

Non-Alcoholic Steato Hepatitis (NASH): A Case Report of Early Onset and Progression to Liver Damage

Sunita Srivastava

Summary

Non-alcoholic steato hepatitis (NASH) is a silent liver disease often resembling cirrhosis of liver but occurring among people who drink little or no alcohol. Its major feature is fat accumulation in liver with inflammation and damage. The presentation may vary from asymptomatic to cirrhosis to development of complications. Present case report explains NASH for a 53-year-old male with overt obesity admitted in Liver intensive care unit (ICU) with typical presentation of deteriorating sensorium, fever and breathlessness. The progression of the disease and the complicated unmanageable state in which the patient presented to the ICU of a super specialised centre for management is noteworthy. He rapidly progressed to fibrosis of liver over few months in spite of his young age. The estimated global prevalence and burden of non-alcoholic fatty liver disease and its advanced stage that is development of steatohepatitis has been considerably increasing adding to hepatic morbidity and mortality. It also poses risk for increased cardiovascular complications, warranting a multidisciplinary approach. The knowledge of healthcare workers including nurses is however limited, especially with specialists outside the field of hepatology and gastroenterology. This paper aims to sensitise the nurses about the disease, symptomatology and management of such a case.

Key words: Non-alcoholic steato hepatitis, Liver Damage, Obesity

The prevalence of Non-alcoholic fatty liver disease (NAFLD) and Non-alcoholic steato hepatitis (NASH) among adults in the United States is 30 percent to 40 percent and 3 percent to 12 percent respectively, and it has been increasing given the increasing prevalence of the predisposing conditions (García-Carretero et al, 2019). Its overall pooled prevalence in India is 38.6 percent among adults and 35.4 percent among children (Shalimar et al, 2022).

Fatty liver disease is defined as the ectopic accumulation of fat in the liver (hepatic steatosis) when no other causes of secondary liver fat accumulation are present. Although minor deposition of fat can occur even in the liver of healthy adults, deposition of fat in at least 5 percent of hepatocytes is considered pathologic. Non-alcoholic fatty liver disease includes both non-alcoholic fatty liver and steato hepatitis which is diagnosed when there is evidence of inflammatory activity and hepatocyte injury in a steatotic liver tissue. The later can progress to liver fibrosis, cirrhosis, and hepatocellular carcinoma with about 30 percent to 40 percent of patients developing fibrosis and is classified into two types, primary which is related to obesity and diabetes in the absence of excessive

alcohol intake, and secondary, which is toxin- or drug-induced (Kartsonaki et al, 2019; Hester et al, 2020; Kaplan et al, 2019; Milosevic et al, 2019.)

Pathophysiology

The aetiological factors include obesity, diabetes, insulin resistance, sedentary lifestyle, and intake of high calorie diet. The degree of adipose tissue inflammation is directly correlated with degree of severity (Korinkova et al, 2020). In the present case all these factors were present.

Significance of selected case: Non-alcoholic fatty liver disease is on the rise in developing countries as well. However, health care workers in general setting are not well aware of it. There has been a significant gap in the knowledge, awareness and management among physicians regarding NAFLD (Driessan et al, 2023). In yet another survey more than half of the responding primary care physicians reported being unfamiliar with the NAFLD and NASH differences. (Polanco-Briceno et al, 2016). Generally, physicians lack awareness about treatment. Nurses working in the community and hospital setting needs to be aware to follow a proactive approach in fatty liver diseases management. The discussion of this case will help increase awareness among nursing fraternity about the chronology of the disease and its management.

The author is Reader (Nursing), Institute of Liver and Biliary Sciences, Vasant Kunj, New Delhi.

Case Study

A 53 years-old male was admitted on 13 October 2023 in Liver Intensive Care Unit with the complaints of altered behaviour, fever and breathlessness for a week. About 6 months back he was diagnosed with this condition for which he was admitted in local hospital and managed accordingly (Fig 1).

Physical examination findings at the time of admission: Well-nourished and bed ridden. Semi-conscious with Glasgow Coma Scale of 6 (E2V1M3). Pupil was reactive to light. Yellow sclera, rough and dry skin. Yellowish discoloration of teeth and bleeding in gums present. Bilateral pitting oedema present in both lower limbs. Overall, the condition of the patient was not good. On eliciting history it was a known case of diabetes and hypothyroidism. These medical conditions predispose a person for NASH. Systemic examination Findings revealed that on inspection the chest was found symmetrical in shape & size. On palpation, there was no tenderness and tactile fremitus was found to be absent. Evident abdominal distension. Scrotal swelling and redness present and urinary catheter present. Bilateral upper and lower limb swelling was present.

Investigations carried out at time of admission at Liver Intensive Care Unit: Hb 9.8 gm/dl showed anaemia as expected in CLD due to chronic haemorrhage into GI tract. In the present case the blood urea: 67 and creatinine: 2.66 values were typically raised akin to NAFLD as it is strongly associated with many predictors of cardiovascular disease such as hypercholesterolemia, hypertriglyceridemia, insulin resistance, central obesity and the metabolic syndrome. Patient had cellulitis in left buttock and on ultrasonography whole abdomen the portal hypertension with splenomegaly was noticed.

Immediate and day-to-day management of the case: As the patient was in semiconscious, confused state with drowsy state, intubation with fixing of endotracheal tube at 22 cm, cuff pressure 28 mm, mouth angle right anterior was done at LICU on 13

October 2023. The patient was put on PSIMV mode of ventilation with FiO₂ (35), Rate (24) Vt (20) PEEP (10) and PS (15).

After 3 days, on 16 October 2023 the patient developed complications as anaemia, coagulopathy, acute kidney injury, hypernatremia, hypomagnesemia. For immediate correction of coagulopathy two units Cryo precipitate was administered to the patient. The patient was in septic shock and blood pressure was falling so inj. noradrenaline was started to have target mean arterial pressure of more than 80 mm Hg.

On 17 October 2023, two units of Packed Red Blood Cells and four units of Cryo precipitated Anti hemohilic Factor was transfused to the patient. On 18 October 2023, all the above problems were decompensated as a result of which ascites, anaemia, septic shock in the patient had relatively increased. Simultaneously coagulopathy, thrombocytopenia and increased bilirubin was also noticed. The decompensation stage of the present case was clearly evident with the deteriorating liver function with marked ascites, portal hypertension and variceal bleeding. Hence he was not considered suitable for liver transplant, which was explained to the family members.

Aggressive management of the case was done involving multipronged approach. All the invasive lines were changed. As per the changes in his blood values the cryoprecipitate antihemophilic factor and Packed red blood cells were also transfused. Due to the increased toxins build-up in the blood stream patient developed hepatic encephalopathy and was highly restless, and was bucking the ventilator was thus sedated with propofol @ 3ml/hr, fentanyl @ 3ml/hr and atracurium 3 ml/hr. The haemodynamic parameters were meticulously maintained with monitoring of heart rate, rhythm, systolic and diastolic and mean arterial pressure monitoring, respiratory rate, temperature and levels of oxygen saturation everyday via the cardiac monitoring of the patient. The invasive monitoring of the patient was done through the

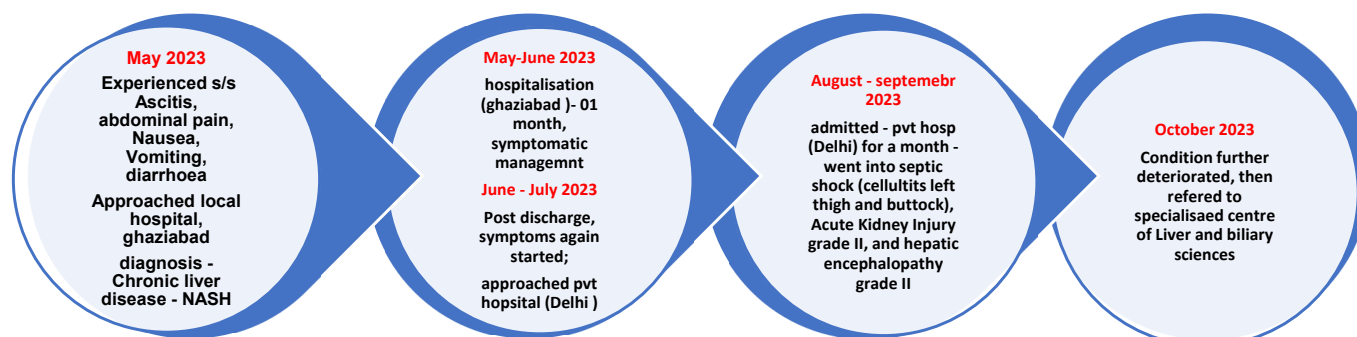


Fig 1: Timeline and progression of disease.

invasive catheters as central line in left jugular vein and arterial line with 16 gauze catheter in right radial artery. Patient had Folley’s catheter (14 Fr) for the urinary output and Ryle stube of 16 gauze inserted for the decompression. Table 1 depicts the alterations in blood values and Table 2 depicts the values of arterial blood gas analysis. The pH has been in the normal range and there had been a considerable increase in PaCO₂ and HCO₃ suggesting mixed acid base disturbances (metabolic alkalosis with respiratory acidosis).

Nursing Management

Nursing management for the patient with NASH is primarily focused on symptomatic management and preventing complications. The patient progressed

to decompensated liver damage where many complications set in.

Management of Ascites/Oedema: Assess using 1-3 score (Ascites: 1, only identifiable with ultrasound; 2, moderate; 3, large or tense. Oedema: 1, mild; 2, moderate; 3, large) The case had grade 3 ascites and oedema. He had bilateral level 3 oedema in both lower limbs. He was on low-sodium diet (< 100 mmol/day). The fluid intake output was meticulously maintained. Monitoring of serum creatinine and electrolytes was done daily. Body weight and urine volume were measured daily. The lower limbs were wrapped with bandages to reduce oedema. Use of saline solutions were avoided. Administered 25% albumin during or following large-volume paracentesis to reduce the

Table 1 : Laboratory parameters of the patient along with corresponding reference rages

S. No.	Investigations	16/10/24	17/10/24	18/10/24	Normal	Remarks
1	Hb/PCV	8.7/26	6.8/19.7	6.7/19.8	13-17 gm/dl 38.3% -48.6%	Low, Anaemia
2	TLC /DLC	26.3	14.27	25.64	5000-10000 cmm	Normal
3	Neutrophils	93.5	88.8	90.5	40-60 (%)	High
4	PT/INR	39.8/3.61	60.4/5.64	73.1/6.91	10 to 13.5/ 0.8 to 1.2	High
5	Sr. Bilirubin (T/D/I)	7.63/4.4/ 3.23	11.07/5.8/ 5.27	15.69/7.7/ 7.9	0.3-1.2 mg/dl	High
6	AST	96	87	131	5-40 IU/L	High
7	ALT	33	30	37	10-45 IU/L	Normal
8	Sr. Alk. Phosphatase	72	51	46	32-92 IU/L	High
9	GGTP	19	32	40	5 to 40 U/L	High
10	GGTP	19	32	40	5 to 40 U/L	High
11	T. Protein	4.9	4.93	5.02	6-8 gm/dL	Normal
12	Albumin	2.64	3.03	3.25	3.5-5.2 gm/dL	Normal
13	Globulin	2.21	1.88	1.77	6.4 and 8.3 g/dL.	Low
14	S. Ammonia	140.2	223.9	231.8	9.5 and 49 micrograms per deciliter	High
15	BUN	34.5	41.8	45.6	7 and 22 mg/dL	High
16	Blood urea	73.9	89.4	97.5	15-40 mg/dl	High
17	Sr. Creatinine	2.8	1.48	1.78	0.2-1.0 mg/dl	Raised
18	Sr. Sodium	113.8	151.6	155.4	136-145 mmol/L	Raised
19	Sr. Potassium	4.76	3.58	3.76	3.5-5 mmol/L	Normal
20	Chloride	85.5	115	117.6	96 to 106 milliequivalents per liter	Raised

Table 2: Blood gas analysis

Date	Time	PH	PaO ₂	PaCO ₂	HCO ₃	Lactate	SBE	SO ₂	Hb	Na	K
16/10	8:30	7.42	127	52.8*	34.5*	3.5	9.8	99.0	7.9	160	3.7
	12:30	7.44	126	48.6*	33*	6.2	8.7	99.4	7	157	4.1
17/10	8:30	7.40	107	48/8*	29.9*	6.9	5.3	98.9	6.6	154	4.0
	12:30	7.38	84.7	48.2*	28.4*	7.6	3.8	98.4	6.2	156	4.0

* increased suggesting mixed and complex respiratory acidosis with metabolic alkalosis.

Table 3: Medications prescribed with their action and nursing responsibilities

Name	Dose	Route	Freq	Action/Nursing responsibility
Inj Polymyxin B	5 Lac	IV	BD	Broad spectrum Antibiotic, look for side effects as eye irritation, burning sensation
Inj Aztreonam	1 gm	IV	TDS	Broad spectrum Antibiotic
Inj Metrogyl	500 mg	IV	TDS	Broad spectrum antibiotic
Inj Alores	1.5 gm	IV	BD	Broad spectrum antibiotic, do not stop abruptly. Look for the side effects
Inj Tigecycline	50 mg	IV	OD	Broad spectrum Antibiotic
Inj Amphotericin B	250 mg	IV	OD	Antifungal, to control & prevent infections
Inj Glutathione	1.2 gm	IV	OD	Peptides- it is antioxidant. Prevents liver damage by releasing free radicals. Look for severe side effects as injection site pain, extravasation, venous thrombosis
Inj optineuron	1 amp	IV	OD	Vitamin B complex injection. Significant for improving the brain functions. Look for the side effects as diarrhoea, tiredness, headache, Nausea
Inj Hydrocortisone	50 mg	IV	QID	Corticosteroid
Tab Thyronorm	75 mcg	IV	OD	Thyroid agent - as the patient was a known case of hypothyroidism, it was administered
Inj M _g SO ₄	2 gm	IV	TDS	Magnesium supplement and anticonvulsant. Look for the side effects as dizziness, drowsiness, low blood pressure etc
Inj. Trenexa	1 gm	IV	TDS	Anti-fibrinolytic, treats bleeding by preventing the rapid breakdown of clots. Closely monitor Bleeding Time, Clotting Time values.
Inj Vit K	1 amp	IV	OD	Supplement of vitamin K, blood clotting
Inj. Calcium Gluconate	1 ampule	IV	TDS	Gluconate salt of calcium
Neb Calistin	3 ml	Inhale	TDS	Antibiotic
Inj Noradrenaline	4 ml/hr	IV	On flow	Vasopressor – used to treat hypotension. Look for the side effects as systemic hypertension, headache, insomnia etc.
Inj Terlipressin	2.1 ml/hr	IV	On flow	analogue of vasopressin and is used in the treatment of bleeding esophageal varices by working on the walls of blood vessels. Observe for the side effects as slow heart rate, breathlessness, respiratory failure, fluid overload, pleural effusion etc.
Inj Propofol	4 ml/hr	IV	On flow	sedative-hypnotic agent – monitor BP regularly as causes hypotension.
Inj Fentanyl	4 ml/hr	IV	On flow	Analgesics – used to manage severe pain. It works by blocking transmission of pain signals to the brain. Observe for respiratory depression as the side effect.
Inj Atracurium	4 ml/hr	IV	On flow	skeletal muscle relaxant – as the patient was on ventilator it was administered to relax the muscles.
Inj KCL	5 ml/hr	IV	On flow	Potassium supplement – essential electrolyte to carry out various life functions. Significant in maintaining water, PH balance in the body.
Inj Hepamerz	4.1 ml/hr	IV	On flow	Amino acids – it protects the liver from damage from free radicals. Observe for the local side effects as pain, swelling, redness etc.

risk of precipitating hepatorenal syndrome. Care was taken to explain dietary, fluid and skin care to family members also.

Management of Gastrointestinal Bleeding: It is a common complication of decompensated liver damage. Several possible lesions may cause GI bleeding in cirrhosis, most of them related to portal hypertension. It can be classified as upper or lower, according to whether the lesion responsible for bleeding is located above or below the angle of Treitz, respectively. The main causes of upper GI bleeding include gastric or esophageal varices

and portal-hypertensive gastropathy, whereas those of lower GI bleeding include rectal varices and portal-hypertensive enteropathy or colopathy. Upper GI bleeding manifests as melena and/or hematemesis, whereas lower GI bleeding manifests as hematochezia. Melena suggesting upper GI bleed was present in this case. Hematoma over both hands was evident. He underwent repeated packed red blood cells transfusions as haemoglobin < 7 g/dL. Due to ascites, dextrose solutions were used. The haemoglobin, liver function test and clotting factors were regularly checked. As he had several episodes of bleeding which could initiate

hepatic encephalopathy, rectal enema was given. He was given terlipressin which helps in controlling bleeding in 80% of the patients with variceal bleeding (Table 3). He was put on cardiac monitoring with regular record of Intake and output. Mental status was frequently assessed. The skin pallor and temperature were recorded every two hours. Monitored the characteristics of emesis or stool for presence of blood (black vs. bright red).

Management of Hepatic Encephalopathy

Hepatic encephalopathy (HE) is characterised by neuropsychiatric symptoms including disorientation, inappropriate behaviour, sleep disturbances, abnormalities in speech, and alterations in consciousness that may progress to coma. The present case had these symptoms at admission which later slipped into highly aggressive and then unconsciousness. HE occurs because substances present in the gut with the capacity to interfere with neuronal function, such as ammonia, bypass the liver and reach the brain due to the presence of shunts between the portal territory and systemic circulation, and hepatic dysfunction (Patidar & Bajaj, 2015; Vilstrup et al, 2014) for the grading of HE. West-Haven criteria was used for the case which was found to be grade 4 (4- Coma: Precipitants of hepatic encephalopathy including infection, constipation, electrolyte disturbance, particularly hypokalaemia, sedative drugs and GI bleeding). The main treatment of acute HE involves correcting the precipitant and encouraging elimination of toxins using lactulose (orally or via nasogastric tube, titrated to bowel frequency, aiming for 2–3 soft stools per day) and enemas. The present case had evidence of infection, GI bleeding and deteriorating liver function. All these precipitants were managed with broad spectrum antibiotics, lactulose via NG tube, correction of fluid and electrolytes and monitoring of the changes in the blood values. Regular monitoring for serum creatinine and electrolytes was done. As the present case became agitated in between and bucking the ventilator also, sedation was used to calm the patient.

Management of Airway Clearance

The present case was intubated; abnormal breath sounds and excessive secretions were there. So, the airway was assessed for clearance. Secretolytics were administered to loosen the thickened secretions, observed for the colour, odour, quantity, and consistency of sputum. Monitored the oxygen saturation prior to and after suctioning using pulse oximetry. Assessed arterial blood gases (ABGs) regularly and for any excessive coughing, increased dyspnoea, high-pressure alarm on the ventilator, and visible secretions in the endotracheal or tracheostomy tube. Turned the patient every two

hours, instituted airway suctioning for the presence of adventitious breath sounds and/or increased ventilatory pressure. Administered intravenous therapy and aerosol bronchodilators. Physiotherapy was done every day.

Management of Infection

As patients with cirrhosis are effectively immunosuppressed, infections are the most common reasons for hepatic decompensation. Most common infections are SBP, urinary tract infections, pneumonia and cellulitis. Gram-negative bacteria (particularly *Escherichia coli*) and gram-positive cocci are the most common pathogens (Bunchorntavakul et al, 2016). He was assessed for the signs of pulmonary infection including increased temperature, purulent secretions, elevated white blood cell count, positive bacterial cultures, and evidence of pulmonary infection on chest X-ray studies. Encouraged the family members, and other staff members to engage in proper hand hygiene. Provided oral hygiene twice daily, including the use of a dental oral antibiotic rinse. Limited the visitors and avoided contact with persons with respiratory infections. Kept the head of the bed elevated to 30 to 45 degrees. Used sterile suctioning procedures and reduced the number of times the ventilator tubes are open. As the patient was on ventilator all precautions to prevent ventilator-associated pneumonia were taken. He was administered broad spectrum antibiotics (as in Table 3) to manage the infections.

Management of Diet: Diet plan included a low-calorie, low-fat diet which can be given through Ryle's tube every 2 hours and ensured that nasogastric nutritional supplements to provide a total energy intake of about 35–40 kcal/kg daily. As refeeding syndrome is expected phosphate, potassium and magnesium were monitored daily, and electrolytes were replaced intravenously. He was also administered optineuron, a vitamin B complex supplement.

Discussion

The present case, a typical presentation of NASH, is similar to yet another case (Yuichi Honma et al, 2018) where it was diagnosed in a 53-year-old male with overt obesity (weight 100 kg, height 5 ft 6 inches, BMI 35.6). This aligns with the preposition that younger patients with obesity can also progress to permanent liver damage.

Chronic liver disease (CLD) is a progressive deterioration of liver functions for more than six months, which includes synthesis of clotting factors, other proteins, detoxification of harmful products of metabolism, and excretion of bile. CLD is a continuous process of inflammation, destruction, and regeneration of liver parenchyma,

which leads to fibrosis and cirrhosis. NAFLD is the most prevalent liver disease world-wide, affecting 20–25 percent of the adult population. In 25 percent of patients, NAFLD progresses to NASH, which increases the risk for the development of cirrhosis, liver failure and hepatocellular carcinoma. In the present case the deteriorated condition of the patient over a short span of 6 months was due to extensive liver damage. NASH patients with significant fibrosis have increased risk of developing cirrhosis and liver failure (Cheng Pang et al, 2020). In the present case also the patient progressed to severe liver damage. NASH is typically characterised by liver steatosis inflammation, and fibrosis driven by metabolic disruptions such as obesity, diabetes, and dyslipidemia. This patient presented with overt obesity and diabetes.

NAFLD tends to be more prevalent in middle-aged to elderly patients as older patients exhibit features of metabolic syndrome (Frith et al, 2009; Williams et al, 2011). The present case also is a middle-aged man with diabetes who was poorly managed at various local hospitals.

Liver enzymes, specifically alanine aminotransferase (ALT) and aspartate aminotransferase (AST), are mildly elevated with an ALT predominance and usually not exceeding 250 IU/L. The mean ALT and AST levels from a large cohort of biopsy-proven NASH patients were recently found to be 69 and 51 IU/L, respectively (Harrison et al, 2003). However in the present case the values of PT/INR/AST/Sr. Alkaline phosphatase /CGTP and Sr. Ammonia were found to be high. The other parameters as reported in the literature could not be correlated in the present case as it is not a fresh case and was already diagnosed with NASH six months back.

Conclusion

NAFLD/NASH may be reversible in the initial stages if diagnosed early and proper treatment and care is given at a well-equipped hospital and health care staff. As the present case was managed poorly at various local and private hospitals his condition kept on deteriorating and progressed to complications. Parameters and the prognosis of the case remains guarded. Meticulous and close nursing care was given to the patient following liver Care bundle Care.

References

- Kartsonaki C, Turnbull I, Guo Y, Chen Y, Clarke R, Bian Z, et al. Adiposity in relation to risks of fatty liver, cirrhosis and liver cancer: A prospective study of 0.5 million Chinese adults. *Scientific Reports* 2019; 9(1): 785. <https://doi.org/10.1038/s41598-018-37106-9>
- Hester D, Golabi P, Paik J, Younossi I, Mishra A, Younossi ZM. Among medicare patients with hepatocellular carcinoma, non-alcoholic fatty liver disease is the most common etiology and cause of mortality. *Journal of Clinical Gastroenterology*, 2020; 54(5): 459-67. <https://doi.org/10.1097/MCG.0000000000001378>
- Kaplan DE, Serper MA, Mehta R, Fox R, John B, Aytaman A, VOCAL Study Group. Effects of hypercholesterolemia and statin exposure on survival in a large national cohort of patients with cirrhosis. *Gastroenterology* 2019; 156(6): 1693-1706.e12. <https://doi.org/10.1053/j.gastro.2018.12.021>
- Milosevic I, Vujovic A, Barac A, Djelic M, Korac M, Radovanovic Spurnic A, et al. Gut-liver axis, gut microbiota, and its modulation in the management of liver diseases: A review of the literature. *International Journal of Molecular Sciences* 2019; 20(2): 395. <https://doi.org/10.3390/ijms20020396>
- García-Carretero R, Barquero-Pérez O, Mora-Jiménez I, Soguero-Ruiz C, Goya-Esteban R, Rodríguez-Castro C et al. Prevalence and clinical features of non-alcoholic steatohepatitis in a hypertensive population. *Hipertensión y Riesgo Vascular* 2019; 36(3): 130-36
- Shalimar, Elhence A, Bansal B, Gupta H, Anand A, Singh TP, Goel A. Prevalence of non-alcoholic fatty liver disease in India: A systematic review and meta-analysis. *Journal of Clinical and Experimental Hepatology* 2022; 12(3): 818-29. <https://doi.org/10.1016/j.jceh.2021.11.010>
- Sharma B, John S. Nonalcoholic steatohepatitis (NASH). In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing 2023. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK470243/>
- Honma Y, Sumida K, Ogino N, Kusanaga M, Minami S, Kumei S, et al. A young adult patient with nonalcoholic steatohepatitis developed severe gastroesophageal varices associated with severe obesity and diabetes mellitus. *Case Reports in Gastroenterology* 2018; 12(2): 487-96. <https://doi.org/10.1159/000492423>
- Frith J, Day CP, Henderson E, Burt AD, Newton JL. Non-alcoholic fatty liver disease in older people. *Gerontology* 2009; 55, 607–613. <https://doi.org/10.1159/000235677>
- Driessen S, de Jong VD, van Son KC, Klompenhouwer T, Colardelle Y, Alings M, et al. A global survey of health care workers' awareness of non-alcoholic fatty liver disease: The Aware NASH survey. *United European Gastroenterol J* 2023; 11(7): 654-62. <https://doi.org/10.1002/ueg2.12445>
- Korínkova L, Prazienkova V, Cerna L, Karnosova A, Zelezna B, Kunes J, et al. Pathophysiology of NAFLD and NASH in experimental models: The role of food intake regulating peptides. *Frontiers in Endocrinology* 2020; 11, 597583. <https://doi.org/10.3389/fendo.2020.597583>
- Patidar KR, Bajaj JS. Covert and overt hepatic encephalopathy: Diagnosis and management. *Clin Gastroenterol Hepatol* 2015; 13: 2048-61. [PMC free article] [PubMed]
- Vilstrup H, Amodio P, Bajaj J, Cordova J, Ferenci P, Mullen KD, et al. Hepatic encephalopathy in chronic liver disease: 2014 practice guideline by American Association for the Study of Liver Diseases and the European Association for the Study of the Liver. *Hepatology* 2014; 60: 715-35 [PubMed]
- Bunchorntavakul C, Chamroonkul N, Chavalitdhamrong D. Bacterial infections in cirrhosis: A critical review and practical guidance. *World J Hepatol* 2016; 8: 307–21 [PMC free article] [PubMed] [Google Scholar]