

Vorasidenib-Induced Acute Liver Injury

Emily A. Lau

Hoag Digestive Health Institute, Hoag Memorial Hospital Presbyterian

David R. Braxton

Hoag Memorial Hospital Presbyterian

Didier Autran

University Hospital Timone

Fengming Chen

Duke University

Aurelie M. Haffner

Aix Marseille University, CHU Timone

Jethro Hu

Cedars-Sinai Medical Center

Katherine B. Peters

Duke University Medical Center

Tse-Ling Fong

tse.ling.fong@hoag.org

Hoag Digestive Health Institute, Hoag Memorial Hospital Presbyterian

Case Report

Keywords: vorasidenib, drug-induced liver injury, autoimmune hepatitis, hepatocellular injury, corticosteroids

Posted Date: June 30th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-9391169/v1>

License:   This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Additional Declarations: Competing interest reported. Katherine Peters is a consultant for Servier and was an investigator in the INDIGO trial. Jethro Hu is a consultant and speaker for Servier.

Version of Record: A version of this preprint was published at Digestive Diseases and Sciences on September 11th, 2026. See the published version at <https://doi.org/10.1007/s10620-026-10244-w>.

Abstract

Background and Aims:

Vorasidenib is a dual inhibitor of isocitrate dehydrogenase-1 and isocitrate dehydrogenase-2 that is used to treat patients with Grade 2 astrocytoma or oligodendroglioma with either mutations after surgery. Vorasidenib has yet to be linked to cases of acute liver injury. We describe 3 cases of severe acute liver injury with features of autoimmune hepatitis associated with vorasidenib. Additionally, we summarize 50 cases of liver injury associated with vorasidenib reported to the United States Food and Drug Administration (FDA).

Methods:

Case series of 3 patients who developed severe acute liver injury while treated with vorasidenib. A search of the FDA Adverse Event Reporting System database revealed 50 cases of suspected drug-induced liver injury from vorasidenib with usable information for analysis.

Results:

Onsets of elevated aminotransferase activities were 13, 31, and 2 weeks respectively after initiation of vorasidenib. Pattern of liver injury was hepatocellular with mild to moderate jaundice without significant coagulopathy. Despite discontinuation of vorasidenib, aminotransferase (ALT) activities continued to increase, which along with histology findings, prompted initiation of corticosteroids for possible drug induced autoimmune-like hepatitis. Liver tests became normal and steroids were tapered over 23, 3, and 7 months respectively.

Among FDA cases, mean latency to greater than 3 times upper limit of normal ALT was 66 days with hepatocellular pattern of liver injury. Nine cases were hospitalized with no reported liver transplants or liver-related deaths. Seven were treated with steroids, and 8 out of 23 cases with bilirubin value met criteria for Hy's Law.

Conclusion:

Vorasidenib induced liver injury is entirely hepatocellular with a subset that presents with autoimmune features which responds to a finite course of corticosteroids.

IMPACT AND IMPLICATIONS

Vorasidenib is a cause of liver injury presenting with hepatocellular pattern of liver test abnormalities. In some instances there are autoimmune features that require a finite course of corticosteroids. Monitoring of liver tests of patients treated with vorasidenib is recommended.

INTRODUCTION

Vorasidenib (Vorango[®]) is a dual inhibitor of isocitrate dehydrogenase-1 (IDH1) and isocitrate dehydrogenase-2 (IDH2) that lowers intratumoral 2-hydroxyglutarate and promotes differentiation in IDH-mutant glioma. This drug was approved by the U.S. Food and Drug Administration in 2024 to treat patients with Grade 2 astrocytoma or oligodendroglioma with a IDH1 or IDH2 mutation after surgery. Common side effects include fatigue, headache, musculoskeletal pain, diarrhea, nausea, and seizures. Vorasidenib is primarily metabolized by the liver through CYP1A2 (with minor contributions from other CYP isoforms and non-CYP pathways) and is known to cause generally mild, self-limited elevations in aminotransferase activities (1).

To date, there are no published cases of acute liver injury due to vorasidenib. We report 3 cases of vorasidenib-induced liver injury with autoimmune features. Two of the 3 cases were amongst the 331 participants (168 patients receiving vorasidenib 40 mg daily and 163 patients receiving placebo) in the phase 3, randomized, double-blinded, INDIGO clinical trial (NCT04164901). When these 2 patients developed liver injury, treatment was unblinded, and the patients were found to be taking vorasidenib (1, 2). The third case was a patient treated with vorasidenib after approval by the Food and Drug Administration (FDA). Liver biopsy was performed on all 3 patients at the peak of illness.

Since FDA approval of vorasidenib from August 6th, 2024 to November 14th, 2025, there were 317 adverse events for vorasidenib reported to the FDA Adverse Event Reporting System (FAERS). When the inquiry using the Freedom of Information Act was limited to liver-related adverse events, a total of 54 cases were identified in the database, of which 50 unique cases with usable information (excluding duplicates) were retrieved and summarized, revealing a potential for severe, hepatocellular-pattern injury.

PATIENTS

Case 1

The patient is a 46 year old female with history of right frontal grade 2 astrocytoma, with isocitrate dehydrogenase 1 R132H mutation, that was sub-totally resected and monitored. She also has heterozygous sickle cell disease. She did not take concomitant medications other than occasional acetaminophen for infrequent headache. Four years later, the patient began treatment with vorasidenib 40 mg by mouth daily as part of the INDIGO clinical trial. Baseline liver tests were normal. Ten weeks after starting the drug, she developed a mild increase in aminotransferase activities (grade 1) which progressively increased until week 16 (grade 3). Meanwhile the patient was treated for 5 days with prednisolone 60 – 40 mg for flu-like illness and liver tests transiently improved during week 14. Vorasidenib was discontinued but her tests continued to worsen in the ensuing 5 weeks. She experienced grade 2 fatigue and grade 2 abdominal pain, grade 2 nausea, grade 1 anorexia, vomiting and constipation but she was afebrile. Physical exam was normal, including vital signs and absence of skin rash. Patient was hospitalized and liver tests revealed total bilirubin 2.34 mg/dL (40.01 µmol/L), alkaline phosphatase 106 U/L, aspartate aminotransferase (AST) 1476 U/L, alanine aminotransferase (ALT) 1596 U/L, IgG immunoglobulin 1870 mg/dL (18.7 g/L) and INR 1.92. Acute viral serologies for hepatitis A, B, C

and E were negative. Nucleic acid amplification tests (NAATs) for hepatitis B virus (HBV), hepatitis C virus (HCV), hepatitis E virus (HEV), cytomegalovirus (CMV), Epstein-Barr virus (EBV), herpes simplex virus (HSV), parvovirus and adenovirus were negative. Serologies were also negative for syphilis, trachomatis and toxoplasmosis. Anti-nuclear antibody (ANA) was positive 1:320, but anti-smooth muscle antibody (ASMA) negative. She started methylprednisolone 60 mg intravenously (IV) daily then switched to prednisone 60 mg, with improvement. When prednisone dose was reduced to 40mg daily during week 34, she relapsed. Prednisone 60 mg was reinstituted with the addition of azathioprine and ursodeoxycholic acid. Her liver tests improved and normalized after 40 weeks, during a taper over 14 months (Fig. 1a).

Case 2

The patient is a 41 year-old female with history of oligodendroglioma, IDH1 mutant, 1p19q co-deleted WHO grade 2, with isocitrate dehydrogenase 1 R132H mutation that was sub-totally resected. Over time, there was slow progression of non-enhancing disease on magnetic resonance imaging, and the patient was enrolled into the INDIGO clinical trial and began vorasidenib 40 mg by mouth daily, 33 months after surgery. Baseline liver tests were normal. Concomitant medications included levetiracetam 1,000 mg orally twice a day and venlafaxine 70 mg orally daily. Nine weeks after initiating vorasidenib, she developed an increase in aminotransferase activities (grade 1) which further increased by week 10 (grade 3 AST, grade 4 ALT). Patient denied use of acetaminophen, new medications, or supplements. Six days before the detection of grade 1 increase in aminotransferase activity, she had had 4 alcohol-containing drinks. Despite discontinuing vorasidenib 9 months after initiation, she developed worsening fatigue, anorexia, nausea and right sided abdominal pain and was hospitalized. Physical exam was normal, including vital signs and absence of rash. Liver tests revealed total bilirubin 2.0 mg/dL (34.2 µmol/L), alkaline phosphatase 141 U/L, AST 1302 U/L, ALT 2209 U/L, gamma-glutamyl transferase 63 U/L. Acute hepatitis A, B and C serologies, ANA, ASMA were nonreactive. NAATs for HSV and CMV were negative. Abdominal ultrasound was unremarkable. She started methylprednisolone 1 mg/kg IV daily for 3 days along with vitamin K orally 10 mg once a day for 3 days. Patient was discharged after completion of IV methylprednisolone and placed on a prednisone taper starting at 60 mg orally once a day, but 7 days later, she required readmission for increased somnolence, although INR was only 1.3. She was put back on IV methylprednisolone 60 mg IV and received rifaximin 60 mg orally bid and lactulose 15 mL orally bid. Serum ammonia level peaked at 51 µg/dL (30 µmol/L) on admission and then recovered. After completion of three doses of IV methylprednisolone, she was discharged on prednisone taper starting at 60 mg, rifaximin 550 mg po bid for 21 day course, lactulose 30 mL po bid for 21 day course. Prednisone was slowly tapered and discontinued finally on week 52 for a total time on steroids of three months.

Case 3

The patient is a 46-year-old male with history of right frontal grade 2 diffuse astrocytoma, with isocitrate dehydrogenase 1 R132H mutation that was resected 13 years ago and subsequently monitored. He had depression and trouble with mental focus and was taking bupropion 300 mg and methylphenidate 10 mg

daily. Following FDA approval, he began therapy with vorasidenib 40 mg by mouth daily. Baseline liver tests were normal. Two weeks after starting vorasidenib, he developed a mild increase in aminotransferase activities which progressively increased in the ensuing 12 weeks. Vorasidenib was discontinued but his tests continued to worsen. He experienced fatigue and mild abdominal discomfort. He had no fever, rash, nausea or vomiting. Vital signs and physical exam were normal. Liver tests revealed total bilirubin 1.6 mg/dL (27.36 μ mol/L), albumin 4.5 g/dL (45 g/L), globulin 3.4 g/dL (34 g/L), alkaline phosphatase 74U/L, aspartate aminotransferase (AST) 711 U/L, alanine aminotransferase (ALT) 1064 U/L, IgG 1231 mg/dL (12.31 g/L). Acute viral serologies for hepatitis A, B, C and E were negative. ANA was positive 1:640, ASMA negative. Abdominal ultrasound showed cholelithiasis without biliary ductal dilatation.

He began prednisone 40 mg by mouth daily with a dramatic improvement in aminotransferase activities. When prednisone dose was gradually reduced to 20 mg after 4 weeks, he relapsed. With reinstitution of prednisone 40 mg and a slower taper, liver tests steadily improved and normalized after 50 weeks (Fig. 1C).

Summary of Clinical Cases

All 3 patients presented with fatigue, 2 of whom also experienced nausea and abdominal pain. The onsets of elevated ALT ($> 3X$ upper limit of normal (ULN)) were 13, 31, and 2 weeks respectively, and peaked at a mean of 25 weeks after initiation of vorasidenib. The pattern of liver injury was hepatocellular with mild to moderate jaundice in all 3 patients, without significant coagulopathy (Table 1). Two cases fulfilled Hy's law (3, 4). Total globulin level was normal, but ANA was reactive in 2 patients. Despite discontinuation of vorasidenib, aminotransferase activities continued to increase which along with the liver biopsy findings, prompted initiation of corticosteroids for possible drug induced autoimmune-like hepatitis (5, 6). Interestingly, all 3 patients experienced mild biochemical relapse when corticosteroids were initially tapered quickly, necessitating a dose increase followed by a slower taper over 23, 3, and 7 months respectively, with eventual normalization (Figs. 1A-C). These patients have maintained normal liver tests after discontinuation of immunosuppression.

Table 1
Summary of Demographic and Laboratory Results of Patients with Vorasidenib-Induced Liver Injury.

Patient characteristics	Case 1 Marseille	Case 2 Duke	Case 3 Hoag
Age (years)	46	41	46
Gender	Female	Female	Male
Time to onset of ALT > 3X upper limit of normal (weeks)	13	31	2
Peak bilirubin (μmol/L)	56.1	114.6	37.6
Peak alkaline phosphatase (U/L)	113	243	86
Peak ALT (U/L)	1596	2209	1438
Peak AST (U/L)	1478	1302	711
Peak INR	1.92	2.1	1.3
R factor	67.8	43.6	80.3
Presentation to peak bilirubin (weeks)	20	38	18
Presentation to peak alkaline phosphatase (weeks)	20	42	18
Presentation to peak ALT (weeks)	18	37	18
Presentation to peak AST (weeks)	18	37	15
Presentation to peak INR (weeks)	19	37	15
Time from peak ALT to resolution (weeks)	40	15	32

Summary of Liver Histology

Representative histological findings of the three patients are shown in Fig. 2. There is consistent centrilobular necrosis with two of the cases showing severe bridging necrosis (Fig. 2B) as well as a subdued but predominantly lymphocytic infiltrate and a minor plasma cell component. One example showed clear interface hepatitis containing plasma cells within the infiltrate (Fig. 2A). No cholestasis was noted in these patients. Special stains were largely unremarkable with the absence of portal fibrosis noted on Masson trichrome stain and only one case of mild perisinusoidal fibrosis present (Fig. 2H). CK7 immunostain highlighted absent to minimal biliary ductular reaction with only one case exhibiting focal mild bile duct injury (Fig. 2D, L). Taken together, the pattern of injury was a range of zonal necrosis beginning in zone 3 with increasingly severe grades of necrosis moving into portal-to-portal bridging/pan acinar necrosis that was accompanied by variably intense predominantly lymphocytic infiltrate with or without biliary reaction and injury.

Summary of FDA Cases

A search of the FAERS database from August 6, 2024, to November 14, 2025 was conducted to identify additional cases of suspected drug-induced liver injury from vorasidenib post-FDA approval and marketing. From a total of 317 adverse events reported for the drug during this period, 54 were identified as liver-related using the terms jaundice, hepatic failure, cholestasis, liver injury, drug-induced liver injury, increased liver function test, abnormal liver function test, hepatic enzyme increase, hepatitis, hepatocellular injury and hepatitis cholestatic. Of the 54 cases identified, 50 provided usable information for analysis after excluding unreadable or duplicate reports.

The completeness of the FDA data was poor with only subsets of demographic and case data available (Table 2). Females comprised 59% of the cases (N = 46). Mean latency from drug introduction to an elevated ALT (> 30 U/L) was 36 days (N = 11). Mean latency to develop an ALT > 3X ULN was 66 days (N = 23). R value was 35.8 (range 5.5–81.6)(N = 16). Nine of the 50 cases were hospitalized with no reported liver transplants or liver-related deaths. Nine cases had a liver biopsy, 7 were treated with steroids, and 8 out of 23 cases that provided total bilirubin value met criteria for Hy's Law.

Table 2
Summary of FAERS Cases of Vorasidenib-Associated
Hepatotoxicity.

Age (years)	N	Range	Mean
	39	17–79	43
ALT (U/L)	30	89-2821	834
AST (U/L)	31	46-1464	428
Total bilirubin (μmol/L)	23	5.1-256.5	57.3
Alkaline phosphatase (U/L)	18	51–250	116
INR	5	0.77–2.1	1.33
R value	16	5.5–81.6	35.8
N=number of cases with evaluable data			

Patients who were treated with steroids (N = 7) had a higher mean peak ALT compared to patients who did not receive steroids (N = 23) (1598 vs. 602 U/L, p = 0.004) (2-tailed t-test). Mean peak bilirubin levels were similar in these 2 groups (57.6 μmol/L (N = 7) vs. 60 μmol/L (N = 16), p = NS). Of the 9 cases that were hospitalized, 5 received a liver biopsy and 4 were treated with steroids. Of the 7 cases that were treated with steroids, 6 had a liver biopsy and 4 were hospitalized.

The cases were further assessed using the criteria set by the International DILI Expert Working Group, and were categorized as 16 mild, 9 moderate, 1 severe, and 0 fatal (6). The remaining 24 cases included

vague verbal descriptions of liver test abnormalities ("hepatotoxicity," "hepatic cytolysis," "altered liver enzymes," "liver enzyme elevated," etc.) or did not meet their threshold to be formally categorized.

DISCUSSION

We describe 3 patients with granular clinical data who had similar presentations with severe idiosyncratic drug-induced acute hepatocellular liver injury caused by vorasidenib which has not been previously reported. Other causes of potential liver injury were excluded. Lack of improvement following discontinuation of the drug and relapse of hepatitis when corticosteroids were rapidly tapered, were some of the clinical features of drug induced autoimmune-like hepatitis.

Using the Council for International Organizations of Medical Sciences (CIOMS) scale to determine causality, the score was 4 (probable) in all 3 cases (7). The 2 domains of CIOMS scale that these 3 cases did not fulfill were the rapid improvement in aminotransferase activities after discontinuation of vorasidenib, and not being rechallenged with the drug (7).

Histological findings show consistent with varying degrees of centrilobular zonal necrosis that correlated with clinical severity. Furthermore, the predominant lymphocytic infiltrates with or without plasma cells along with the centrilobular zonal necrosis suggests an immune mediated injury. While a typical idiosyncratic drug induced injury cannot be excluded, the lack of typical spotty necrosis and/or cholestasis favor alternative mechanisms of injury.

Elevated alanine and aspartate aminotransferase activities were reported in 38.9% (Grade ≥ 3 9.6%) and 28.7% (Grade ≥ 3 , 4.2%), respectively, of patients receiving vorasidenib in the pre-registration trial (1, 2). The FAERS database search yielded 50 cases with usable information of vorasidenib-associated hepatotoxicity within its first 15 months of marketing. These events showed a consistent hepatocellular injury pattern and a significant potential for severe DILI, shown by 9 hospitalizations and 8 cases fulfilling Hy's Law criteria. We recognize the many limitations of MedWatch reports but there is some utility in confirming a signal for vorasidenib-induced liver injury in the post-marketing setting.

CONCLUSION

This report highlights the clinical manifestations of vorasidenib-induced liver injury which is predominantly hepatocellular pattern. There is a subset of patients that presents with more severe disease and autoimmune features which respond to a finite course of corticosteroids.

DECLARATIONS

Conflicts of Interest:

Katherine Peters is a consultant for Servier and was an investigator in the INDIGO trial. Jethro Hu is a consultant and speaker for Servier. Emily A. Lau, David R. Braxton, Didier Autran, Fengming Chen, Aurelie Haffner, and Tse-Ling Fong have no disclosures.

Financial Support Statement

There is no financial support for this paper.

Competing Interests

Katherine Peters is a consultant for Servier and was an investigator in the INDIGO trial. Jethro Hu is a consultant and speaker for Servier.

Author Contribution

E.L. and T.F. wrote the main manuscript text. E.L. performed data analysis, searched the FDA database, and prepared figure 1 and tables 1-2. D.B. prepared figure 2, and D.B., F.C., and J.H. provided pathology data. D.A., K.P., and T.F. provided and wrote case report data. T.F. conceptualized and supervised the project. All authors reviewed the manuscript.

Data Availability

Clinical data can be obtained through the corresponding author, Tse-Ling Fong. FDA data can be obtained through the Freedom of Information Act on the FDA Adverse Event Reporting System.

REFERENCES

1. Mellinghoff IK, van den Bent MJ, Blumenthal DT, Touat M, Peters KB, Clarke J, Mendez J, Yust-Katz S, Welsh L, Mason WP, Ducray F, Umemura Y, Nabors B, Holdhoff M, Hottinger AF, Arakawa Y, Sepulveda JM, Wick W, Soffietti R, Perry JR, Giglio P, de la Fuente M, Maher EA, Schoenfeld S, Zhao D, Pandya SS, Steelman L, Hassan I, Wen PY, Cloughesy TF; INDIGO Trial Investigators. Vorasidenib in IDH1- or IDH2-Mutant Low-Grade Glioma. *N Engl J Med*. 2023;389(7):589–601. doi: 10.1056/NEJMoa2304194. Epub 2023 Jun 4. PMID: 37272516; PMCID: PMC11445763.
2. VORANIGO® (vorasidenib). Prescribing information. Servier Pharmaceuticals, 2024. Accessed November 28, 2025. https://www.accessdata.fda.gov/drugsatfda_docs/label/2024/218784s000lbl.pdf
3. European Association for the Study of the Liver. EASL Clinical Practice Guidelines: Drug-induced liver injury. *J Hepatol*. 2019;70(6):1222–1261. doi: 10.1016/j.jhep.2019.02.014. Epub 2019 Mar 27. PMID: 30926241.

4. Fontana RJ, Liou I, Reuben A, Suzuki A, Fiel MI, Lee W, Navarro V. AASLD practice guidance on drug, herbal, and dietary supplement-induced liver injury. *Hepatology*. 2023;77(3):1036–1065. doi: 10.1002/hep.32689. Epub 2023 Feb 17. PMID: 35899384; PMCID: PMC9936988.
5. Czaja AJ. Drug-induced autoimmune-like hepatitis. *Dig Dis Sci*. 2011;56(4):958–76. doi: 10.1007/s10620-011-1611-4. Epub 2011 Feb 16. PMID: 21327704.
6. Andrade RJ, Aithal GP, de Boer YS, Liberal R, Gerbes A, Regev A, Terziroli Beretta-Piccoli B, Schramm C, Kleiner DE, De Martin E, Kullak-Ublick GA, Stirnimann G, Devarbhavi H, Vierling JM, Manns MP, Sebode M, Londoño MC, Avigan M, Robles-Diaz M, García-Cortes M, Atallah E, Heneghan M, Chalasani N, Trivedi PJ, Hayashi PH, Taubert R, Fontana RJ, Weber S, Oo YH, Zen Y, Licata A, Lucena MI, Mieli-Vergani G, Vergani D, Björnsson ES; IAHG and EASL DHILI Consortium. Nomenclature, diagnosis and management of drug-induced autoimmune-like hepatitis (DI-ALH): An expert opinion meeting report. *J Hepatol*. 2023;79(3):853–866.
7. Hayashi PH, Lucena MI, Fontana RJ, Björnsson ES, Aithal GP, Barnhart H, Gonzalez-Jimenez A, Yang Q, Gu J, Andrade RJ, Hoofnagle JH. A revised electronic version of RUCAM for the diagnosis of DILI. *Hepatology*. 2022;76(1):18–31. doi: 10.1002/hep.32327. Epub 2022 Mar 22. PMID: 35014066; PMCID: PMC9233102.

Figures

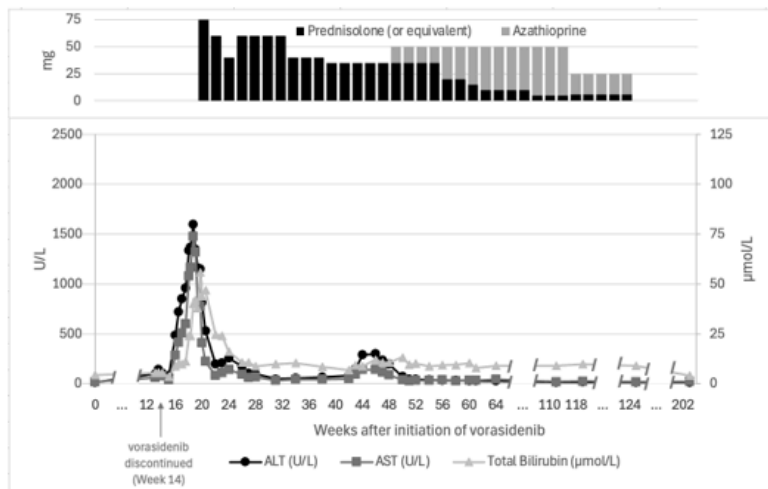
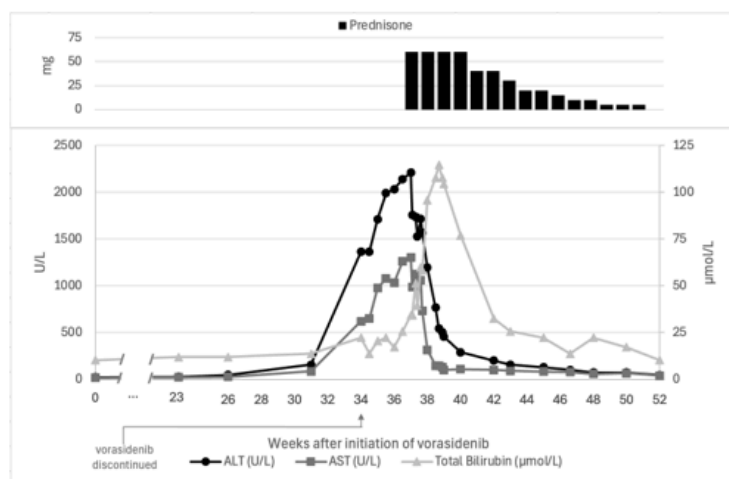
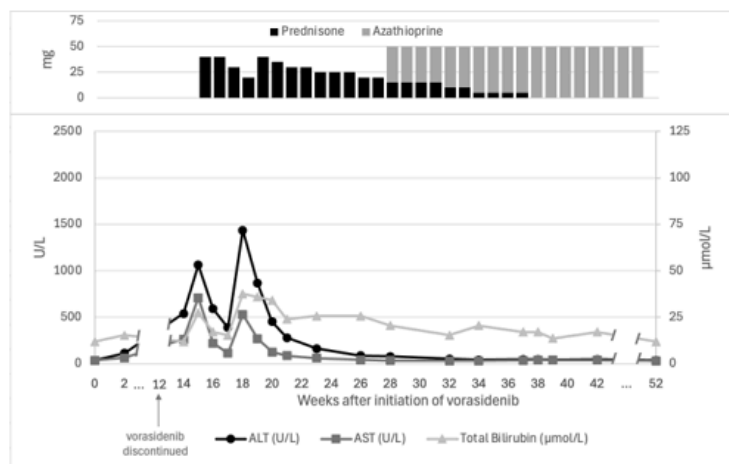


Figure 1

Alanine Aminotransferase, Aspartate Aminotransferase, and Total Bilirubin Activity Trends. (A) Patient 1. (B) Patient 2. (C) Patient 3.

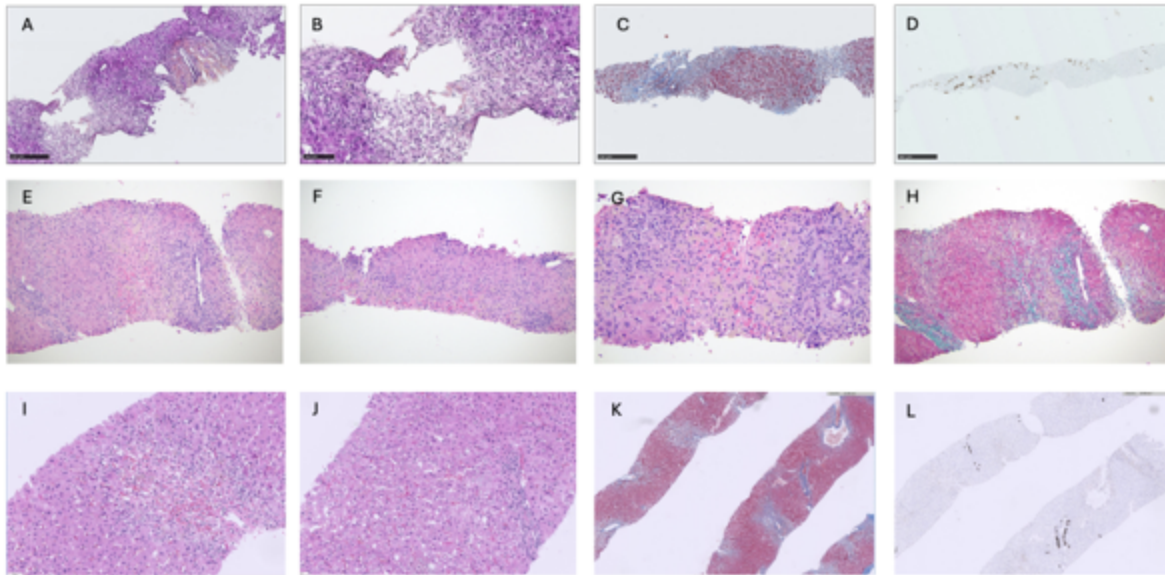


Figure 2

Liver Histology.

Patient 1 (A-D): A: H&E; Portal tract shows interface hepatitis with lymphocytes, plasma cells, neutrophils, and eosinophilic infiltrates. B: H&E; Centrolobular zonal necrosis. C. Masson's Trichrome stain shows no significant fibrosis. D. CK7 IHC: Bile ducts highlighted with few ductular reaction and no ductopenia.

Patient 2 (E-H) E. H&E; Centrilobular regions show marked congestion and extensive hepatocellular necrosis. The necrotic zones are sharply demarcated, creating a characteristic "cookie-cutter" pattern (100×). F. H&E; Necrosis extends to portal tracts, resulting in portal–portal bridging necrosis with intervening parenchymal collapse (pan-acinar necrosis) (100×). G. H&E; Interlobular bile ducts exhibit bile duct injury characterized by cytoplasmic vacuolization, loss of nuclear polarity, and degenerative epithelial changes, accompanied by patchy acute and chronic inflammation (200×). H. Masson trichrome stain shows only minimal centrilobular perisinusoidal fibrosis (100×).

Patient 3 (I-L) I. H&E; higher power view of the centrilobular zone shows zonal necrosis with minimal inflammatory infiltrates and few associated apoptotic hepatocytes. No bridging necrosis or pan lobular necrosis was identified. No significant plasmacytic infiltrates are present. (200×) J. H&E; Portal tract with no significant inflammation, necrosis, nor biliary injury apparent. (200×) K. Masson Trichrome: No significant portal or sinusoidal fibrosis. Pale blue areas correspond to zonal necrosis without significant fibrosis. (50×) L. CK7 IHC: CK7 immunostain highlights intact and unremarkable bile ducts without evidence of biliary proliferation, biliary injury, or ductopenia (50×).

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [VorasicidenibSupplementary.docx](#)