MARVIX AI

SAMPLE NEPHROLOGY SOAP NOTE

AKI ON CKD STAGE 3B — CONTRAST AND NSAID EXPOSURE

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1 PATIENT INFORMATION

NAME	Gerald P. Whitcombe
DOB	01/19/1963 (63 yrs)
AGE / SEX	63 / Male
MRN / PATIENT ID	REN-704228
REFERRAL SOURCE	Cardiology (Dr. L. Ferraro) following vascular imaging
DATE OF SERVICE	March 6, 2026
PROVIDER	Anita Deshmukh, MD
CREDENTIALS	MD, Board-Certified Nephrologist
VISIT TYPE	Nephrology Consultation — urgent outpatient referral
PRIMARY CARE PROVIDER	Dr. Susan Kang, Family Medicine

CC CHIEF COMPLAINT

"My kidney numbers went the wrong way after that dye scan, and my legs are puffy again." Referred urgently for a rise in creatinine and worsening proteinuria following CT angiography three weeks ago.

S SUBJECTIVE

3a REASON FOR NEPHROLOGY EVALUATION

Evaluation of acute kidney injury superimposed on known CKD stage 3b, with worsening proteinuria, lower-extremity edema, and hyperkalemia identified on outpatient labs following contrast-enhanced CT angiography performed for peripheral arterial disease workup on February 12, 2026.

3b KIDNEY DISEASE HISTORY

CKD first documented in 2018 in the context of long-standing type 2 diabetes and hypertension. Baseline creatinine has run 1.5–1.6 mg/dL with eGFR 42–45 mL/min/1.73m² (CKD stage 3b) over the past two years, with slow, predictable decline of roughly 2–3 mL/min per year. Current creatinine 2.4 mg/dL with eGFR 26 — a clear departure from baseline. One prior AKI episode in 2022 during a hospitalization for cellulitis with sepsis, which recovered to baseline within three weeks. No prior nephrology care until this referral; CKD had been managed by primary care.

3c URINARY SYMPTOMS

Reports increasingly foamy urine over the past two months. Nocturia has increased from once to three times nightly. Denies gross hematuria, dysuria, flank pain, stone passage, hesitancy, or retention. Urine output subjectively preserved, though he notes it is "less than it used to be" on the days his legs are most swollen.

3d VOLUME STATUS SYMPTOMS

Bilateral lower-extremity edema to mid-shin, worse in the evenings, present for six weeks and progressively worsening. Weight up 9 lb over that interval despite no dietary change. Two-pillow orthopnea, new in the last two weeks. Mild exertional dyspnea climbing one flight. Denies PND, chest pain, or dizziness. Appetite mildly reduced.

3e BLOOD PRESSURE HISTORY

Hypertension since approximately 2004. Home readings over the past month have run 150–165/85–95 mmHg, up from his usual 130s. Reports good adherence to lisinopril and amlodipine but admits inconsistent adherence to sodium restriction, with frequent restaurant and canned foods. No hypertensive urgency or emergency history. No orthostatic symptoms.

3f DIABETES / METABOLIC HISTORY

Type 2 diabetes for 19 years. Most recent A1c 8.2% (up from 7.4% one year ago). Complications include background diabetic retinopathy and mild peripheral neuropathy. BMI 33.4. Recurrent gout with three flares in the past year, for which he self-treats with over-the-counter naproxen. Hyperuricemia documented; not on urate-lowering therapy. Dyslipidemia on atorvastatin.

3g MEDICATION AND NEPHROTOXIN EXPOSURE

Significant and multifactorial nephrotoxin exposure: (1) iodinated contrast during CT angiography on 02/12/2026; (2) chronic intermittent NSAID use — naproxen 440–880 mg daily for roughly three weeks during a recent gout flare; (3) lisinopril 40 mg daily, contributing to reduced glomerular filtration pressure and hyperkalemia in the setting of volume shifts. Also on amlodipine, atorvastatin, metformin, glipizide, and omeprazole (long-term, a potential interstitial nephritis contributor). No lithium, chemotherapy, or herbal supplements. Not currently on an SGLT2 inhibitor.

3h DIET / FLUID HISTORY

High sodium intake, estimated well above 4 g daily, largely from processed and restaurant foods. No prior renal diet counseling. Fluid intake approximately 2–2.5 L daily. No potassium or phosphorus restriction, and he reports frequent orange juice, bananas, and salt substitute use — the last being a likely potassium contributor. Alcohol: 2–3 beers weekly.

3i RELEVANT MEDICAL HISTORY

Type 2 diabetes, essential hypertension, peripheral arterial disease, dyslipidemia, gout, obesity, and obstructive sleep apnea (CPAP-adherent). Prior cellulitis with sepsis in 2022. No heart failure diagnosis, though current symptoms raise the question. No liver disease, autoimmune disease, malignancy, nephrolithiasis, or urinary obstruction. No prior transplant. Family history notable for a father with “kidney failure” on dialysis in his seventies, etiology unclear, and a sister with type 2 diabetes.

3j PRIOR WORKUP AND TREATMENT

Renal ultrasound in 2018 showed normal-sized kidneys with mildly increased echogenicity and no hydronephrosis. No prior kidney biopsy. Urine albumin/creatinine ratio was 180 mg/g in 2023, now substantially higher. No prior serologic workup, dialysis access planning, or renal diet counseling. CKD had been monitored with annual labs by primary care only.

3k PERTINENT NEGATIVES

Denies anuria, gross hematuria, severe flank pain, fever, chills, rash, joint swelling beyond his typical gout, chest pain, severe dyspnea, confusion, pruritus, metallic taste, or hiccups. No symptoms of hypertensive emergency — no headache, visual change, or focal neurologic deficit. No bleeding.

ROS NEPHROLOGY REVIEW OF SYSTEMS

CONSTITUTIONAL / UREMIC

Mild fatigue and reduced appetite; denies confusion, pruritus, nausea, or vomiting

CARDIOPULMONARY

Positive for edema, two-pillow orthopnea, and mild exertional dyspnea; denies chest pain or palpitations

URINARY

Positive for foamy urine and nocturia ×3; denies hematuria, dysuria, or flank pain

NEUROLOGIC / BP-RELATED

Denies dizziness, orthostasis, headache, or vision changes

MUSCULOSKELETAL / ELECTROLYTE

Occasional leg cramps; denies bone pain or paresthesias

INFECTIOUS / AUTOIMMUNE

Denies fever, chills, rash, joint pain beyond gout, or recurrent infections

MEDICATION-RELATED

Positive for recent NSAID use and iodinated contrast exposure

O OBJECTIVE

5a VITALS

TEMPERATURE

98.2 °F

OXYGEN SATURATION

96% on room air

BLOOD PRESSURE

168/92 mmHg (repeat 164/90)

WEIGHT

231 lb (up 9 lb from 01/2026)

HEART RATE

78 bpm, regular

BMI

33.4

RESPIRATORY RATE

16 / min

ORTHOSTATIC VITALS

Not obtained — no orthostatic symptoms

5b EXAMINATION

GENERAL APPEARANCE

Obese male, comfortable at rest, mildly volume-overloaded in appearance; well nourished, alert and fully oriented

CARDIOVASCULAR

Regular rate and rhythm; no murmur or gallop; JVP elevated to approximately 9 cm; 2+ pitting edema to mid-shin bilaterally

PULMONARY

Fine bibasilar crackles at both bases; no wheezing; normal work of breathing; no supplemental oxygen required

ABDOMEN

Obese, soft, non-tender; no ascites; no abdominal or flank bruits; no flank tenderness; no suprapubic fullness

GENITOURINARY

Not formally assessed; no catheter; no reported retention

EXTREMITIES

SKIN

2+ symmetric pitting edema to mid-shin; dorsalis pedis pulses diminished bilaterally (known PAD); no ulcers; no dialysis access present

Warm and dry; no rash, purpura, excoriations, or uremic frost; no vasculitic lesions

NEUROLOGIC

Alert and oriented ×3; no asterixis; mild stocking-distribution sensory loss consistent with diabetic neuropathy; strength intact

DA RENAL / DIALYSIS ACCESS EXAMINATION, IF APPLICABLE

Not applicable at this visit — no dialysis access present. Vein preservation counseling provided given CKD stage and progression risk: advised avoidance of IVs, blood draws, and PICC lines in the non-dominant left arm to protect future fistula options.

L LABORATORY AND DIAGNOSTIC RESULTS

Kidney Function:

Creatinine 2.4 mg/dL (baseline 1.5–1.6); eGFR 26 mL/min/1.73m² (baseline 42–45); BUN 46 mg/dL. Trend: 1.6 on 01/08/2026 → 2.7 on 02/22/2026 → 2.4 today, indicating a peak with early partial improvement. Cystatin C not obtained.

Electrolytes / Acid-Base:

Sodium 137, potassium 5.9 mmol/L (elevated), chloride 106, bicarbonate 18 mmol/L (metabolic acidosis), calcium 8.6, phosphorus 4.9, magnesium 2.0. Anion gap 13.

Urine Studies:

Urinalysis: 3+ protein, trace blood, no leukocyte esterase or nitrites, no casts on microscopy, no eosinophils. Urine albumin/creatinine ratio 1,850 mg/g (up from 180 mg/g in 2023). Urine protein/creatinine ratio 2.6 g/g. Urine sodium 12 mmol/L, consistent with a prerenal/effective-volume component. Urine culture not indicated.

CKD / Mineral Bone Disease Labs:

PTH 148 pg/mL (elevated); 25-OH vitamin D 18 ng/mL (deficient); calcium 8.6; phosphorus 4.9; alkaline phosphatase 96.

Anemia Labs:

Hemoglobin 10.4 g/dL, hematocrit 31.5%; ferritin 68 ng/mL; TSAT 15% — consistent with anemia of CKD with absolute iron deficiency. B12 and folate normal.

Serologic Workup:

Sent given the magnitude of proteinuria: ANA, ANCA, C3/C4, anti-GBM, SPEP/UPEP with free light chains, and hepatitis B/C serologies. Results pending. HIV testing offered and declined.

Imaging:

Renal ultrasound 03/04/2026: right kidney 10.4 cm, left 10.1 cm, both with increased cortical echogenicity and preserved corticomedullary differentiation. No hydronephrosis, stones, or masses. Post-void residual 30 mL. CT angiography 02/12/2026 (with iodinated contrast) confirmed infrarenal aortoiliac disease without renal artery stenosis.

Pathology:

No kidney biopsy performed to date. Biopsy under consideration pending serologic results and response to intervention.

Prior Records Reviewed:

Primary care lab trends 2018–2026, cardiology consultation and CT angiography report, prior renal ultrasound (2018), medication history including pharmacy fill records confirming NSAID purchases, and 2022 hospitalization records documenting the prior AKI episode.

A ASSESSMENT

Acute kidney injury superimposed on CKD stage 3b, multifactorial. Creatinine has risen from a stable baseline of 1.5–1.6 to a peak of 2.7 with modest improvement to 2.4, corresponding to a fall in eGFR from 42–45 to 26. The injury is best explained by a convergence of three insults occurring within the same window: iodinated contrast exposure on 02/12/2026, roughly three weeks of near-daily NSAID use for gout, and continued full-dose ACE inhibition — a combination that impairs both afferent and efferent autoregulation. A low urine sodium of 12 mmol/L supports a significant hemodynamic component, and the early downtrend in creatinine favors a largely reversible process rather than established intrinsic injury. Long-term omeprazole use raises the possibility of a contributing interstitial nephritis, though the bland urine sediment without eosinophils or white cell casts makes this less likely.

Underlying CKD stage 3b, presumed diabetic nephropathy. Nineteen years of type 2 diabetes with retinopathy and neuropathy, gradual eGFR decline, and echogenic kidneys of preserved size all fit diabetic kidney disease. However, the escalation of albuminuria from 180 mg/g to 1,850 mg/g over roughly three years is steeper than expected from diabetes alone and warrants exclusion of a superimposed glomerular process — hence the pending serologic workup. Worsening glycemic control (A1c 8.2%) and uncontrolled hypertension are both accelerating factors.

Volume overload. Nine-pound weight gain, elevated JVP, bibasilar crackles, orthopnea, and 2+ edema indicate genuine volume expansion driven by high sodium intake, reduced GFR, and heavy proteinuria with resultant hypoalbuminemia.

Hyperkalemia (5.9 mmol/L) attributable to reduced GFR, ACE inhibition, metabolic acidosis, and dietary potassium including salt-substitute use — a frequently overlooked contributor. **Metabolic acidosis** (bicarbonate 18) is a CKD complication that itself accelerates progression and potentiates hyperkalemia. **Anemia of CKD with absolute iron deficiency** (TSAT 15%, ferritin 68). **Early CKD-mineral bone disease** with secondary hyperparathyroidism and vitamin D deficiency.

Progression risk and RRT planning. By KFRE estimation, this patient carries a meaningful 2-year and 5-year risk of kidney failure. He is not currently dialysis-dependent and has no uremic indications, but the trajectory warrants early modality education and vein preservation now rather than reactively later.

P PLAN

Kidney function monitoring:

Repeat BMP and urine studies in 5–7 days, then every 2 weeks until creatinine plateaus. Recheck urine protein/creatinine ratio at 6 weeks after intervention. Follow pending serologies closely.

AKI on CKD management:

Address all three insults concurrently — no further contrast unless essential (and with pre-hydration if unavoidable), strict and permanent NSAID avoidance, and temporary ACE inhibitor management as below. Avoid additional nephrotoxins. Discontinue omeprazole and switch to famotidine given the interstitial nephritis question and absence of a strong ongoing indication.

Blood pressure management:

Hold lisinopril temporarily given AKI with hyperkalemia. Increase amlodipine to 10 mg daily and add hydralazine if needed for interim control. Target below 130/80 once stabilized. Reintroduce ACE inhibition, likely at a reduced dose, once creatinine and potassium stabilize — the long-term antiproteinuric benefit is important and should not be abandoned permanently. Home BP log twice daily.

Proteinuria management:

Once AKI resolves and potassium normalizes, resume renin-angiotensin blockade at reduced dose and initiate an SGLT2 inhibitor (dapagliflozin 10 mg daily), which offers substantial renal and cardiovascular protection in proteinuric diabetic CKD. Defer initiation until the acute injury has settled. Finerenone may be considered later if potassium permits.

Volume management:

Start furosemide 40 mg daily with close monitoring; titrate to symptoms and weight. Sodium restriction to under 2 g daily with formal counseling. Daily home weights with a log; instructed to report a gain exceeding 3 lb in a week. Fluid intake moderation to approximately 1.5–2 L daily.

Electrolyte and acid-base management:

For potassium 5.9: hold lisinopril, stop salt substitute entirely, start dietary potassium restriction, and initiate patiromer 8.4 g daily. Recheck potassium in 5 days. For bicarbonate 18: start sodium bicarbonate 650 mg twice daily, targeting bicarbonate above 22, balancing the small sodium load against diuretic therapy.

Anemia of CKD management:

Absolute iron deficiency present. Start IV iron sucrose given oral intolerance risk and better efficacy in CKD — arrange a course, then recheck iron studies and hemoglobin in 4 weeks. ESA therapy deferred; reassess if hemoglobin remains below 10 after iron repletion. No transfusion indicated.

CKD-mineral bone disease management:

Start cholecalciferol 2,000 IU daily for vitamin D deficiency. Phosphorus currently acceptable — no binder yet, but counsel on dietary phosphorus and avoidance of phosphate additives in processed foods. Recheck calcium, phosphorus, PTH, and vitamin D in 3 months.

Medication review:

Full reconciliation performed. Metformin held while eGFR is below 30 and to be reassessed at recovery. Renal dosing reviewed for all agents. Permanent NSAID avoidance emphasized — for gout, use colchicine or a short prednisone course instead, and consider urate-lowering therapy with allopurinol (renally dosed) once stable. Coordinated with primary care and cardiology by direct message.

Diet and lifestyle counseling:

Renal dietitian referral placed for sodium, potassium, phosphorus, and protein counseling (target protein approximately 0.8 g/kg/day). Diabetes optimization discussed — endocrinology referral placed given A1c 8.2%. Weight management and continued CPAP adherence encouraged. He is a lifelong non-smoker.

Dialysis planning:

Given CKD 3b trending toward stage 4 with heavy proteinuria, initiated early education. Vein preservation counseling given today, with a left-arm protection wristband provided. Formal modality education to be scheduled when eGFR approaches 20. Transplant referral to be initiated at eGFR 20 or sooner if decline continues.

Referral plan:

Renal dietitian, endocrinology, and CKD education class placed today. Vascular surgery and transplant nephrology deferred pending trajectory. Kidney biopsy to be discussed with the patient if serologies return positive or if proteinuria fails to improve with therapy.

Patient education:

Reviewed CKD stage, what the numbers mean, and the specific role each of the three insults played — he was previously unaware that over-the-counter naproxen posed a kidney risk. Warning signs reviewed: decreased urine output, rapid weight gain, worsening breathlessness, confusion, and severe weakness. Written materials and a medication card listing agents to avoid were provided.

F FOLLOW-UP

Return in 2 weeks for repeat labs and clinical reassessment of volume status, blood pressure, and potassium, with an interim BMP in 5–7 days after starting patiromer and furosemide. At the 2-week visit: review pending serologies, reassess suitability for reintroducing ACE inhibition and initiating an SGLT2 inhibitor, and evaluate proteinuria response. If creatinine fails to return toward baseline by 4–6 weeks, or if serologies are positive, proceed to kidney biopsy. Nephrology will assume co-management of CKD going forward, with visits every 3 months once stable.

TD TIME DOCUMENTATION, IF APPLICABLE

TOTAL TIME SPENT

65 minutes

COUNSELING / COORDINATION OF CARE TIME

30 minutes

RECORDS REVIEW TIME

20 minutes (external records, imaging, and lab trends)

TB BILLING CONSIDERATIONS

E/M LEVEL

99205 — New patient, high complexity

BASIS FOR BILLING

Medical Decision Making (high) — supported by total time of 65 minutes

PRIMARY RENAL DIAGNOSIS CODE + DESCRIPTION

N17.9 — Acute kidney failure, unspecified (AKI on CKD)

SECONDARY DIAGNOSIS CODE(S)

N18.32 (CKD stage 3b); E11.22 (T2DM with diabetic CKD); I12.9 (hypertensive CKD); E87.5 (hyperkalemia); D63.1 (anemia in CKD); N25.81 (secondary hyperparathyroidism of renal origin); R80.9 (proteinuria)

SO SIGNATURE

PROVIDER NAME	CREDENTIALS	SPECIALTY	DATE	TIME
Anita Deshmukh, MD	MD, FASN	Nephrology	03/06/2026	16:15