WELCOME TO
AK LIVER DISEASE ECHO

ALASKA NATIVE TRIBAL HEALTH CONSORTIUM

NPAIHB
Indian Leadership for Indian Health

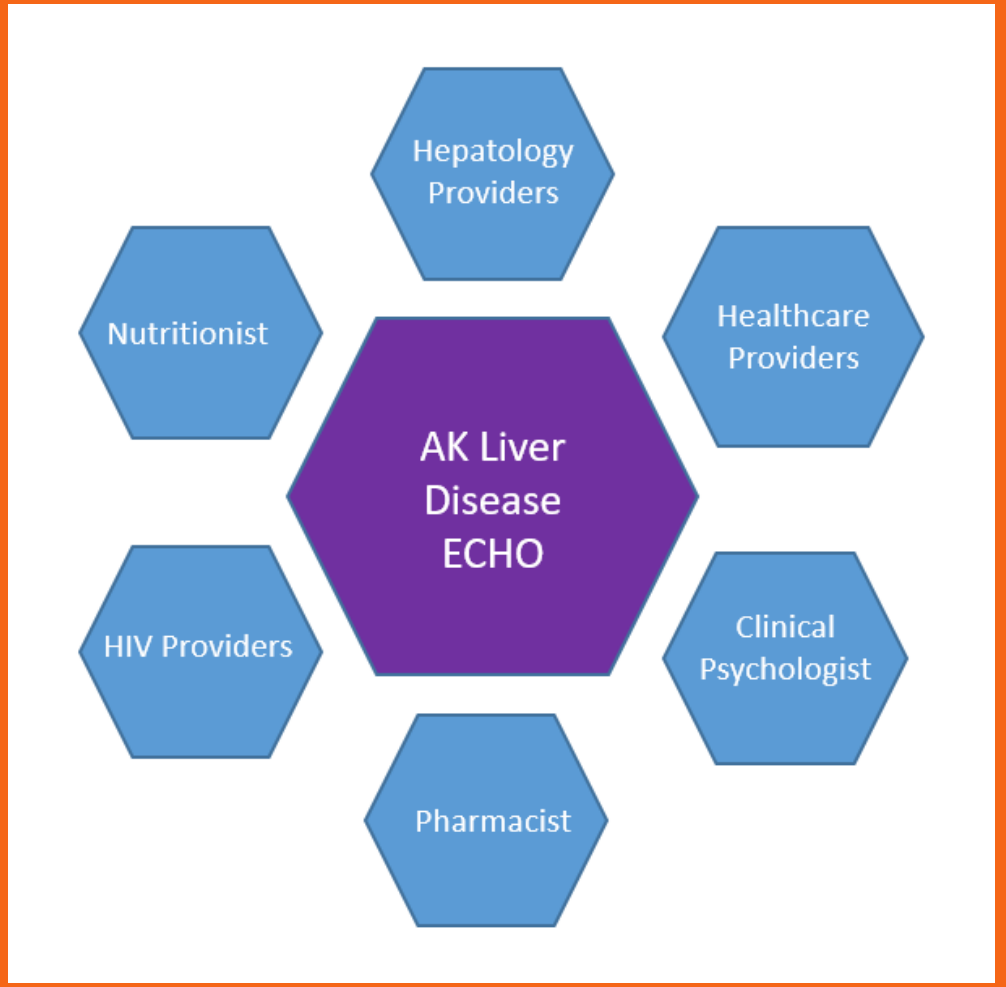
This project is supported by a grant from the Northwest Portland Area Indian Health Board and funding is provided from the HHS Secretary's Minority HIV/AIDS Fund.

WHAT WE DO

- Didactic Presentations pertaining to ECHO topics
- We're accepting **case presentations and questions pertaining to:**
  - Elevated Liver Function Tests
  - Cirrhosis
  - Managing Complications of Decompensated Cirrhosis – Ascites, encephalopathy, esophageal varices
  - Alcohol-related liver disease, including Alcohol Hepatitis
  - Autoimmune liver disease – Autoimmune Hepatitis, Primary Biliary Cholangitis, Overlap
  - Nonalcoholic fatty liver disease/Nonalcoholic steatohepatitis
  - Hepatocellular carcinoma
- Provide Expert Panelists

CONSULTANT TEAM

- Brian McMahon, MD Hepatologist
- Youssef Barbour, MD Hepatologist
- Lisa Townshend, ANP Hepatology Provider
- Annette Hewitt, ANP Hepatology Provider
- Leah Besh, PA-C HIV/Hepatology Provider
- Anne Fleetwood, MS, RDN, NDN
- Brittany Keener, PharmD, MPH, BCPS
- Rebecca Robinson, PhD Clinical Psychologist



Welcome to Alaska Liver Disease ECHO

Approved Provider Statements:

JOINTLY ACCREDITED PROVIDER™
INTERPROFESSIONAL CONTINUING EDUCATION

In support of improving patient care, Alaska Native Medical Center (ANMC) is jointly accredited by the Accreditation Council for Continuing Medical Education (ACCME), the Accreditation Council for Pharmacy Education (ACPE), and the American Nurses Credentialing Center (ANCC), to provide continuing education for the healthcare team.

Contact Hours:

ANMC designates this activity for a maximum of 12 contact hours, including 3 total pharmacotherapeutics contact hours, commensurate with participation.

Financial Disclosures:

Youssef Barbour, MD & Lisa Townshend-Bulson, APRN / faculty for this educational event, are primary investigators in an ANTHC sponsored hepatitis C study funded in part by Gilead Sciences. All of the relevant financial relationships listed have been mitigated.

Requirements for Successful Completion:

To receive CE credit please make sure you have actively engaged in the entire activity, your attendance is recorded by the facilitator, and complete the course evaluation form found here: https://forms.gle/R8vibUZgMbRcoScw9.

For more information contact
jlfielder@anthc.org or (907) 729-1387

ALASKA NATIVE TRIBAL HEALTH CONSORTIUM

LIVER DISEASE ECHO SCHEDULE AT A GLANCE

- December 16th Portal Vein Thrombosis – Youssef Barbour, MD
- Stay tuned for our 2022 LD ECHO calendar!

LAND ACKNOWLEDGEMENT

Out of great respect, we acknowledge the Creator of all living things and the original inhabitants upon whose traditional lands we respectively now gather or reside.

Let us, forever, remember the benefits and bounty of the vast resources that stem from these traditional lands, and thank the original inhabitants for their past and present stewardship of the resources (i.e., waters, plants, animals), and the spiritual practices which they cherish on the lands we now call home.

PORTAL VEIN THROMBOSIS (PVT)

YOUSSEF BARBOUR MD

ANTHC LIVER DISEASE & HEPATITIS PROGRAM

DISCLOSURE

No conflict of interest to disclose for this presentation

This presentation is based on AASLD 2020 practice guidance published in Hepatology, https://doi.org/10.1002/hep.31646

WHICH STATEMENT IN THE FOLLOWING IS CORRECT

- A. thrombophilia (a tendency for blood clotting or hypercoaguble state) work up is indicated for all patients diagnosed with PVT
- B. treatment is indicated for all patients with PVT
- C. DAOCs (Direct Acting oral anticoagulants) are contraindicated for the treatment of PVT
- D. EVL (EGD with rubber band ligation) can be done while patient is getting anticoagulation for PVT
- E. PVT is a progressive disease

TERMINOLOGY AND CLASSIFICATIONS

- PVT is a heterogeneous condition with respect to etiology, manifestations, natural history, and therapeutic options. For this reason, terminology and classification systems also vary extensively in the literature.
- Guidance statement: In any patient with PVT, a standardized documentation of initial site, extent, degree of luminal obstruction, and chronicity of clot formation is recommended in order to make objective serial assessments of spontaneous regression or treatment response.

DESCRIPTION OF PVT

Time course: ***Recent:*** PVT presumed to be present for <6 months

Chronic: PVT present or persistent for >6 months

Percent occlusion of main PV:

Completely occlusive: No persistent lumen

Partially occlusive: Clot obstructing >50% of original vessel lumen

Minimally occlusive: Clot obstructing <50% of original vessel lumen

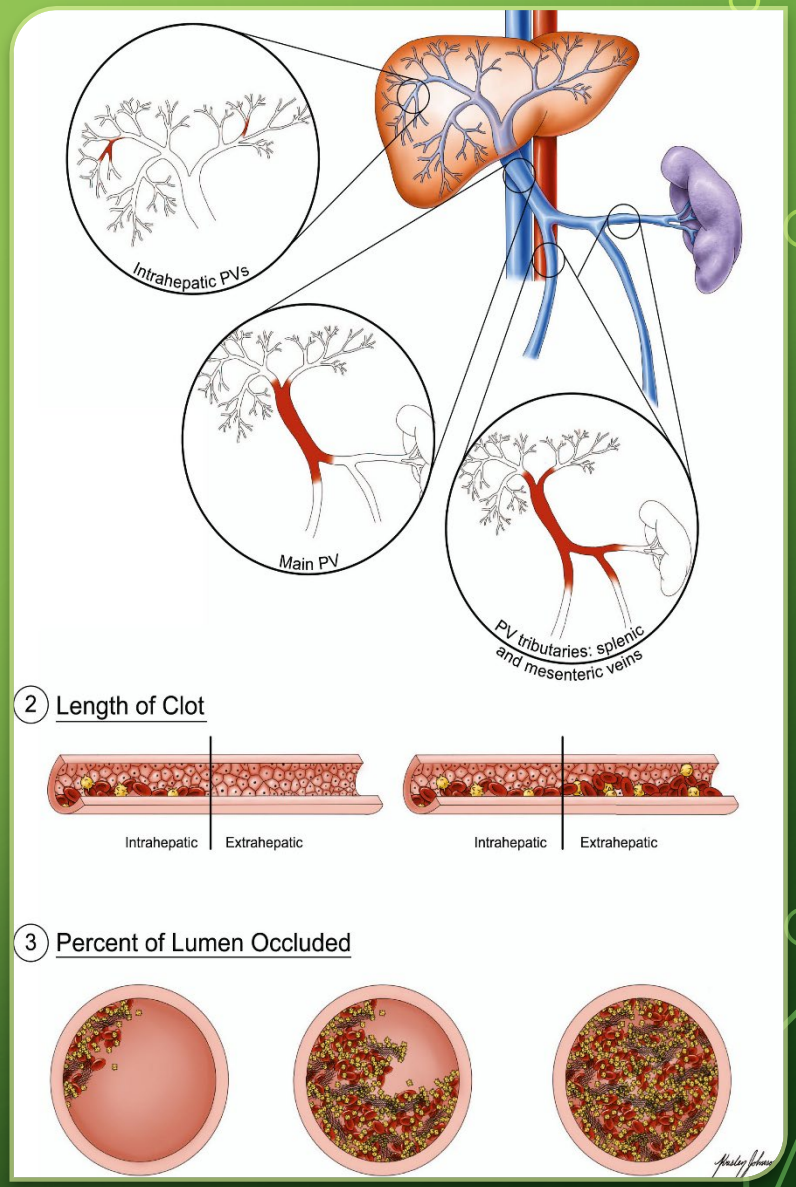
Cavernous transformation: Gross portoportal collaterals without original PV seen

Response to treatment or interval change:

Progressive: Thrombus increases in size or progresses to more complete occlusion

Stable: No appreciable change in size or occlusion

Regressive: Thrombus decreases in size or degree of occlusion



RISK FACTORS: IN CIRRHOSIS

- **Severity of portal HTN and liver disease** in patients with cirrhosis
- **Decreased velocity of PV flow** at baseline in patients with cirrhosis (ranging from 10-15 cm/s by Doppler
- **Obesity, metabolic syndrome and NASH** are recognized independent risk factors
- **HCC** invading the PV and/or HV (HCC should be ruled out with every new PVT diagnosis
- A meta-analysis demonstrated an increased incidence in PVT in patients with cirrhosis taking **nonselective beta-blockers**, however, analysis of confounding variables in this study has raised questions about the independent effect of nonselective Beta-blockers in development of PVT
- The potential role of an **altered endothelium** has been under investigated and data on the role of **inflammation/bacterial translocation** are still limited
- Thrombophilia work up is not routinely recommended in patient with cirrhosis unless clinically indicated

RISK FACTORS: PATIENTS WITHOUT CIRRHOSIS

- Rare, require hematology consultation
- Require thrombophilia work up, and rule out of myeloproliferative neoplasia. (the G20210A prothrombin mutation is most prevalent inherited thrombophilia to cause PVT)
- Surgery or inflammatory conditions affecting the digestive system organs or the spleen.

INFLUENCE OF PVT ON MORTALITY IN PATIENTS WITH CIRRHOSIS

- Guidance Statements:
- 1- outside of LT candidates, it is unknown whether PVT in an individual patient with cirrhosis is merely a reflection of progressive portal HTN or independently causative of increased mortality
- 2- in LT recipients, the presence of PVT at the time of transplant is associated with increased post transplant mortality
- 3- there are insufficient data to recommend pre transplant treatment of PVT with goal of improving post transplant outcomes

GOALS OF THERAPY AND RATIONALE FOR TREATMENT:

PATIENTS WITH CIRRHOSIS

- In patients **with cirrhosis,** existing clinical trial data are weak regarding treatment indications for PVT without ischemic symptoms. **Treatment should be considered on a case-by-case basis.** Decisions for treatment of an individual patient should be based on **expected benefit and minimization of clot extension risk that could potentially lead to progression of portal hypertension or hinder LT.**
- In patients with cirrhosis who have **recent** thrombosis of small **intrahepatic sub-branches** of the PV or **minimally occlusive** (<50% obstruction of the lumen) thrombosis of the **main PV**, *observation with serial imaging every 3 months without therapy is reasonable. Treatment for progressive clot should then be considered in this setting.*

- In patients with cirrhosis with **recent** **occlusive or partially occlusive** (>50% obstruction of the lumen) thrombosis of the **main PV or mesenteric veins**, ***antithrombotic therapy should be considered to avoid thrombosis progression that may hinder a future LT or cause progression of portal hypertension.***
- In patients with **chronic** **complete occlusion of the main PV or cavernous transformation of the PV** with established collaterals, there is *no established benefit of anticoagulant or interventional therapy, and treatment should be targeted at management of portal hypertension complications.*
- Data suggest that **EVL** can be performed safely without stopping therapeutic anticoagulation. Based on the available safety data, **anticoagulation should be initiated as soon as possible and not delayed until variceal eradication or adequate beta-blockade is achieved.**

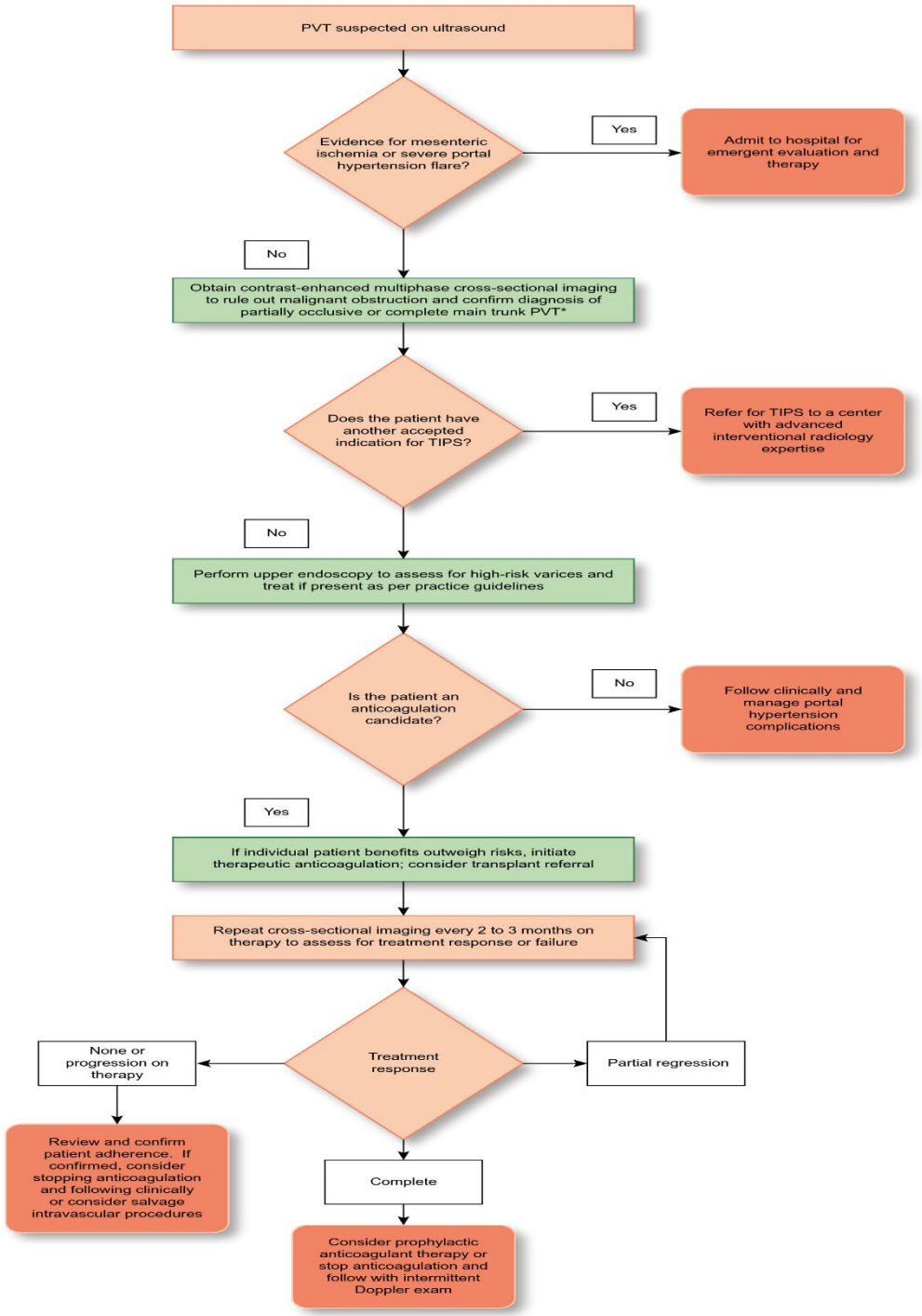
GOALS OF THERAPY AND RATIONALE FOR TREATMENT:

PATIENTS WITHOUT CIRRHOSIS

- In patients **without cirrhosis** and with recent PVT, directed antithrombotic therapy should be considered in order to avoid intestinal ischemia and prevent the development of chronic PVT with portal hypertension.

IN ALL PATIENTS WITH RECENT PVT AND CONCERN FOR INTESTINAL ISCHEMIA, IMMEDIATE CONSULTATION WITH SURGERY, CRITICAL CARE, INTERVENTIONAL RADIOLOGY, AND HEMATOLOGY IS ADVISED. ANTICOAGULATION IS ESSENTIAL, WITH THE NEED FOR SURGERY IN CASES OF INTESTINAL INFARCTION.

If minimally occlusive (<50% obstructed main PV lumen), serial imaging to assess for spontaneous regression in 2-3 months before intervention is reasonable.



TREATMENT OPTIONS

- Thrombolysis and interventional vascular procedures
- Medical therapies (VKAs, LMWH, DAOCs)

- Local or systemic thrombolytic therapy should only be considered in very selected cases of recent PVT in whom intestinal ischemia persists despite anticoagulation.
- PVR followed by TIPS should be considered in LT candidates with chronic PVT that hinders a physiological anastomosis between the graft and recipient PV. This decision is made as part of a multidisciplinary management process, including surgical and interventional radiology expertise.
- PVR followed by TIPS should be considered in patients with chronic PVT and recurrent bleeding and/or refractory ascites not manageable medically or endoscopically.
- The choice of agent for anticoagulant therapy (LMWH (low molecular weight heparin), VKAs (vitamin K antagonists), and DOACs) in PVT should be individualized. Consultation with a hematologist and/or expert hepatologist should be considered in deciding on anticoagulant agents and duration.
- Therapeutic anticoagulation in patients with cirrhosis appears to have similar non-portal hypertensive bleeding complication rates compared to the general population. Portal hypertension–related bleeding in patients with cirrhosis appears unchanged by the use of anticoagulants.
- DOACs are emerging as a common therapy for general medical patients with thrombosis. PVT data remain limited regarding safety and efficacy of these agents in patients with and without cirrhosis. In patients with cirrhosis, caution is advised in patients with advanced portal hypertension, and expert consultation is recommended.

ABSTRACTS FROM AASLD 2021

ANTICOAGULATION FOR PORTAL VEIN THROMBOSIS IS SAFE AND EFFECTIVE IN PATIENTS WITH CIRRHOSIS

- This systematic review and meta-analysis shows that patients treated with anticoagulation had a statistically significant increase in recanalization rate as well as improved survival.
- • The treated group additionally demonstrated significantly fewer variceal bleeding events compared to the untreated group. This may be due to the prevention of further progression of portal hypertension, thus reducing the likelihood of variceal bleeding and conferring a survival benefit in these patients.
- • Therefore, the current data suggest that anticoagulants may be safe and effective in patients with cirrhosis and PVT.

PORTAL VEIN THROMBOSIS IMPACTS ON SURVIVAL INCIRRHOTICS WITH HEPATOCELLULAR CARCINOMA

- HCC was identified as an independent risk factor for the development of PVT, presenting apparently lower rates of spontaneous recanalization compared to patients without HCC
- Baseline larger tumor volume and lack of response to ablative treatment would appear to increase the risk of PVT and predict thrombosis progression, respectively
- Complete/progressive PVT was an independent factor associated with mortality, therefore thromboprophylaxis should be evaluated in case of high tumor volume or lack of response to treatments.

COMPARATIVE EFFICACY AND SAFETY OF DIRECT-ACTING ORAL ANTICOAGULANTS WITH VITAMIN K ANTAGONIST FOR CIRRHOSIS PATIENTS AND PORTAL VEIN THROMBOSIS: A SYSTEMATIC REVIEW AND META-ANALYSIS.

- We included a total of 11 studies• 4 types of DOACs (rivaroxaban, apixaban, edoxaban and abigatran) were evaluated
- For the treatment of PVT in patients with cirrhosis, the bleeding risk was comparable between DOACs and VKAs. However, DOACs were associated with a higher pooled rate of PVT recanalization. While these findings are likely applicable to patients with CTP-A and -B cirrhosis, dedicated randomized studies are needed to confirm this finding.

THE IMPORTANCE OF PORTAL VEIN THROMBOSIS IN PATIENTS WITH CIRRHOSIS – IMPACT OF TIMING

- In conclusion, we have shown that when a patient presents with PVT at their first presentation of cirrhosis, their outcomes are worse than if they develop PVT later in their course of cirrhosis.
- PVT seems to be associated with acute clinical decompensation in cirrhosis

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Alaska Liver Disease ECHO Historical Case

Presenter: Youssef Barbour, MD
Date: 12-16-21
ECHO ID#: LD-16H

General Information/Demographics

Age: 49 Sex at Birth: **x**M ☐F Gender Identity: Hispanic/Latinx: ☐Y xN

Race (check all that apply): xAlaska Native/American Indian ☐Asian ☐Black/ African American ☐Native Hawaiian/ Pacific Islander ☐White ☐Other: _____

Primary Question(s):

In the space below, please describe the primary question/concern you would like the ECHO team to address.

What is the presentation of someone with PVT?

Past Medical History

Alcohol use disorder with 10+ year history of heavy alcohol use, smoking.

Liver Related History (check all that apply)

☐ Autoimmune Hepatitis/ Primary Biliary Cholangitis/Overlap X Alcohol Related Liver Disease

☐ Hepatitis B ☐ Hepatitis C ☐ Hepatocellular Carcinoma (HCC)

☐ Non-Alcoholic Fatty Liver (NAFLD) ☐ Non-Alcoholic Steatohepatitis (NASH) ☐ Cirrhosis*

☐ Other (specify):

Comments (dx date, relevant details, etc.):

* Indicate any markers for decompensation: ☐ Ascites ☐ Encephalopathy ☐ Variceal Bleed

Presentation at rural hub: Epigastric abd pain, early satiety. Diagnosed with biliary colic. Given tramadol and pain improved somewhat but returned to ER with pain radiating across R abdomen and suprapubic region. Had 1 episode of N/V at onset of pain. No changes in bowel Habits, no black stools.

Subjective: Never had pain like this before. Pain worsens after eating a few bites and patient feels Bloated and uncomfortable.

Imaging

Include any imaging or transient elastrography results (eg. Ultrasound, FibroScan, CT, MRI/MRE, EGD).

Imagining Technique	Date	Results
CT and US done at rural Hub	3/7/15	Possible portal vein thrombosis.
Ultrasound (ANMC)	3/8/15	NO FLOW WITHIN THE SPLENIC VEIN FROM A CT OF THE PANCREAS AS WELL AS WITHIN THE MAIN PORTAL VEIN AND INTRAHEPATIC PORTAL VEINS WITHOUT HEPATIC MASS. THE HEPATIC VEINS ARE PATENT. FLOW IS NOTED TOWARDS THE LIVER WITHIN THE SPLENIC VEIN TO THE LEVEL OF THE PANCREATIC TAIL.

Patient Objectives/Labs

BMI:	Date:	Weight:	(☐ lbs ☐ kgs)	Height:	(☐ in ☐ cm)

LFTs: If available, provide historical LFTs separated by at least 1 month.

Lab	Result	Date	Result	Date	Result	Date
ALT	14					
AST	17					
Alk Phos	95					
Albumin	3.4					
T Bili	0.79					

Lab	Result	Date
WBC	6.63	
ANC	4.04	
HGB	13.9	
HCT	42.4	
Platelets	396	
PT	14.9	
INR	1.2	
Creatinine	0.9	
eGFR		
NA (if cirrhosis or alcohol hepatitis	139	
K+ (if cirrhosis or alcohol hepatitis)	3.4	
HgbA1C		
Triglycerides		
Cholesterol		
HDL		
LDL		
TSH		

Serology	Result	Date
HAV-Total (HepA Ab total)		
HBcAb (Hep B core antibody)		
HVsAb (Hep B surface antibody)		
HBV DNA (in/mL)		
HCV RNA (in/mL)		

Calculations*	Value
APRI (liver fibrosis)	0.11
FIB-4 (liver fibrosis)	0.48
MELD (cirrhosis)	7
CTP (cirrhosis)	5
FIBROSPECT	30

If patient has elevated LFTs of unknown etiology, please obtain these additional labs:

Lab	Result	Date
ANA		
Actin		
IgG (total)		
AMA (if alk phos is elevated		
IgM (if alk phos is elevated)		
Iron Total		
TIBC		
Iron Saturation %		
Ferritin		
HCV Ab Screen		
HIV Ab Screen		

* To calculate APRI, FIB-4, MELD, and CTP, visit: https://www.hepatitisc.uw.edu/page/clinical-calculators/apri
To calculate NAFLD Fibrosis Score, visit: https://www.mdcalc.com/
To calculate Hepatic Steatosis Index, visit: MDApp.co/hepatology/
For help with laboratory data/ calculations, contact Cynthia Decker (cadecker@anthc.org, 907-729-4636, TigerText) or Wileina Rhodes (wsrhodes@anthc.org, 907-729-1569, TigerText).

Medications

Medication	Dosage/Frequency/Comments	Medication (cont)	Dosage/Frequency/Comments
In hospital: Treated with Lovenox			
After discharge, placed on warfarin	5mg daily x 6 months		

Imaging

After treatment, follow up

Imagining Technique	Date	Results
(After treatment) Abd Ultrasound with flow studies	9/2/15	Hepatopetal flow is seen in the patent main, right, and left portal veins. Normal direction flow is seen within the patent right, middle, and left hepatic veins. Arterial tracing is seen in the hepatic artery. The splenic vein is patent.

Liver Disease Initial Presentation

Presenter: Leah Besh Date: 12/16/2021 ECHO ID#: LD-ECHO-15

General Information/Demographics:

Age: 44	**Sex at Birth:** ■ M ☐ F	**Gender Identity:** he/him	**Hispanic/Latinx:** ☐ Y ☐ N

Race (check all that apply): ■ Alaska Native/American Indian ☐ Asian ☐ Black/ African American ☐ Native Hawaiian/ Pacific Islander ☐ White ☐ Other: _____

Past Medical History:

Medical Diagnoses (check all that apply): ☐ Asthma ☐ Chronic Pain ☐ COPD ☐ CAD ☐ Diabetes ■ HIV ☐ HTN ☐ Renal Insufficiency ☐ Solid Organ Transplant ☐ Obstructive Sleep Apnea ■ Hyperlipidemia ☐ Autoimmune Dx (Type):_____ ☐ Cancer (Type): _____
■ Other (specify): FAS, Pre-Diabetes, obesity with BMI 33
Comments (*dx date, relevant details, etc.*):

Liver Related History (check all that apply): ☐ Autoimmune Hepatitis/ Primary Biliary Cholangitis/Overlap ☐ Alcohol Related Liver Disease ☐ Hepatitis B ☐ Hepatitis C ☐ Hepatocellular Carcinoma (HCC) ☐ Non-Alcoholic Fatty Liver (NAFLD) ☐ Non-Alcoholic Steatohepatitis (NASH) ☐ Cirrhosis*
☐ Other (specify): _____
Comments (dx date, relevant details, etc.):

* Indicate any markers for decompensation: ☐ Ascites ☐ Encephalopathy ☐ Variceal Bleed

Behavioral/Social History:

Psychiatric Diagnoses (select all that apply): ☐ Anxiety ☐ Depression ☐ Mania/Hypomania (Bipolar) ☐ Schizophrenia ☐ Trauma/ PTSD ■ Other (specify): FAS
Comments (dx date, relevant details, etc.): Has a legal guardian, lives in group home. Well controlled.

Diagnostic Scores: *PHQ-9 Score, Date:* *GAD-7 Score, Date:*

ECHO Question #1: Please give objective information related to strengths and challenges, emotional or other stressors, trauma history, mental and behavioral health concerns.	
ECHO Question #2: Do you have any specific behavioral health related questions about this case? (psychotherapy, psychological assessment, substance use treatment, behavior modification, medication adherence, connecting individual to behavioral health services, encouraging client to attend appointments, enhancing rapport or patient -provider relationship, etc.)	
Substance Use Details	Does the patient currently drink alcohol? ☐ Yes ■ No If no, has the patient ever struggled with alcohol use? ☐ Yes ☐ No Date of last drink:_____ Audit-C Score*: _____ *Please use Audit-C form appended to generate score.
	Other drug use (check all that apply): ☐ Marijuana ☐ Opioids ☐ Benzodiazepines ☐ Stimulants ☐ Tobacco (type):_____ ☐ Other (specify): _____ *Comments (relevant details about usage, events, etc.):*

Current Medications/ Vaccine Status:

Medication	Dosage/Frequency/Comments	Medication (cont.)	Dosage/Frequency/Comments
Bictarvy	1qd	risperidone	1mg 1qAM, 2qPM
cholecalciferol	5000ID 1qd	prazosin	7mg TID
fluoxetine	40mg BID	rosuvastatin	20mg 1qhs
Mirtazapine	15mg 1qhs	clonidine	0.1mg BID

Vaccine History (indicate vaccinated, unvaccinated, unknown or immune for each, along with # of shots)**:**
Hepatitis A: immune (fully vaccinated) Hepatitis B**:** HsAg neg, cAb neg, sAb neg (full

Patient Objectives/Labs:

BMI: Date: 11/30/21 Weight: 78.4 (☐ lbs ■ kgs) Height: 154 (☐ in ■ cm)

LFTs: *If available, provide historical LFTs separated by at least 1 month.*	Lab	Result	Date	Result	Date	Result	Date
	ALT	54	8/2/21	77	2/9/21	109	9/7/21
	AST	51		124		132	
	Alk Phos	151		179		138	
	Alb	4.3		4.3		4.4	
	T Bili	0.6		0.7		0.8	

Lab	Result	Date
WBC	7.67	11/30/21
ANC		
HGB	14.4	
HCT	42.3	
Platelets	181	
PT		
INR		
Creatinine	1.16	8/2/21
eGFR	76	8/2/21
Na (if cirrhosis or alcohol hepatitis)	139	8/2/21
K+ (if cirrhosis or alcohol hepatitis)	4.3	8/2/21
HgbA1C	6.1	11/30/21
Triglycerides		
Cholesterol		
HDL		
LDL		
TSH		

If patient has elevated LFTs of unknown etiology, please obtain these additional labs.

Lab	Result	Date
ANA	neg	11/30/21
Actin	15	
IgG (total)	2131	
AMA (if alk phos elevated)	<20	
IgM (if alk phos elevated)	56	
Iron Total	103	11/30/21
TIBC	419	
Iron Saturation %	25	
Ferritin	159	
HCV Ab Screen	neg	11/30/21
HIV Ab Screen	Pos	1996

Serology	Result	Date
HAV-Total (HepA Ab Total)	neg	2007
HBcAb (Hep B core antibody)	neg	
HBsAb (Hep B surface antibody)	neg	
HBV DNA (in/mL)		
HCV RNA (in/mL)		

Calculations*	Value
APRI (liver fibrosis)	
FIB-4 (liver fibrosis)	
MELD (cirrhosis)	
CTP (cirrhosis)	
NAFLD Fibrosis Score (fatty liver)	
Hepatic Steatosis Index (fatty liver)	

* To calculate APRI, FIB-4, MELD, and CTP, visit: https://www.hepatitisc.uw.edu/page/clinical-calculators/apri
To calculate NAFLD Fibrosis Score, visit: https://www.mdcalc.com/
To calculate Hepatic Steatosis Index, visit: MDApp.co/hepatology/
For help with laboratory data/ calculations, contact Cynthia Decker (cadecker@anthc.org, 907-729-4636, TigerText) or Wileina Rhodes (wsrhodes@anthc.org, 907-729-1569, TigerText).

Imaging:

Include any imaging or transient elastrography results (eg. ultrasound, FibroScan, CT, MRI/MRE, EGD).		
Imaging Technique	**Date**	**Results/ comments**
MRE	11/30/21	8.1kPa (>5 stage 4 cirrhosis)
US	5/28/19	Liver measuring 18.2cm with diffuse echogenicityand focal sparing along gallbladder fossa. PV patent with hepatopetal flow.
Fibroscans		10/26/2018: 6.1kPa/10%, CAP score 357/11%. 4/15/21: 47kPa/11%, CAP score 231/31%

Primary Question(s):

In the space below, please describe the primary question/concern you would like the ECHO team to address.

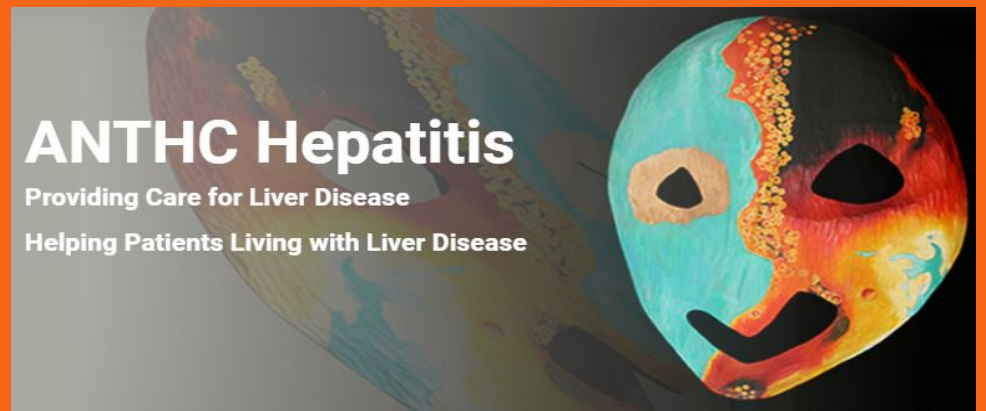
44yo male with well controlled HIV , virally suppressed since at least 2008.

LFTs demonstrating elevation at least back to 2007, peaking 2013, 2020 and showing some recent improvement has been focusing on decreasing soda/improving diet over past year. Given inconsistent fibroscans a MRE was ordered demonstrating cirrhosis. WHat should the next step in work-up be?

Is there a risk HIV meds contributed to fibrosis. Historical meds: stavudine, didanosine, ritonavir, indinavir, lamivudine, zidovudine, saquinavir.

ADDITIONAL LEARNING OPPORTUNITIES

- AK ID ECHO: HCV, HIV, PrEP, STIs
  - The 2nd Tuesday of every month from 12:00-1:00PM Alaska Standard Time
    - 1CE/CME offered per session
  - anthc.org/project-echo/hcv-hiv-prep-stis-echo
- LiverConnect Webinar Program
  - Second Tuesday of every month 8:00-9:00AM Alaska Standard Time
    - Full Hour didactic topics on Liver Disease and related topics 1CE/CME offered
  - anthc.org/what-we-do/clinical-and-research-services/hep/liverconnect/



AK LIVER DISEASE ECHO -TEAM CONTACTS

- Lisa Townshend-Bulson, MSN, FNP-C, Program Manager, ltownshend@anthc.org
- Danielle Varney, Program Coordinator, dvarney@anthc.org
- Wileina Rhodes, RN Nurse CE Coordinator, wsrhodes@anthc.org
- Annette Hewitt, FNP-C Pharmacology Content Reviewer, amhewitt@anthc.org
- Cindy Decker, RN Liver Disease ECHO Nurse Case Manager, cadecker@anthc.org
- ANTHC Liver Disease and Hepatitis Program: 907-729-1560
- Northwest Portland Area Indian Health Board
  - David Stephens: Director Indian Country ECHO, dstephens@npaihb.org
  - Jessica Leston: Clinical Programs Director, jleston@npaihb.org

Thank you

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