Thieves’ Market

JUNE 6, 2026

What is a Thieves' Market?

- Present a case
  - History
  - Physical Exam
  - Lab and imaging studies
  - Reveal diagnosis
- Review teaching points about the case
- Repeat for a second case

Your Role in Thieves' Market

- Steal the diagnosis… but how?

- Interrupt and shout out the diagnosis

Requirements to Win

Shout out the diagnosis before it appears on the screen!

Nonspecific diagnoses won’t count

You must say and I must repeat what you said

I will not confirm whether a guess is correct until the end

What happens if I win?

Case 1 History

CC: Dizziness

HPI: 76 yo woman with progressive dizziness over days

Further history is limited… “just dizzy”

Daughter notes

- Generalized malaise & weakness x 1 week
- Decreased mental acuity
- “off balance” when walking

Review of Systems

POSITIVE

Nausea without vomiting

Anorexia

NEGATIVE

Tinnitus

Hearing changes

Vision changes

Diplopia

Loss of coordination

Speech changes

Headache

Vomiting

fever

Weight loss

Joint pain

Rashes

History

Past Medical History:

1. DM type 1 (> 50 years)
2. HTN
3. Cirrhosis c/b hepatic encephalopathy (Dg 1 year ago, admission 3 weeks ago for HE)
4. Heart failure with grade 1 diastolic dysfunction
5. Diffuse large B-cell lymphoma (diagnosed May 2018, s/p R-CHOP w/consolidative radiation)

Procedure History:

1. Cesarean Section x2 (over 20 yrs ago)
2. Hysterectomy (over 20 yrs ago)

History

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Medications:

Lisinopril 5mg Rifaximin 550[2]

Pantoprazole 40mg Dapagliflozin 10

Lactulose 20[3] Insulin dedludec 20u, aspart 20u[3]

Social Hx:

- No prior or active alcohol, tobacco or drug use
- Lives in Montgomery with her daughter
- Works as a medical tech

Family Hx:

Father: HTN Paternal GM: Colon Cancer

Daughter: Type 1 DM

UAB

Physical Exam

Vitals: T: 98.4 HR: 93 BP: 67/37 RR: 19 O2: 98% on RA BMI: 23

Gen: NAD
HEENT: upper lip hematoma w/ slight oozing on buccal side of the lip.
"accidentally bit her lip while nurses turning her"
Neck: no cervical lymphadenopathy, supple. JVD not visible
Lungs: Normal work of breathing. No wheezing or crackles.
CV: NRRR, good peripheral pulses
Abd: NT, ND, normal BS

Skin: No rashes

Lymph: no palpable lymph nodes
Neuro: AOx2, CN 2-12 intact, motor strength 5/5, sensation intact, no asterixis
Ext: 2+ pitting edema BLE

Case 1 Evolution

50 g albumin
IVF
Still hypotensive
ICU Admission for shock

Labs

132	97	27
5.5	30	1.4

134

Ca: 10

Mag: 2.0

Phos: 2.7

PT: 18.4

INR 1.52

PTT: 33

TP	5.1	3.2	Alb
TBil	5.6	1.3	DBil
AST	58	54	ALT
AΦ	172		

Urinalysis:
1.008, neg protein, 2+ glucose

VBG 7.47/41/31/27

	10.8	
4.5	31	133

MCV: 104
RDW: 17.5

Differential:
- 66% Neutrophils
- 12% Lymphocytes
- 19% Monocytes
- 2%Eosinophils
- 1% Basophils

Initial Imaging

CXR:
Mild interstitial prominence throughout both lungs with possible retrocardiac consolidation and small bilateral pleural effusions.

CT Head w/o Contrast: No acute intracranial abnormality. Age related changes.

Labs continued

BNP 308
hs Troponin 31 -> 32

Ammonia 31
Lipase 49
Amylase 47
GGT 85

Total Cholesterol 64
Triglycerides 56
HDL 18
LDL 37

AM Cortisol 7.2 (on pressors)
TSH 0.75
FT4 1.77

Blood cultures Negative
Urine cultures Negative

Case 1 Evolution

50 g albumin
IVF
Still hypotensive
ICU Admission for shock

LFTs continue to increase
T Bili= 6.1, alk phos 200
AST 223
ALT 330

Adrenal Insufficiency (suspected autoimmune)
Hydrocortisone started
Weaned off pressors
Confusion/dizziness resolved

Labs continued

ANA <1:80
AMA <1:20
ASMA <1:20
IgG 853

A1AT 72 (L)
A1AT mutation NOT detected

Ceruloplasmin 13.7 (L)
Alpha Fetoprotein 2.86
Acetaminophen <10
Salicylates <2.5

HepB surface Ab Reactive
HepB surface Ag Non-Reactive
HepBe Ag Non-Reactive
HepBe Ab Non-Reactive
HepC Ab Non-reactive
HepA IgG Non-Reactive

PEth Negative

CMV IgM Negative
CMV Quant Negative
EBV IgG Positive
EBV IgM Positive
EBV Quant <35

Labs continued

Ferritin 636
Iron 89
TIBC 154
%Sat 58
HFE gene heterozygous for C282Y

Folate >22.3
Vitamin B12 >1500
Vitamin D 42

Thiamine 7 (L)
Zinc 39 (L)

UDS Negative

HIV I/II Ag/Ab Non-Reactive

Histoplasma Ag Negative

21-Hydroxylase Ab Negative
Thyrotropin Receptor Ab Negative

Imaging continued

CT Chest w/ Contrast:

- Cardiomegaly with findings of pulmonary edema and small R>L pleural effusions
- Mediastinal lymphadenopathy
- Multinodular thyroid goiter
- Mild superior endplate compression fracture of T6 is new, age indeterminate

CT Abdomen and Pelvis w/ Contrast:

- Cirrhosis. No suspicious hepatic lesions. Liver is markedly steatotic relative to previous PET (5yrs prior), slightly enlarged (15.9cm)
- Signs of portal venous hypertension including trace ascites, venous collaterals
- Gallbladder sludge and stones without signs of gallbladder inflammation.
- Portal colopathy

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BUE swelling R>L

More Imaging

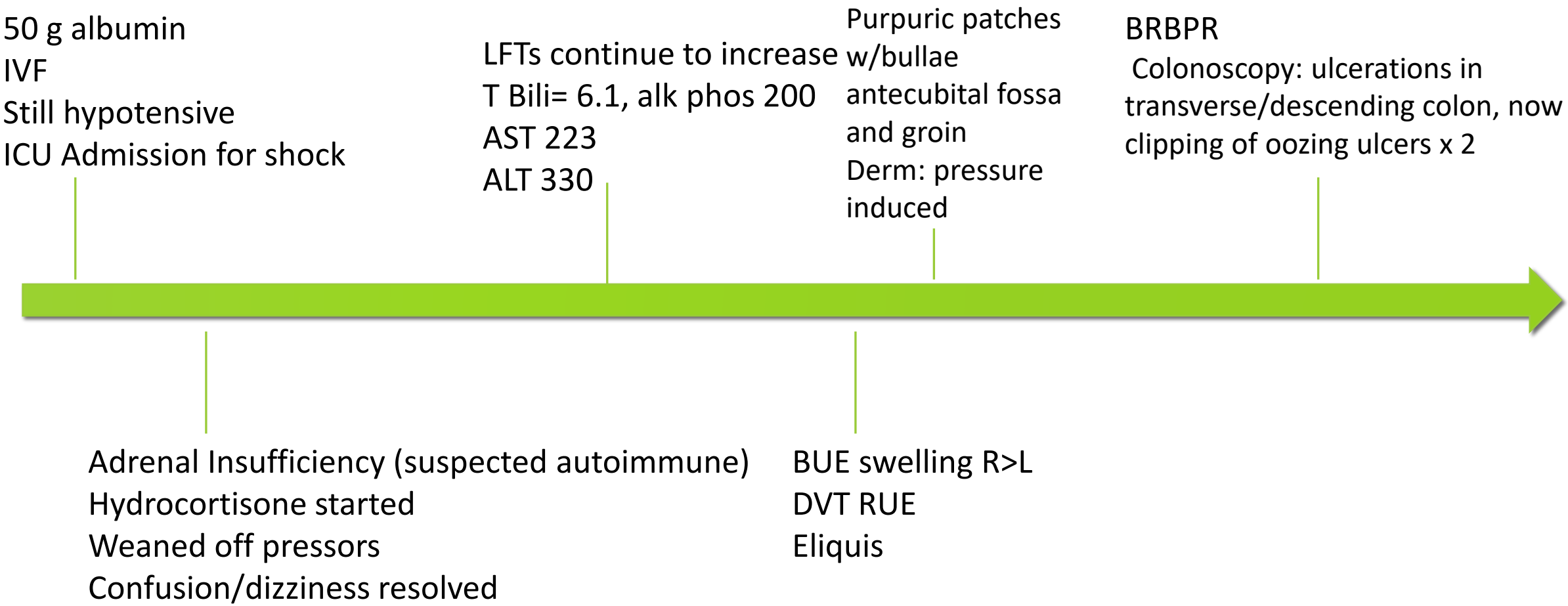
ECHO:

Ejection Fraction = 55-60%.
There is mild concentric left ventricular hypertrophy.
The diastolic function is indeterminate.
The right ventricular systolic function is normal.
The left atrium is moderately dilated.
No significant mitral valve stenosis.
There is no pericardial effusion.

Upper Ext DVT US:

There is a short segment of nonocclusive thrombus in the medial right subclavian vein. Occlusive superficial thrombus is noted in the right basilic and left cephalic veins.

Case 1 Evolution



Labs continued

Kappa FLC 3151
Lambda FLC 8.4
FLC Ratio 375

SPEP:
Free kappa light chain M-spike = 0.10 g/dL

IFE
Monoclonal free kappa light chains

UPEP
Free Kappa Light Chain M-spike = 90% of total urine protein

U IFE
Positive for free kappa light chain M-protein

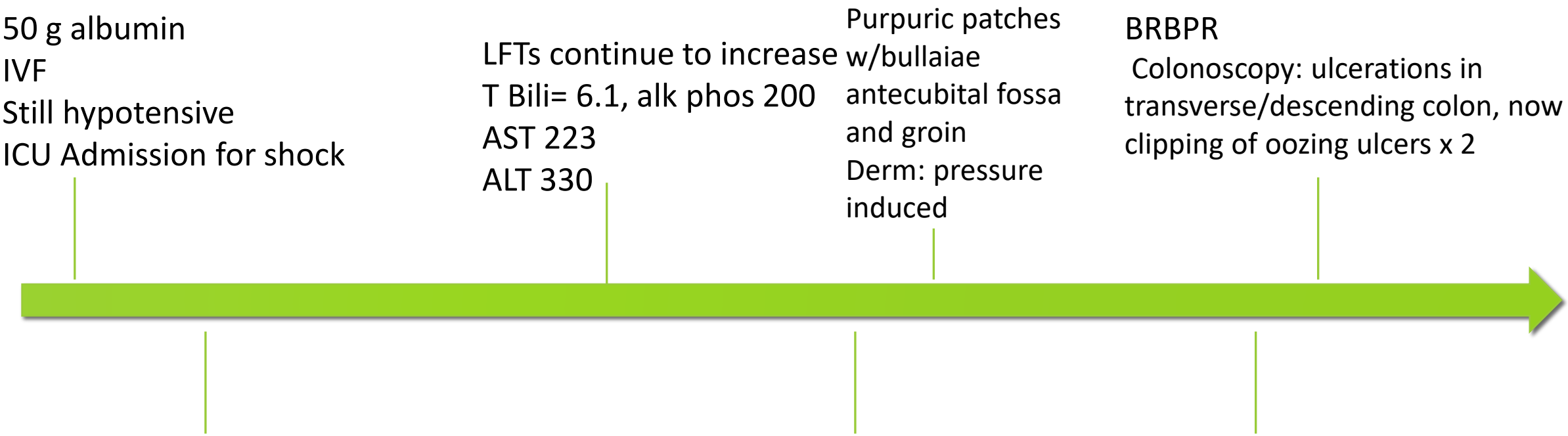
Pathology

Diagnostic Colonoscopy:

- "Right sided ulcers," biopsy:
  - Fibrinoinflammatory exudate consistent with ulceration
  - Congo red stain is positive for amorphous material

- "Polyp," biopsy:
  - Tubular adenoma and lymphoid aggregate.
  - Congo red stain is positive for amorphous material

Case 1 Evolution



Final Diagnosis

AL Amyloidosis

w/ definite GI involvement
Likely adrenal, liver, cardiac and skin involvement

Amyloidosis

Native proteins misfold (into beta-pleated sheet configuration) & self-assemble into rigid, unbranched fibers (fibrils)

The previously soluble proteins, now misfolded, are insoluble & deposit in tissues, causing end-organ damage

Amyloid deposits in tissues have birefringence with polarized light microscopy when stained with Congo red, which has a typical "apple-green" appearance

Causes

19 described types of systemic and 28 localized forms of amyloidosis from different native proteins

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Common Types of Amyloidosis	Misfolded protein	Primary Organs affected	%
AL amyloidosis	Monoclonal immunoglobulin light chain fragments due to plasma cell dyscrasia (MM, Waldrenstroms, lymphoma, CLL) ★ ★ ★	Kidneys, heart, GI tract, PNS	56%

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ATTR amyloidosis	Transthyretin (prealbumin)	Heart, PNS	21%

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AA amyloidosis	Acute phase reactant serum amyloid A protein from disease with ongoing inflammation: RA, spondyloarthritis, IBD, chronic infections	Kidneys, liver, GI tract	8%

Causes

19 described types of systemic and 28 localized forms of amyloidosis

Rarer types of Amyloidosis	Misfolded protein
Dialysis-related amyloidosis	Beta2-microglobulin
AApoCII	Hereditary misfolding of Apolipoprotein C-II (ApoCII)
	And many, many more…

AL Amyloidosis

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Adrenal involvement with AL Amyloidosis is described, but rare

Amyloid Mental Triggers

- New nondiabetic proteinuria
- New CHF with low voltage EKG
- New autonomic instability
- Bilateral Carpal Tunnel syndrome
- Macroglossia
- Easy bruising/bleeding (skin purpura)
- Unexplained organ enlargement

Amyloidosis Diagnosis

Biopsy

- Affected organ, abdominal fat pad, or bone marrow

AL Amyloid: free light chains, SPEP, IFE

Amyloidosis Treatment

AL Amyloidosis:

- Treatment by Hematology/Oncology
- Daratumumab, cyclophosphamide, bortezomib, and dexamethasone (Dara-CyBorD) vs Hematopoietic stem cell transplant

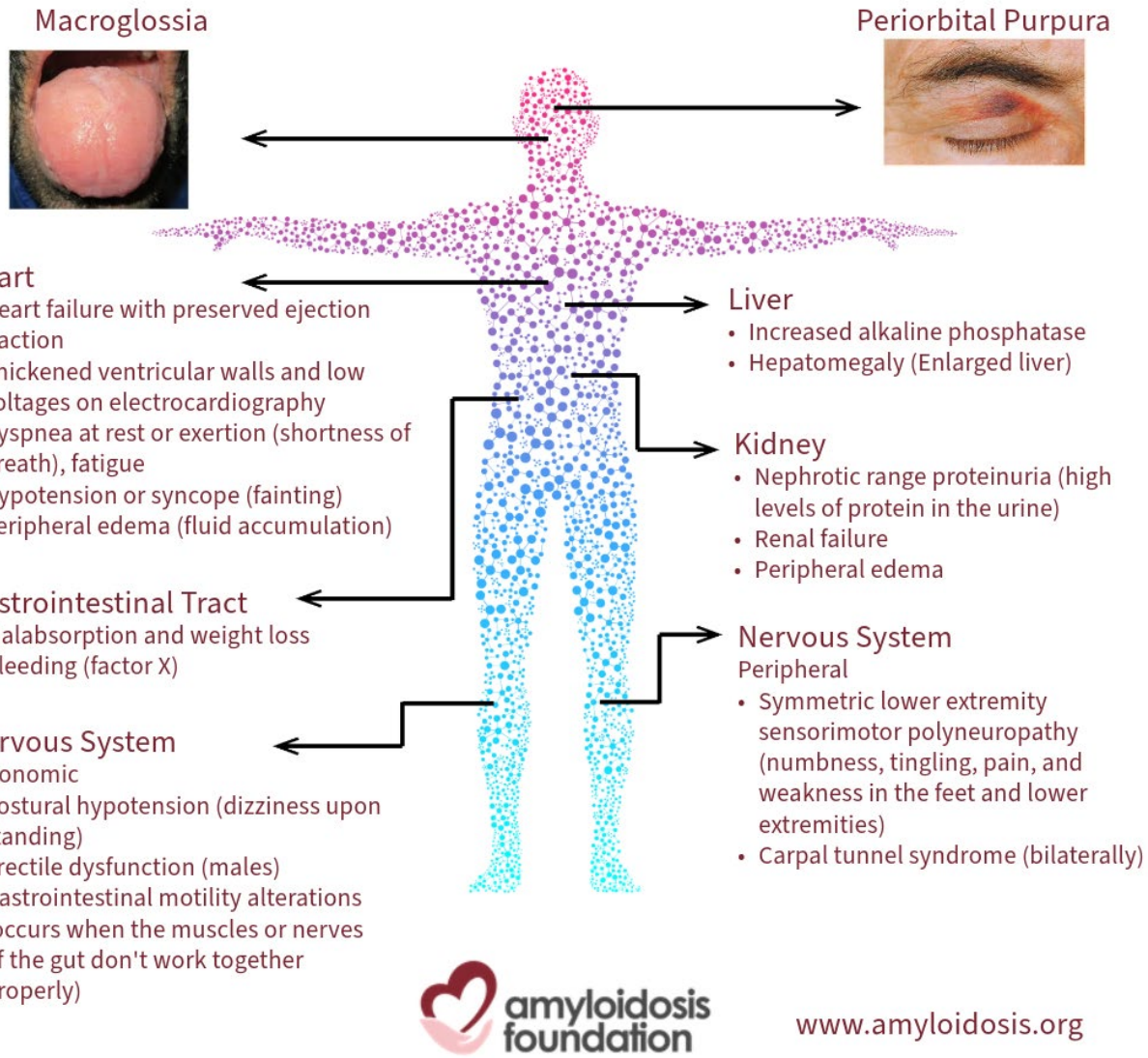
ATTR Amyloidosis (NYHA class I to III HF symptoms):

- tafamidis, acoramidis, or vutrisiran
- Prevents misfolding of transthyretin reducing new amyloid deposition

AA Amyloidosis:

- Treat the underlying inflammatory condition

Organs involved with AL Amyloidosis



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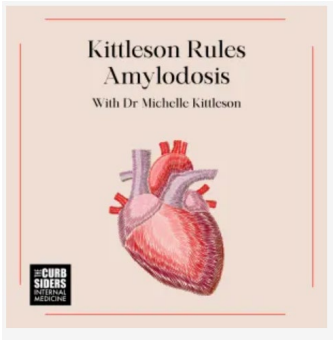
Ruberg FL, Maurer MS. Cardiac Amyloidosis Due to Transthyretin Protein: A Review. *JAMA.* 2024;331(9):778–791. doi:10.1001/jama.2024.0442

Uptodate

Listening to Learn



#427 Kittleson Rules Amyloidosis

Case 2

A 26-year-old man presents to establish primary care after moving to Birmingham

No complaints or issues

Past Medical History

Hypertension (dg 23yo): well controlled on Olmesartan/HCTZ

Elevated Liver Enzymes: as a teenager, while takingisoretinoin for acne

History continued

Review of systems

Completely negative

Except:

Slight lightheadedness when rising from a seated position too quickly

Muscle soreness after strenuous exercise (e.g. 2 hours of basketball)

Past Surgical History

None

Social History

Lifelong nonsmoker, noetoh/drug use

Internal Medicine Intern

Family History

Father, 60yo: good health

Mother, 55yo: HLD

Older brother: good health

Grandfather: deceased from MI in 70s

No FH of HTN or premature CV disease

Physical Exam

Vitals: T: 98.6 HR: 62 BP: 124/78 BMI: 24

General: This is a very pleasant, white male sitting up in chair in no acute distress.
HEENT: Normocephalic. Atraumatic. Extraocular movements: Intact. Pupils equal, round and reactive to light.
Chest: Clear to auscultation bilaterally. No wheezes, rales or rhonchi.
Cardiovascular: Regular rate and rhythm. No murmurs, rubs or gallops.
Abdomen: Bowel sounds positive. Soft. Nontender and nondistended.
Extremities: No clubbing, cyanosis or edema. Dorsalis pedis: 2+.
Skin: No rashes. No lesions.
Neuro: Moves all extremities well. Gait is intact.
Psych: Normal affect. Good eye contact.

Labs

140	104	27	95
4.2	30	1.1	

Ca: 9.3

Mag: 2.0

TP	6.5	4.1	Alb
TBili	0.6		
AST	295	100	ALT
АФ	55		

6.8	14	178
	40	

MCV: 92

History Continued

Elevated Liver Enzymes:

16yo: ALT 117 and AST 106 on routine labwork (on isoretinoin for acne tx)

Bilirubin, alkaline phosphatase, PT/PTT, and albumin normal

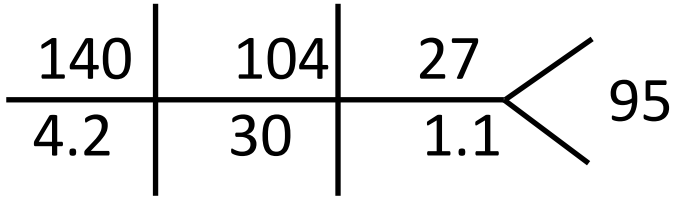
Negative screening: hemochromatosis, Wilson's disease, hepatitis A,B,C

Liver biopsy done without signs of inflammation or fibrosis

18yo: repeat ALT of 56 and AST of 94

Second liver biopsy normal, diagnosis of idiopathic liver enzyme elevation

Labs



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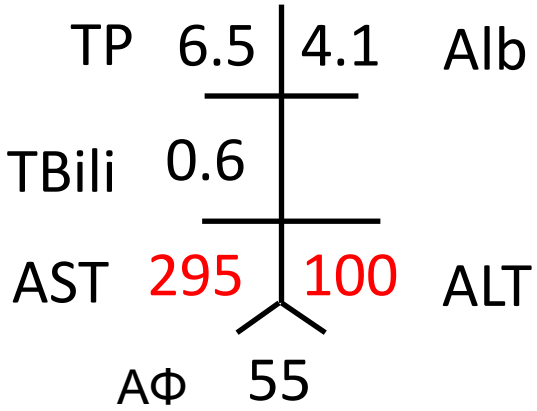
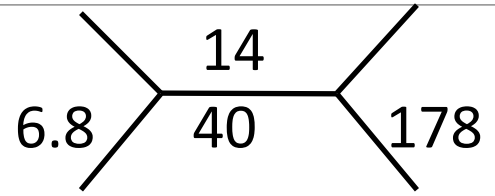
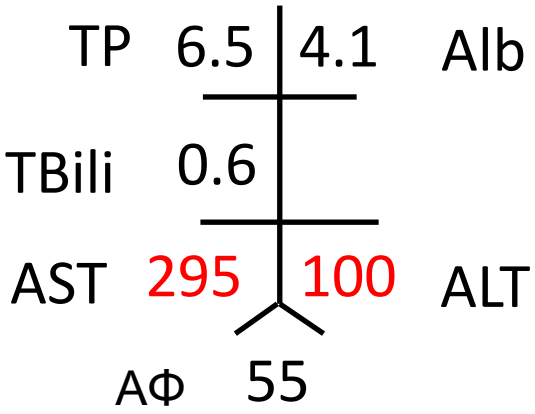




MCV: 92

Lab add on: CK 11,778

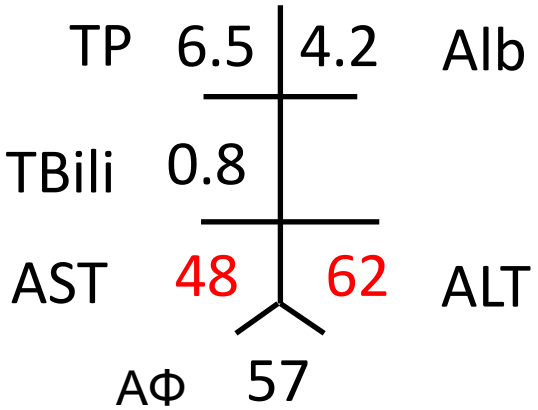
Labs, continued



CK 11,778

Held Benicar/HCTZ
(Case reports)

No strenuous activity
X9 days



CK 1,122

Exercise Rechallenge

CK 8,341

3 weeks later CK 1,022

Additional Labs

HIV: neg

TSH: 1.2

ESR: 0

ANA: <1:80

RF: negative

IgA: 230

IgG: 1,040

IgM: 88

EMG/NCS

NCS:

1. Normal motor NCS in the right median, ulnar, posterior tibial, and peroneal nerves.
2. normal sensory NCS in the right median, ulnar, and sural nerves.

EMG:

1. Fibrillations and the right biceps and vastus lateralis muscles.
2. SASD MUPs in the right biceps, deltoids, and iliopsoas muscles.

These findings are indicative of active myopathy.

Further History

Christmas break

Father:

- Muscle soreness after exercise too
- hx of hyperCKemia in his early 40s with ck of around 10K
- Ischemic forearm test (to detect metabolic causes of muscle weakness by measuring lactic acid levels after several minutes of repetitive exercise while blood flow is occluded to the forearm) = negative
- EMG/NCS
- Muscle biopsy

- Myopathy of unknown cause

- Father noticed to have large calf muscles

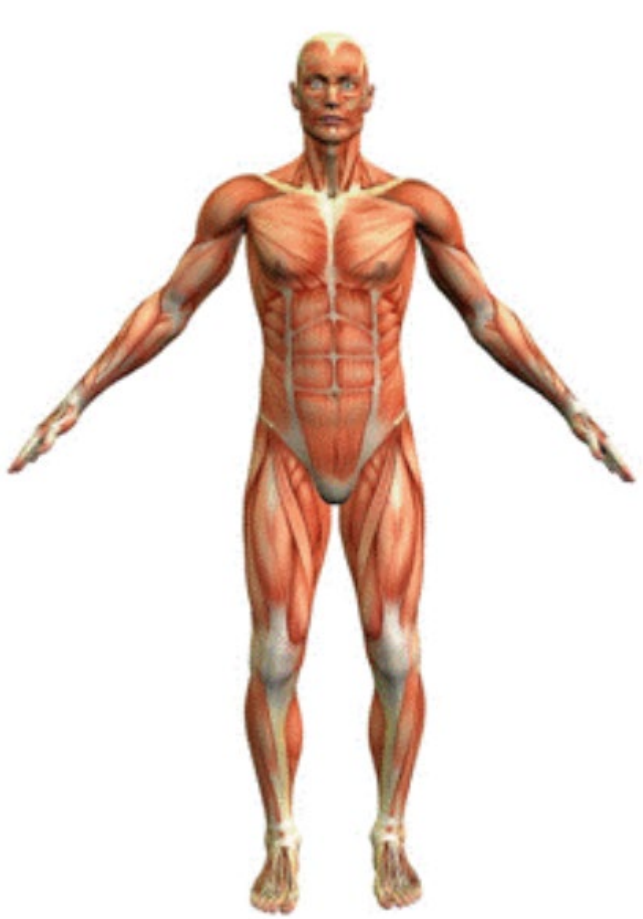
Gene Sequencing Testing

Autosominal Dominant limb-girdle muscular dystrophy type 1C
(CAV 3 mutation, caviolinopathy)

Aminotransferases (AST/ALT)

- Enzymes that catalyze reactions that convert alanine and aspartate into alpha-ketoglutarate, providing a source of nitrogen for the urea cycle
- Highest concentration in the liver
- Also found in other tissues: muscles, heart, kidneys, erythrocytes

Lash T, Kraemer RR. Elevated liver enzymes indicating a diagnosis of limb-girdle muscular dystrophy. J Gen Intern Med. 2014 May;29(5):813-5



ALT: 4x

AST: 26x

Table 2. Extrahepatic Causes of Elevated Transaminases

Rhabdomyolysis
Aquired disorders of muscle
Inflammatory myopathy
Vasculitis
Etc.
Congenital disorders of muscle
Muscular dystrophy
Inborn errors of metabolism
Etc.
Exercise
Heat stroke
Seizure
Sepsis
Renal failure
Thyroid disorders
Diabetes
Hemolysis
Myocardial infarction
Adrenal insufficiency
Anorexia nervosa
Celiac disease

→ RBC AST/ALT ratio 40:1

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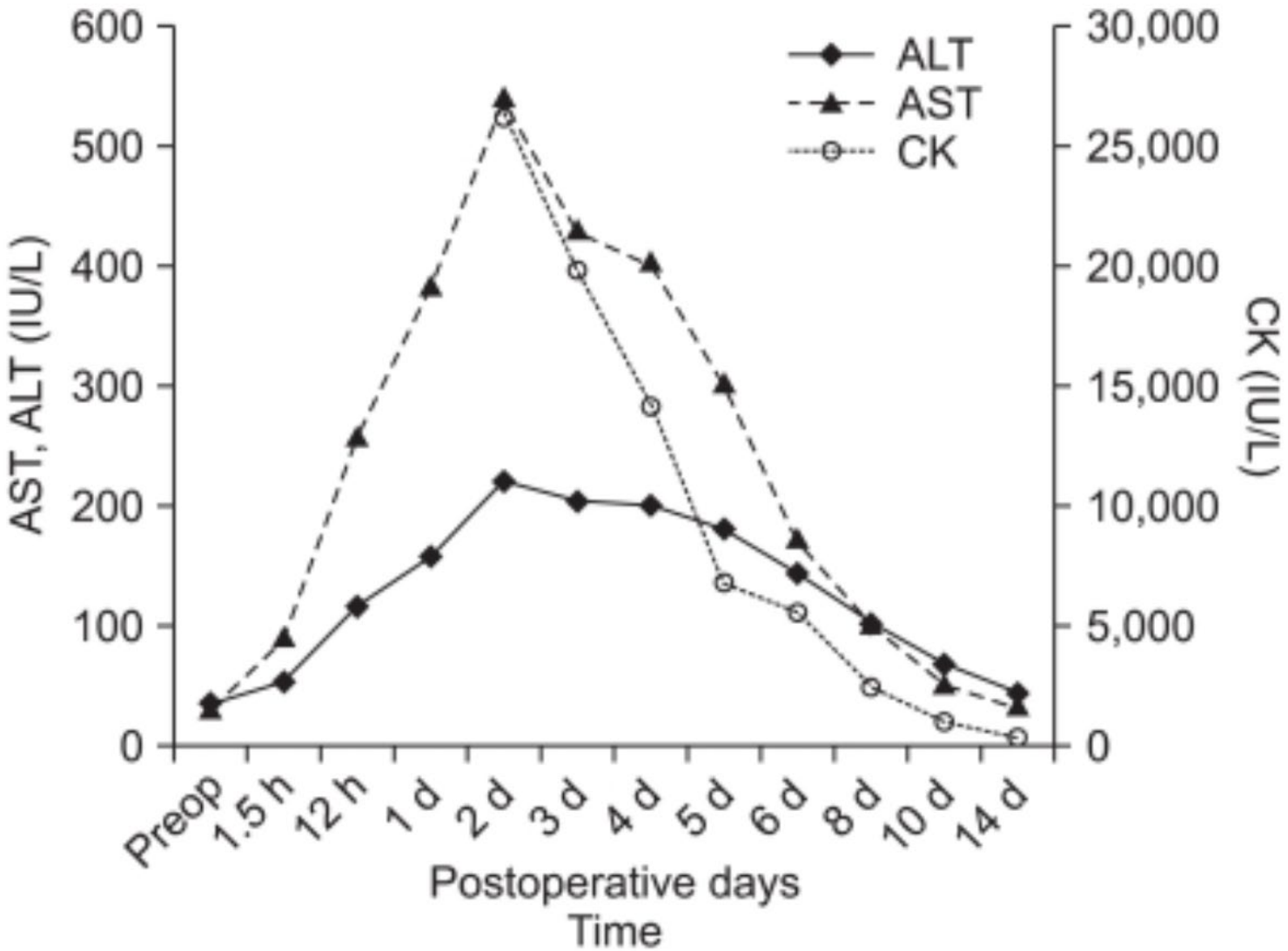
When CK >1,000:

- 93% elevated AST
- 71% elevated ALT

- Average ratio AST 3 / ALT 1 with initial injury

- AST half life= 17 hours
- ALT half life = 47 hours

- Average ratio 1:1 after 3 days

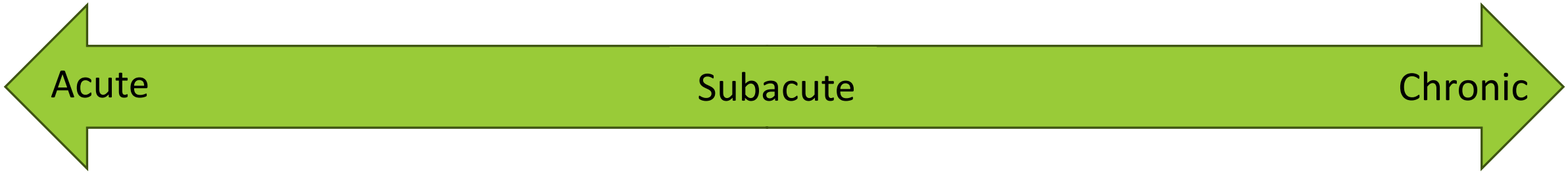


Nathwani RA, Pais S, Reynolds TB, Kaplowitz N. Serum alanine aminotransferase in skeletal muscle diseases. Hepatology. 2005;41(2):380–2.

Price CP, Alberti KGMM. Biochemical Assessment of Liver Function. In: Wright R, Alberti KGMM, Karran S, Millward-Sadler GH, eds. Liver and Biliary Disease—Pathophysiology, Diagnosis, Management. London: W.B. Saunders; 1979:381–416.

Lee G, Lee H, Kim Y. Rhabdomyolysis recognized after elevation of liver enzymes following prolonged urologic surgery with lateral decubitus position -A case report- Korean Journal of Anesthesiology 2011/01/01.

Approach to hyperCKemia



Rhabdomyolysis

Acquired Myopathy

Genetic Myopathy

Muscle weakness or asymptomatic?

Muscle pain and/or stiffness?

Exercise intolerance with or without rhabdomyolysis

Cardiomyopathy, cardiac arrhythmia, or respiratory insufficiency

Approach by Duration of Symptoms

- Acute = Rhabdomyolysis
  - Strenuous exercise, medications, toxins, substance use, hypokalemia, hypomagnesemia, viral infections, etc.
- Subacute = acquired myopathies
  - Thyroid, HIV, toxins, meds (statins), critical illness
  - Inflammatory myopathies = inflammatory myopathy panel
    - Immune mediated necrotizing myopathy (Anti-SRP, Anti-HMGCR)
  - EMG/NCS + biopsy
- Chronic = genetic myopathies
  - Rule out acquired causes & EMG/NCS
  - DMD gene test & Next Generation Sequencing gene panel
  - If negative, muscle biopsy/additional genetic testing for mitochondrial disorders, other disorders not detected on NGS

Courtesy of Teerin Liewluck, MD.

UpToDate®

Muscular Dystrophy

- Inherited disorders of genes for normal muscle function
- Clinical Manifestations: mild asymptomatic hyperCKemia to severe weakness/debility/early death
- Duchenne Muscular Dystrophy: severe weakness from early childhood
- Becker's Muscular Dystrophy: weakness onset 5-60yo
  - Due to genetic defects in the dystrophin gene (x chromosome) which results in muscle degradation

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UAB

Limb-Girdle Muscular Dystrophies

Autosominal Dominant limb-girdle muscular dystrophy type 1C (CAV 3 mutation, caviolinopathy)

- Defect in the caveolin 3 gene in the sarcolemma of skeletal muscle which helps maintain plasma membrane integrity
- Can cause **Rippling Muscle Disease:** Direct percussion of the muscle produces continuous rolling, rippling waves of muscle contractions that spread across the muscle

Other types of LGMD: Dysferlinopathies, Collagenopathies, Sarcoglycanopathies, Anoctaminopathies, Dystroglycanopathies, etc

Autosomal Dominant LGMD type 1C = good prognosis

JGIM

Clinical Practice: Clinical Vignettes

Elevated Liver Enzymes Indicating a Diagnosis of Limb-Girdle Muscular Dystrophy

[redacted], *MD*[1] *and Ryan R. Kraemer, MD*[2]

[1]Tinsley Harrison Internal Medicine Residency Program, The University of Alabama at Birmingham, Birmingham, AL, USA; [2]Division of General Internal Medicine, The University of Alabama at Birmingham, Birmingham, AL, USA.

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