt habitually. No prior hypertensive urgency or emergency. Occasional lightheadedness on standing as noted.

3g CARDIAC HISTORY

No coronary artery disease — coronary CTA during admission was clean. No prior myocardial infarction, PCI, or CABG. **Heart failure with preserved ejection fraction, newly diagnosed this admission.** No known cardiomyopathy prior to this. Echocardiogram findings detailed below. No significant valvular disease. No congenital heart disease. **New-onset atrial fibrillation.** No pacemaker or ICD. No stroke or TIA. No peripheral artery disease. No prior cardiac procedures. Family history: mother died at 71 of "heart failure," father had a stroke at 68. Notably, an older brother has been "told something about his heart muscle being thick" — detail she could not expand on and which had not been recorded previously.

3h MEDICATION HISTORY AND TREATMENT RESPONSE

Current, as actually taken: apixaban 5 mg twice daily (adherent, no missed doses); metoprolol succinate 50 mg daily (adherent); amlodipine 10 mg daily; losartan 100 mg daily; atorvastatin 40 mg; metformin 1,000 mg twice daily; empagliflozin 10 mg daily, started at discharge. **Furosemide 40 mg daily — prescribed at discharge but self-discontinued after 4 days.** On direct and non-judgmental questioning she explained she works 10-hour shifts on a hospital ward with limited break coverage, had two episodes of urinary urgency and near-incontinence at work, and stopped it out of embarrassment. She did not tell anyone, and no one had asked. This is the single most important piece of history in the visit and directly explains the weight gain and persistent congestion. No prior antiarrhythmics. Tolerating apixaban with no bleeding.

3i RISK FACTORS

Hypertension, long-standing and uncontrolled. Type 2 diabetes for 9 years, most recent A1c 8.1%. Hyperlipidemia on statin. Never smoked. **Obesity with BMI 36.4**. Chronic kidney disease stage 3a with eGFR 54. Family history of premature cardiovascular disease is not clearly established, though her mother's heart failure and brother's reported "thick heart muscle" are relevant. **Obstructive sleep apnea — diagnosed 3 years ago with an AHI of 32, CPAP prescribed and abandoned within 2 months due to mask intolerance, never followed up.** Sedentary. No inflammatory disease. No prior radiation or cardiotoxic chemotherapy.

3j LIFESTYLE AND FUNCTIONAL CAPACITY

Functional capacity approximately 3 METs — limited by dyspnea climbing one flight. Not enrolled in cardiac rehabilitation and unaware it was available to her. Diet is high in sodium, largely cafeteria-based, with limited home cooking given shift work. Alcohol: one or two glasses of wine weekly. Caffeine: 3–4 cups of coffee daily, which she wonders about in relation to palpitations. Never smoked. Sleep apnea untreated. Occupationally active on her feet during shifts but without structured exercise. Daily activities are increasingly limited — she has stopped taking the stairs and has reduced her shopping and household activity.

3k RECENT TESTING / HOSPITALIZATIONS

Admission 11/17–11/21/2026 for acute decompensated heart failure. During that stay: ECG documenting atrial fibrillation with rapid ventricular response; transthoracic echocardiogram; coronary CTA excluding obstructive coronary disease; laboratory workup including a markedly elevated NT-proBNP; and IV diuresis with 6 kg fluid removal. Discharged in sinus rhythm on the regimen above. No emergency visits since discharge. No procedures. This is her first cardiology contact since discharge.

3l PERTINENT NEGATIVES

Denies active chest pain, resting dyspnea, hemoptysis, syncope, focal neurologic deficit, new unilateral leg swelling or calf pain, and bleeding or unusual bruising on apixaban. No fever. No signs of decompensation requiring immediate admission — she is comfortable at rest, speaking in full sentences, and saturating normally on room air.

ROS CARDIOLOGY REVIEW OF SYSTEMS

CHEST SYMPTOMS

Denies chest pain, pressure, or exertional angina; no nitroglycerin use; reports non-painful chest fullness with palpitations

HEART FAILURE SYMPTOMS

Positive — NYHA III dyspnea, two-pillow orthopnea, PND ×2 this week, ankle edema, 2.7 kg weight gain, abdominal bloating

ARRHYTHMIA SYMPTOMS

Positive — irregular palpitations 3–4× weekly with lightheadedness; denies syncope

CONSTITUTIONAL

Positive — marked fatigue and exercise intolerance; denies diaphoresis or nausea

PERIPHERAL VASCULAR

Positive for bilateral ankle edema; denies claudication, cyanosis, or unilateral swelling

NEUROLOGIC

Denies TIA or stroke symptoms, focal weakness, speech disturbance, or visual loss

BLEEDING / ANTICOAGULATION

Denies bleeding, bruising, hematuria, or melena on apixaban; no missed doses

SUBSTANCES

Never smoked; 1–2 alcoholic drinks weekly; 3–4 coffees daily; no stimulants

O OBJECTIVE

5a VITALS

TEMPERATURE
36.7 °C

OXYGEN SATURATION
95% on room air

BLOOD PRESSURE
158/92 mmHg (repeat 154/90)

WEIGHT
96.8 kg — up 2.7 kg from discharge weight of 94.1 kg

HEART RATE
92 bpm, irregularly irregular

BMI
36.4

RESPIRATORY RATE
18 / min

ORTHOSTATIC VITALS, IF OBTAINED
Seated 158/92, standing 142/84 with mild lightheadedness — no significant orthostatic drop

5b EXAMINATION

GENERAL APPEARANCE
Obese woman, comfortable at rest and speaking in full sentences; mildly volume-overloaded in appearance; no respiratory distress; no accessory muscle use

CARDIOVASCULAR
Irregularly irregular rhythm at 92 — she is in atrial fibrillation today, not sinus. No murmur, rub, or gallop appreciated. JVP elevated to approximately 10 cm at 45 degrees. No carotid bruits. Peripheral pulses 2+ and symmetric, irregular in timing. Capillary refill under 2 seconds. 2+ pitting edema to mid-shin bilaterally.

PULMONARY
Fine bibasilar crackles to the lower third bilaterally; no wheeze; mildly increased work of breathing on exertion during the visit; no oxygen requirement; dullness to percussion at the right base suggesting a small effusion

ABDOMEN
Obese, soft, mildly distended; non-tender; hepatomegaly with the liver edge palpable 3 cm below the costal margin and mildly tender — consistent with hepatic congestion; no ascites clinically detectable given habitus; no abdominal bruits

EXTREMITIES
2+ symmetric pitting edema to mid-shin; no unilateral swelling, calf tenderness, or cords to suggest DVT; pulses intact; no cyanosis, clubbing, or ulceration; mild varicosities bilaterally

SKIN
Warm and well perfused; no pallor, diaphoresis, or cyanosis; no bruising or bleeding on apixaban; no surgical scars or device pockets

NEUROLOGIC
Alert and fully oriented; no focal deficit; gait steady; **positive Tinel and Phalen signs bilaterally at the wrists, with reported nocturnal hand numbness for approximately 3 years, treated previously as "carpal tunnel from typing"**

DE DEVICE / ACCESS EXAMINATION, IF APPLICABLE

Not applicable — no pacemaker, ICD, or loop recorder in place, and no recent catheterization or surgical access site. Coronary CTA during admission was non-invasive. No device pocket, incision, or vascular access findings to document. An implantable loop recorder is under consideration as outlined in the plan.

CT CARDIAC TESTING / DIAGNOSTIC RESULTS

EKG:

Today: atrial fibrillation at 92 bpm, confirming she is not maintaining sinus rhythm. Normal axis. **Low voltage in the limb leads** with a poor R-wave progression pattern across the precordium. QTc 442 ms. No acute ischemic changes. **Comparison with the admission ECG shows the same low-voltage pattern** — which, taken alongside the echocardiographic left ventricular hypertrophy, is a discordant combination that deserves specific attention rather than being read past.

Echocardiogram:

From admission 11/18/2026: LVEF 58% (preserved). **Concentric left ventricular hypertrophy with a septal wall thickness of 15 mm.** Left atrium severely dilated at 48 mL/m². Grade 2 diastolic dysfunction with elevated filling pressures, E/e' ratio 18. Estimated pulmonary artery systolic pressure 46 mmHg, consistent with pulmonary hypertension. Mild mitral and tricuspid regurgitation. No significant valvular stenosis. Small pericardial effusion without tamponade physiology. **Global longitudinal strain was reported at –13% with an apical-sparing pattern** — a finding that was documented but not commented upon in the discharge summary.

Stress Testing:

Not performed. Not indicated at this time given a clean coronary CTA and the absence of anginal symptoms; her exercise limitation is clearly attributable to heart failure and atrial fibrillation rather than ischemia.

Coronary Imaging / Cath:

Coronary CTA 11/19/2026: no obstructive coronary artery disease, coronary calcium score 42 (low). No PCI or CABG. This effectively excludes an ischemic etiology for her heart failure and is an important negative.

Rhythm Monitoring:

Telemetry during admission captured atrial fibrillation with rapid ventricular response of 140–150 on day 1, converting to sinus rhythm with rate control by day 3. **No ambulatory monitoring has been performed since discharge**, which is a gap — her symptom pattern of 3–4 episodes weekly plus atrial fibrillation on today's ECG suggests a substantially higher burden than the discharge summary assumed. A 14-day ambulatory monitor is ordered today.

Heart Failure / Lab Data:

NT-proBNP 4,820 pg/mL at admission; **repeat today 2,940 pg/mL** — improved but persistently and substantially elevated, consistent with ongoing congestion. Troponin mildly elevated at admission and now normal. BMP today: sodium 136, potassium 4.1, creatinine 1.24 with eGFR 54 (CKD 3a, stable), bicarbonate 25. Magnesium 1.9. LFTs mildly elevated with AST 46 and ALT 52, consistent with hepatic congestion. TSH 2.2 (normal — important to exclude as an atrial fibrillation driver). **Iron studies: ferritin 68 ng/mL, TSAT 14% — iron deficiency without anemia, with hemoglobin 12.6.** A1c 8.1%. Lipids: LDL 88, HDL 42, triglycerides 190.

Vascular Studies:

No carotid ultrasound performed — no bruits and no cerebrovascular symptoms. No ABI — no claudication and palpable pulses. No venous duplex — edema is bilateral and symmetric with no features suggesting DVT. No aortic imaging indicated.

Prior Records Reviewed:

Complete hospital record from 11/17–11/21/2026 including admission notes, daily progress notes, telemetry summaries, the full echocardiogram report with strain imaging, the coronary CTA report, and the discharge summary and medication reconciliation. Primary care records from Dr. Rahimi covering the hypertension and diabetes history. The 2023 sleep study report documenting an AHI of 32 and the subsequent CPAP non-adherence note. Pharmacy fill history, which corroborated that furosemide was collected once and not refilled.

A ASSESSMENT

Heart failure with preserved ejection fraction, NYHA class III, with persistent congestion at 12 days post-discharge — and the reason is identifiable and fixable. She has gained 2.7 kg since discharge, has an elevated JVP, bibasilar crackles, hepatic congestion, and an NT-proBNP still at 2,940. This is not treatment failure: **she stopped her furosemide after four days because urinary urgency during 10-hour ward shifts caused two near-incontinence episodes and she was too embarrassed to tell anyone.** No one had asked. Any plan that does not solve the timing and practicality of her diuretic will fail again, and readmission for HFpEF within 30 days is a realistic prospect if this is not addressed today.

Atrial fibrillation — present on today's ECG, meaning she is not maintaining sinus rhythm as the discharge summary assumed. With 3–4 symptomatic episodes weekly and atrial fibrillation documented at this visit, her burden is clearly higher than recognized. CHA_2DS_2-VASc score is 4 (hypertension 1, diabetes 1, heart failure 1, female sex 1), giving an annual stroke risk of roughly 4–5%; apixaban is correctly indicated and she is adherent, which is the most important thing already going right. HAS-BLED is low. The rate-versus-rhythm question now needs revisiting: in HFpEF with a severely dilated left atrium and symptomatic episodes, atrial fibrillation and congestion drive each other, and there is a reasonable case for a rhythm-control strategy and electrophysiology input rather than rate control alone.

The unexamined question: is this cardiac amyloidosis? Four findings point that way and have not been connected. Left ventricular hypertrophy with a 15 mm septum on echocardiography, yet *low voltage* on ECG — hypertrophy normally produces high voltage, and this discordance is a recognized red flag. Global longitudinal strain of –13% with an *apical-sparing* pattern, documented in the report but not commented on, is close to characteristic. Bilateral carpal tunnel syndrome for three years, attributed to typing — amyloid deposition causes carpal tunnel syndrome that frequently predates cardiac presentation by years. A small pericardial effusion. Add a brother reportedly told he has a "thick heart muscle," and hereditary transthyretin amyloidosis becomes a serious consideration. This matters enormously: transthyretin cardiac amyloidosis has disease-modifying therapy available, alters anticoagulation and rate-control decisions, and changes prognosis and family screening. It would be a significant miss to continue treating this as ordinary hypertensive HFpEF without excluding it.

Uncontrolled hypertension at 158/92 with home readings of 152–164 systolic, contributing to both diastolic dysfunction and atrial fibrillation burden. **Untreated obstructive sleep apnea** with an AHI of 32 and CPAP abandoned three years ago after mask intolerance, never followed up — a major and reversible driver of atrial fibrillation recurrence, hypertension, and pulmonary hypertension, and one of the highest-yield interventions available. **Iron deficiency without anemia** (TSAT 14%, ferritin 68) — in heart failure this warrants IV iron, which improves symptoms and quality of life independent of hemoglobin. **Type 2 diabetes with A1c 8.1%**, obesity with BMI 36.4, CKD stage 3a, and pulmonary hypertension at 46 mmHg likely secondary to left heart disease and sleep apnea. **Favorable factors:** no obstructive coronary disease, preserved ejection fraction, excellent anticoagulation adherence, empagliflozin already started, and a patient who is engaged, works in healthcare, and disclosed the diuretic problem honestly once asked directly.

P PLAN

Heart failure plan:

Restart diuresis with a regimen built around her actual working day, which is the crux of this visit. Restart furosemide 40 mg but dosed after her shift rather than in the morning, so the diuresis occurs at home — discussed and agreed with her, and this simple change addresses the exact reason she stopped. Alternatively torsemide, with its more predictable absorption and longer action, if timing remains problematic. Written instruction provided to take it consistently and never to stop without telling us. **Daily morning weights** on a supplied scale with a written log, and a clear rule: gain of 1.5 kg in 2 days or 2.5 kg in a week means call the clinic, not wait. Sodium target under 2 g daily with specific, practical guidance for hospital cafeteria eating rather than generic advice. Continue empagliflozin, which has proven benefit in HFpEF. Fluid moderation to approximately 1.5–2 L. Repeat BMP in 1 week after restarting the diuretic given CKD 3a. **Cardiac rehabilitation referral placed** — she was unaware she was eligible.

Arrhythmia plan:

14-day ambulatory monitor ordered today to quantify true atrial fibrillation burden, which is almost certainly higher than the discharge assumption given today's ECG and her symptom frequency. Continue metoprolol succinate 50 mg for rate control; adequate at 92 today but to be reassessed on monitor data. **Electrophysiology referral placed** to discuss a rhythm-control strategy — in symptomatic atrial fibrillation with HFpEF and a severely dilated left atrium, restoring sinus rhythm may meaningfully improve her symptoms and congestion, and early rhythm control has a reasonable evidence base. Ablation candidacy to be assessed there. Address the reversible atrial fibrillation drivers in parallel: sleep apnea, hypertension, obesity, and caffeine. No antiarrhythmic started today pending electrophysiology input and the amyloid workup, since that result would influence agent selection.

Anticoagulation plan:

Continue apixaban 5 mg twice daily — correctly dosed, as she does not meet dose-reduction criteria (age under 80, weight above 60 kg, creatinine below 1.5). CHA_2DS_2-VASc 4 gives a clear indication and HAS-BLED is low. She is fully adherent with no bleeding, which was reinforced positively. Reviewed bleeding precautions, the importance of never missing doses, and what to do if a dose is missed. Renal function to be monitored as eGFR is 54 and apixaban dosing depends on it. Peri-procedural instructions to be provided ahead of any ablation or biopsy. She was counselled explicitly that anticoagulation continues regardless of whether sinus rhythm is restored.

Chest pain or CAD plan:

No obstructive coronary disease on CTA and no anginal symptoms — no antianginal therapy, no further ischemic testing, and no catheterization indicated. Continue atorvastatin 40 mg for cardiovascular risk reduction with an LDL of 88; consider intensification toward an LDL under 70 given diabetes and CKD. No aspirin — correctly avoided, as adding it to apixaban without a coronary indication would increase bleeding risk without benefit.

Diagnostic testing ordered — amyloidosis workup:

This is the most consequential new action of the visit. The combination of LVH with low ECG voltage, apical-sparing strain, three years of bilateral carpal tunnel syndrome, a small pericardial effusion, and a brother with reported "thick heart muscle" requires that transthyretin cardiac amyloidosis be excluded rather than assumed away. Ordered: serum and urine immunofixation with serum free light chains to exclude AL amyloidosis first, which is essential because AL is a haematological emergency requiring different and urgent treatment; and a **technetium pyrophosphate scan**, which is non-invasive and, in the absence of a monoclonal protein, can establish ATTR cardiac amyloidosis without biopsy. If positive, genetic testing for hereditary ATTR follows, with implications for her brother and children. Cardiac MRI with late gadolinium enhancement if scintigraphy is equivocal. Referral to the amyloidosis programme prepared pending results. Explained to her honestly as "a test to check for a specific cause we should not miss," without alarming overstatement.

Hypertension plan:

Blood pressure 158/92 today with home readings of 152–164 systolic — uncontrolled and directly worsening both diastolic dysfunction and atrial fibrillation burden. Restarting the diuretic will itself help. Increase losartan from 100 mg is not possible at maximum dose, so **add spironolactone 25 mg daily**, which addresses hypertension and has a role in HFpEF, with potassium and creatinine monitoring at 1 and 4 weeks given CKD 3a and a current potassium of 4.1. Continue amlodipine 10 mg. Target below 130/80. Continue home monitoring with the log she has already started, which she does well. Sodium restriction as above is central rather than incidental.

Lipid and risk reduction plan:

Continue atorvastatin 40 mg; consider increasing to 80 mg or adding ezetimibe to reach an LDL below 70 given diabetes and CKD. **Diabetes control needs attention** — A1c 8.1% with empagliflozin already providing cardiorenal benefit; endocrinology or primary care referral for intensification, with a GLP-1 receptor agonist particularly attractive here given obesity, cardiovascular benefit, and weight reduction. **Weight management** with BMI 36.4 — weight loss improves HFpEF symptoms, atrial fibrillation burden, sleep apnea, and blood pressure simultaneously, making it one of the highest-yield targets; referral to a medical weight management programme offered and accepted. Never smoked. Reduce caffeine from 3–4 cups daily given the palpitation association. Alcohol already minimal; advised to keep it so.

Valvular disease plan:

Only mild mitral and tricuspid regurgitation, likely functional and secondary to atrial and ventricular remodelling with elevated filling pressures. No stenosis. No intervention indicated. Surveillance echocardiography in 12 months, or sooner if symptoms change disproportionately or a murmur develops. No endocarditis prophylaxis indicated. Both regurgitant lesions may improve with effective decongestion and rhythm control.

Syncope plan:

No true syncope — presyncope only, occurring during palpitations and on rapid standing, without exertional features. No significant orthostatic drop on measurement today. This is most consistent with rate-related hypotension during atrial fibrillation, and the ambulatory monitor should correlate symptoms with rhythm directly. No tilt testing indicated. Counselled on slow position changes and adequate hydration within her fluid allowance. No driving restriction required as there has been no loss of consciousness, though she was advised not to drive if she feels presyncopal.

Referral or coordination plan:

Sleep medicine referral — high priority. Untreated OSA with an AHI of 32 is a major driver of her atrial fibrillation, hypertension, and pulmonary hypertension, and it has gone unaddressed for three years since she abandoned CPAP. Referral specifically requests mask refitting and alternatives including nasal pillows, an auto-titrating device, or consideration of a mandibular advancement device or hypoglossal nerve stimulation if CPAP is truly not tolerable — the previous failure was a fit problem, not a reason to abandon treatment. Electrophysiology as above. Amyloidosis programme pending results. Cardiac rehabilitation. Medical weight management. Endocrinology or primary care for diabetes intensification. **IV iron arranged** for TSAT 14% with ferritin 68 — ferric carboxymaltose improves symptoms and functional capacity in heart failure irrespective of anemia. Dr. Rahimi updated in full.

Patient education:

Spent substantial time on the diuretic problem specifically — explained why stopping it caused the weight gain and breathlessness, thanked her genuinely for telling me, and made clear that a practical scheduling problem is something we solve together rather than a compliance failure. She was visibly relieved. Taught daily weights with the specific call thresholds written down. Explained atrial fibrillation, why anticoagulation must continue regardless of rhythm, and stroke warning signs using the FAST framing. Reviewed heart failure warning signs requiring urgent contact: rapid weight gain, worsening breathlessness or orthopnea, resting breathlessness, or new chest pain. Explained the amyloid testing honestly and without alarm. Reframed the sleep apnea as a cardiac treatment rather than a sleep inconvenience, which she had not appreciated. Written materials, a weight log, and direct clinic contact details provided.

F FOLLOW-UP

Return in 1 week for weight, volume status, blood pressure, and a BMP to check potassium and renal function after restarting furosemide and adding spironolactone — the short interval reflects the readmission risk in the 30-day post-discharge window. Then in 3–4 weeks to review the 14-day monitor and the amyloidosis workup, with repeat NT-proBNP. The technetium pyrophosphate scan and immunofixation with free light chains should be completed within 2–3 weeks; results will be discussed by phone as soon as available rather than held to the next visit. Electrophysiology within 4 weeks. Sleep medicine within 4 weeks, expedited if possible. IV iron infusion within 2 weeks. Cardiac rehabilitation intake when volume status is stable. Repeat echocardiogram in 3 months, or sooner if the amyloid workup is positive. Instructed to contact the clinic for a weight gain of 1.5 kg in 2 days or 2.5 kg in a week, worsening breathlessness, or new orthopnea; and to present to the emergency department for chest pain, breathlessness at rest, syncope, or any stroke symptoms.

TD TIME DOCUMENTATION, IF APPLICABLE

TOTAL TIME SPENT

55 minutes

RECORDS REVIEW TIME, IF APPLICABLE

20 minutes (hospital record, echocardiogram with strain, coronary CTA, 2023 sleep study, pharmacy history)

COUNSELING / COORDINATION OF CARE TIME

25 minutes

DEVICE / TEST REVIEW TIME, IF APPLICABLE

15 minutes (telemetry summary, serial ECG comparison identifying the low-voltage pattern)

TB BILLING CONSIDERATIONS

E/M LEVEL

99215 — Established patient, high complexity

PROCEDURE CODE(S), IF APPLICABLE

None performed this visit

DIAGNOSTIC TEST INTERPRETATION CODE(S), IF APPLICABLE

93000 — ECG with interpretation and report, performed in office today

BASIS FOR BILLING

Medical Decision Making (high) — also supported by total time of 55 minutes

PRIMARY CARDIOVASCULAR DIAGNOSIS CODE + DESCRIPTION

I50.31 — Acute diastolic (congestive) heart failure (HFpEF, NYHA III)

SECONDARY DIAGNOSIS CODE(S)

I48.0 (paroxysmal atrial fibrillation); I11.0 (hypertensive heart disease with heart failure); Z79.01 (long-term anticoagulant use); E11.22 (T2DM with diabetic CKD); N18.30 (CKD stage 3a); G47.33 (obstructive sleep apnea); E66.01 (morbid obesity); D50.9 (iron deficiency); I27.20 (pulmonary hypertension); G56.03 (bilateral carpal tunnel syndrome); R93.1 (abnormal cardiac imaging — amyloidosis under investigation)

SO SIGNATURE

PROVIDER NAME	CREDENTIALS	SPECIALTY	DATE	TIME
Miriam Halvorsen, MD	MD, FACC	Cardiology	12/03/2026	11:10