ELMER PRESS

Case Report

J Med Cases. 2023;14(6):185-190

Unique Variant of Zieve Syndrome With a Normal Reticulocyte Count

Apurva Vedire[a, b], Steven Imburgio[a], Harshavardhan Sanekommu[a], Rushi Patel[a], Haley Johnson[a], Sobaan Taj[a], Mohammad Hossain[a]

Abstract

Zieve syndrome is a rare diagnosis seen in patients with chronic alcohol use which consists of a distinct clinical triad of hyperlipidemia, hemolytic anemia, and jaundice. Patients typically have an elevated reticulocyte count due to the hemolytic nature of the anemia. We present the case of a 44-year-old female who was discovered to have an unusual variant of Zieve syndrome with a normal reticulocyte count, which was believed to be due to suppression of bone marrow from excessive alcohol consumption. She was treated with steroids and complete alcohol cessation, with remarkable improvement on subsequent follow-up. An exhaustive literature review of 31 documented cases of Zieve syndrome was conducted to better understand the clinical presentation and overall prognosis of these patients. This case report and literature review aimed to improve patient outcomes through increased recognition of this underrecognized syndrome.

Keywords: Zieve syndrome; Hyperlipidemia; Hemolytic anemia; Jaundice

Introduction

The triad of hyperlipidemia, hemolytic anemia, and jaundice in the setting of chronic alcohol abuse and liver damage compose Zieve syndrome [1]. This constellation of clinical findings was first described by Leslie Zieve in 1957 [2]. It is estimated that this syndrome is seen in approximately 1 in 1,600 hospital admissions [3]. The exact pathogenesis remains elusive, and our current knowledge remains confined to multiple hypotheses. Identification of Zieve syndrome is essential to provide timely treatment and high-value care without subjecting the patient to unnecessary testing in the search for a diagnosis.

Manuscript submitted March 13, 2022, accepted April 21, 2023
Published online xx xx, 2023

[a]Department of Medicine, Jersey Shore University Medical Center, Neptune City, NJ 07753, USA
[b]Corresponding Author: Apurva Vedire, Department of Medicine, Jersey Shore University Medical Center, Neptune City, NJ 07753, USA.
Email: apurva.vedire@hmhn.org

doi: https://doi.org/10.14740/jmc4077

The syndrome is slowly gaining recognition in the literature, and as a result, the clinical significance is not well understood. We present a rare case of Zieve syndrome with the unusual finding of a normal reticulocyte count to bring further awareness of this diagnosis, with an emphasis on the pathogenesis, presenting symptoms and labs, prognosis, and clinical management. We explain why an unconventional treatment strategy might prove beneficial in this unique variant. Additionally, we reviewed the literature for cases of Zieve syndrome dating back to 1958 to better characterize the clinical presentation and overall survival rates of this condition.

Case Report

Investigations

A 44-year-old female with a past medical history significant for hypothyroidism and alcohol use disorder, presented with a complaint of jaundice for the last 10 days. She also reported a 2-month history of weakness and lethargy. A targeted review of the systems was found to be negative. She reported a history of drinking three to four glasses of hard liquor every night for many years. Her only medication was 50 µg of levothyroxine. In the emergency department, the patient had tachycardia of 103 beats per minute with all other vitals within normal limits. Physical exam was significant for mild tachycardia, scleral icterus, jaundice, and mild abdominal distension with no fluid shift noted. She reported no family history of autoimmune disorders or liver disease, no recent blood transfusions, and no other over the counter medications or herbal supplements.

Diagnosis

Initial laboratory values on admission are shown in Table 1.

Other workup included a blood alcohol level of 0.306% (reference range < 0.005%), negative beta human chorionic gonadotropin hormone, normal ceruloplasmin levels and normal hepatitis panel. Peripheral blood smear showed mild anemia, mild thrombocytopenia, along with slight anisocytosis, slight macrocytes, moderate target cells. Direct Coomb's showed a negative elution test and hence it was not considered significant by the lab. The anemia was hemolytic and non-immune in origin. Thus, laboratory findings were consistent with

Articles © The authors | Journal compilation © J Med Cases and Elmer Press Inc™ | www.journalmc.org
This article is distributed under the terms of the Creative Commons Attribution Non-Commercial 4.0 International License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited

Table 1. Initial Laboratory Values on Admission

Labs	Value (unit)	Reference range
White blood cell	4.4 × 10^3/µL	4.5 - 11.0 × 10^3/µL
Red blood cell	2.85 × 10^6/µL	4.10 - 5.10 × 10^6/µL
Hemoglobin	10.2 g/dL	12 - 16 g/dL
Platelet count	110 × 10^3/µL	140 - 450 × 10^3/µL
Total bilirubin	16.9 mg/dL	0.2 - 1.3 mg/dL
Direct bilirubin	12.8 mg/dL	0.0 - 0.3 mg/dL
Alkaline phosphatase	287 U/L	46 - 116 U/L
Aspartate aminotransferase	333 U/L	0 - 34 U/L
Alanine transaminase	25 U/L	10 - 49 U/L
Reticulocyte count	2.06%	0.40-2.50%
Haptoglobin	1 mg/dL	40 - 280 mg/dL
Lactate dehydrogenase	326 U/L	120 - 246 U/L
Ferritin	2,718.3 ng/mL	7.3 - 270.7 ng/mL
Iron	85 µg/dL	50 - 170 µg/dL
Total iron binding capacity	157 µg/dL	250 - 425 µg/dL
Transferrin	68 mg/dL	250 - 380 mg/dL
Thyroid-stimulating hormone	39.660 µIU/mL	0.550 - 4.780 µIU/mL
Gamma glutamyl transpeptidase	461 U/L	< 38 U/L
Immunoglobulin A	560 mg/dL	40 - 350 mg/dL
Ammonia	76 µmol/L	11 - 32 µmol/L
Cholesterol	232 mg/dL	< 200 mg/dL
Triglycerides	249 mg/dL	0 - 150 mg/dL
High-density lipoprotein	< 20 mg/dL	40 - 60 mg/dL
Low-density lipoprotein	134 mg/dL	< 100 mg/dL

Zieve syndrome. Hemoglobin continued to downtrend during the patient's hospital stay (Table 2).

Treatment

Due to downtrending hemoglobin, gastroenterology was consulted. Additionally, a psychiatry consult was placed for assistance with alcohol cessation. The patient was started on appropriate therapy with furosemide and spironolactone, along with lactulose. She was monitored for alcohol withdrawal and counseled extensively on alcohol cessation. She was also recommended for an outpatient esophagogastroduodenoscopy to evaluate for varices. Discriminant function [4] was noted to be elevated at 47 so she was started on a 1-month course of prednisolone daily. Her condition improved and she was discharged with advice to follow-up with both gastroenterology and psychiatry as an outpatient.

Table 2. Follow-Up Hemoglobin Values

Hospital day	Hemoglobin value (reference range 12 - 16 g/dL)
1	10.2 g/dL
2	8.5 g/dL
3	7.9 g/dL

Follow-up and outcomes

At a follow-up visit 1 month later, the patient abstained from alcohol and completed the prescribed steroid course. Repeat labs (Table 3) at this time showed improvement in all three cell lines and a decrease in the total bilirubin suggesting resolution of hemolysis.

Table 3. Follow-Up Laboratory Values 1 Month Later

Labs	Value	Reference range
Total bilirubin	4.1 mg/dL	0.2 - 1.3 mg/dL
White blood cell	9.8 × 10^3/µL	4.5 - 11.0 × 10^3/µL
Hemoglobin	12.6 g/dL	12 - 16 g/dL
Platelet count	188 × 10^3/µL	140 - 450 × 10^3/µL

Articles © The authors | Journal compilation © J Med Cases and Elmer Press Inc™ | www.journalmc.org

Discussion

Chronic alcohol use predisposes individuals to a wide range of adverse medical conditions such as macrocytic anemia, alcoholic hepatitis, and cirrhosis [5]. The goal of our case is to bring awareness to a rarely documented and an underrecognized complication of alcohol abuse known as Zieve syndrome. This disease process was first described by Dr. Leslie Zieve in 1958 and is composed of a specific constellation of clinical and laboratory findings which includes hyperlipidemia, hemolytic anemia, and jaundice [2]. The pathophysiology of Zieve syndrome remains poorly defined, but multiple theories have been proposed. It is suspected that the hemolysis seen in these patients results primarily from a profound elevation in triglycerides. The altered synthesis of lipoproteins, cholesterol, and fatty acids may induce hemolysis via instability of the erythrocyte membrane [6]. Recent studies now suggest that both toxic metabolites from ethanol and alcohol-induced vitamin E deficiency may also compound the red blood cell destruction through impaired functioning of crucial metabolic enzymes [7, 8]. The multifactorial mechanism for red blood cell destruction directly results in hemolytic anemia, contributing also to an elevation in unconjugated bilirubin levels. Often the hyperbilirubinemia that is seen is substantial as concurrent alcohol-mediated hepatocellular toxicity induces chronic cholestatic injury [9].

Clinicians should have a high degree of clinical suspicion for Zieve syndrome in individuals with a known history of excessive alcohol use who present with hemolytic normocytic anemia and hyperbilirubinemia, especially when subsequent workup is negative for gastrointestinal bleeding. The hemolytic nature of anemia can aid as a useful diagnostic tool to differentiate Zieve syndrome from isolated cases of alcohol dependence which often involves macrocytic anemia [10]. While Zieve syndrome typically can be identified by the classic triad, additional clinical findings may aid in the diagnosis. For example, abdominal pain was a universal finding seen in all 20 patients that Leslie Zieve studied [2]. Additional nonspecific symptoms that have been reported in the literature include fatigue, fever, nausea, and vomiting [11]. Given the hemolytic nature of anemia, labs generally reveal elevated reticulocyte count, increased lactate dehydrogenase (LDH), and decreased haptoglobin, with elevations in direct bilirubin and alkaline phosphatase. Coomb's test is also negative [12]. The clinical importance of recognizing this syndrome comes from the fact that most patients rapidly recover within weeks of alcohol cessation [13]. Following alcohol abstinence, there is an intracellular shift of lipids from the plasma to adipose tissue in hepatocytes, thus eliminating the hypertriglyceridemia which is a suspected inciting trigger for the development and propagation of this unique syndrome [12].

The excessive alcohol consumption that is seen in this specific patient demographic can have toxic effects on the bone marrow as well. Suppression of hematopoietic stem cells results in impaired production of all three major cell lines, leading to decreased production of white blood cells, red blood cells, and platelets [14, 15]. It is possible that the bone marrow suppression induced by chronic alcohol use in our patient was insufficient to mount a proper response to the hemolytic environment in our patient. The most effective treatment for Zieve syndrome continues to be complete abstinence from alcohol [16]. Unconventional therapies such as plasmapheresis in a patient with massive elevations in triglyceride have also been reported [17]. It is believed that the commonly used scoring system of Maddrey discriminant function, which screens alcoholic patients who benefit from steroids, is ineffective in Zieve syndrome due to the temporary elevations in bilirubin levels that normalize after alcohol cessation [18]. In fact, the recommendation is to avoid corticosteroids entirely due to a lack of any proven efficacy [19]. While this may be true for patients with Zieve syndrome that have functional marrow, our patient was seen to benefit from both alcohol cessation in addition to steroids. We believe this to be due to the effect of steroids to stimulate the release of cells from the bone marrow, which has been proven in prior studies [20]. Our success mirrors that of other conditions such as hereditary spherocytosis, where corticosteroids can result in appropriate elevation in reticulocyte count with subsequent improvement in hemoglobin [21].

To date, there is a substantial gap in knowledge regarding the clinical presentation and overall prognosis of patients affected by Zieve syndrome. Following the initial pronouncement of this syndrome by Leslie Zieve in 1958, there have been over 100 documented cases in the literature. The only aggregate analysis of these case reports that we identified was by Liu et al in 2017, with a brief review focusing on the time period, country of origin, and subtype of each publication [22]. As a result, we aimed to conduct a more thorough review of documented case reports of Zieve syndrome with an emphasis on clinical presentation, lab values, and treatment outcomes while also including any additional cases that were published after 2017. After an extensive PubMed literature review of all case reports dating back to 1958 using the search term "Zieve syndrome", we identified 31 documented cases [1, 3, 8, 11, 16, 17, 19, 22-31]. Inclusion criteria included: publications dated after Leslie Zieve's original manuscript and cases that met all three components of the clinical triad including hyperlipidemia, hemolytic anemia, and jaundice. Exclusion criteria included: articles that were not open access or written in English text.

We document and summarize our findings below (Table 4 [1, 3, 8, 11, 16, 17, 19, 22-31], and Table 5, respectively). Overall, patients that met the triad for Zieve syndrome were on average 42.2 years of age with 54.8% being male. While abdominal pain was reported in nearly all of Zieve's original 20 patients and has historically been considered a classic associated feature of this syndrome, our analysis demonstrates that this complaint is seen in just slightly over half of patients. Over the 31 patients we collected data on, the average initial triglyceride level was significantly elevated at 822.5 mg/dL. A majority of patients were anemic with an average hemoglobin of 8.2 g/dL and all patients reported so far had an elevated reticulocyte count. Overall, we demonstrate that Zieve syndrome carries a favorable prognosis, with 93.5% [29, 31] of individuals being successfully discharged and only two patients experiencing inpatient mortality.

It is important to recognize the typical findings that characterize Zieve syndrome since this diagnosis is relatively un-

Table 4. Literature Review Findings

Study	Age (years old)	Sex	Abdominal pain present?	Initial triglyceride levels (mg/dL)	Initial hemoglobin level (g/dL)	Initial reticulocyte count (%)	Initial total bilirubin level (mg/dL)	Initial direct bilirubin level (mg/dL)	Alive at discharge?
Gitlin, 1969 [23]	48	Male	Yes	441	12.5	5.0	10.0	9.0	Yes
Benraad et al, 1977 [24]	28	Male	Yes	137.4	8.7	3.7	1.2	0.2	Yes
	31	Male	Yes	130.0	7.9	5.8	1.6	0.5	Yes
	50	Male	Yes	3.85	8.0	5.1	1.8	1.0	Yes
	33	Female	Yes	3.03	8.1	13.5	0.9	0.4	Yes
	56	Female	No	3.27	8.7	14.0	25.2	20.4	Yes
	49	Female	Yes	327	6.3	21.0	5.0	3.0	Yes
	41	Male	Yes	3.4	8.2	12.2	2.3	1.1	Yes
	41	Male	No	4.14	7.3	13.2	3.2	1.8	Yes
	63	Male	No	N/A	7.4	5.55	13.5	8.8	Yes
	65	Male	No	4.71	8.0	20.0	13.1	10.4	Yes
	43	Male	No	34.7	6.8	5.4	2.1	1.3	Yes
	30	Female	No	4.05	6.8	8.1	3.6	2.2	Yes
	31	Female	No	2.7	6.6	8.3	2.2	1.1	Yes
	32	Female	Yes	4.0	5.8	12.6	1.7	0.7	Yes
	29	Male	Yes	10.7	7.7	7.6	1.8	0.9	Yes
Pickens et al, 1979 [25]	66	Female	No	168.3	5.7	11.5	16.08	N/A	No
Martin et al, 1996 [26]	59	Female	Yes	1,700.5	8.8	5.8	2.4	N/A	Yes
Pilcher et al, 1996 [27]	39	Male	Yes	11,425.5	13.2	N/A	1.0	N/A	Yes
Piccini et al, 2003 [3]	47	Female	Yes	N/A	7.7	3.5	4.2	2.3	Yes
Hashmi et al, 2014 [16]	30	Female	No	578.0	10.6	8.1	38.0	26.0	Yes
Shukla et al, 2015 [11]	45	Male	No	N/A	6.5	N/A	16.0	6.3	Yes
Senatore et al, 2016 [19]	32	Male	Yes	464	8.5	N/A	20.0	13.0	Yes
Liu et al, 2017 [22]	30	Female	No	311.9	7.7	6.1	21.3	13.5	Yes
Gremida et al, 2018 [1]	46	Female	Yes	208.0	7.1	11.0	12.0	2.0	Yes
Abughanimeh et al, 2019 [28]	58	Male	Yes	N/A	5.5	16.2	4.0	N/A	No
Choudhry et al, 2019 [8]	46	Male	Yes	4,425.0	12.6	6.2	2.9	2.0	Yes
Achufusi et al, 2020 [29]	31	Female	No	8,890.0	8.1	N/A	19.1	8.7	Yes
Gosal et al, 2021 [30]	38	Female	Yes	172.0	6.9	5.7	11.2	2.0	Yes
Sams et al, 2022 [17]	30	Male	Yes	5,679.0	12.5	N/A	2.1	N/A	Yes
Sivanandam et al, 2022 [31]	42	Male	No	215.0	8.4	N/A	10.9	4.4	Yes

N/A: not available.

known. A high index of clinical suspicion is warranted in patients with alcohol use disorder who present with hemolytic anemia and jaundice. Additionally, a special emphasis should be placed on studying rare variants, such as our patient with a normal reticulocyte count, as they might help uncover undiscovered mechanisms at play. Ultimately, while our data suggest that this condition carries a favorable prognosis with timely identification and treatment interventions, further research

Table 5. Summary of Table 4 Findings

	Age	Sex	Abdominal pain present?	Initial triglyceride levels	Initial hemoglobin level	Initial reticulocyte count	Initial total bilirubin level	Initial direct bilirubin level	Alive at discharge?
Average	42.2 years old	54.8% male	58.1%	822.5 mg/dL	8.2 g/dL	9.4%	8.7 mg/dL	5.5 mg/dL	93.5%

Articles © The authors | Journal compilation © J Med Cases and Elmer Press Inc™ | www.journalmc.org

regarding the pathophysiology of this syndrome is needed to better target future treatment strategies.

Learning points

Our case aims to bring further awareness to the rare combination of clinical features, along with the complex pathophysiology, which underlies Zieve syndrome. Additionally, we present a unique subtype of Zieve syndrome with bone marrow suppression as demonstrated by a paradoxically low reticulocyte count which benefited from the use of corticosteroids. Following an extensive review of the literature, it is evident that Zieve syndrome can be promptly identified by the classical triad of clinical features on presentation including hyperlipidemia, hemolytic anemia, and jaundice. Additionally, abdominal pain may not be as reliable of a clinical finding as once previously thought. We demonstrate that appropriate intervention, primarily alcohol cessation, can result in promising recovery for these patients. Ultimately, we hope that our contribution to the knowledge of Zieve syndrome may lead to improved patient outcomes and a more robust treatment protocol in the future. We also hope to increase recognition of this rare variant of Zieve syndrome.

Acknowledgments

None to declare.

Financial Disclosure

None to declare.

Conflict of Interest

None to declare.

Informed Consent

Informed consent was taken from the patient before writing this case.

Author Contributions

Each author was individually involved in and made substantial contributions to conceptions and designs, acquisition of data, analysis, interpretation of data, drafting, and editing the manuscript. AV and SI contributed to the designs, acquisition of data, analysis, interpretation of data, drafting, and editing of the manuscript. HS and RP contributed to the drafting and editing of the manuscript. ST contributed to the designs and analysis of the manuscript. MH contributed to the designs, analysis, interpretation of data, and editing of the manuscript.

Data Availability

The authors declare that data supporting the findings of this study are available within the article.

References

1. Gremida A, Paleti S, Perez ET, McCarthy D. Hemolysis in alcoholic liver disease: Zieve's syndrome. Dig Dis Sci. 2018;63(3):601-604. doi pubmed
2. Zieve L. Jaundice, hyperlipemia and hemolytic anemia: a heretofore unrecognized syndrome associated with alcoholic fatty liver and cirrhosis. Ann Intern Med. 1958;48(3):471-496. doi pubmed
3. Piccini J, Haldar S, Jefferson B. Cases from the Osler medical service at Johns Hopkins University. Zieve syndrome. Am J Med. 2003;115(9):729-731. doi pubmed
4. Degre D, Wandji LCN, Moreno C, Louvet A. Alcoholic hepatitis: Towards an era of personalised management. United European Gastroenterol J. 2020;8(9):995-1002. doi pubmed pmc
5. Rehm J. The risks associated with alcohol use and alcoholism. Alcohol Res Health. 2011;34(2):135-143. pubmed pmc
6. Hilgard P, Schreiter T, Stockert RJ, Gerken G, Treichel U. Asialoglycoprotein receptor facilitates hemolysis in patients with alcoholic liver cirrhosis. Hepatology. 2004;39(5):1398-1407. doi pubmed
7. Goebel KM, Goebel FD, Schubotz R, Schneider J. Red cell metabolic and membrane features in haemolytic anaemia of alcoholic liver disease (Zieve's syndrome). Br J Haematol. 1977;35(4):573-585. doi pubmed
8. Choudhry F, Kathawa J, Kerton K, Farshadsefat S, Piper M. Zieve's syndrome presenting with severe hypertriglyceridemia. ACG Case Rep J. 2019;6(7):e00133. doi pubmed pmc
9. Mueller S, Chen C, Mueller J, Wang S. Novel insights into alcoholic liver disease: iron overload, iron sensing and hemolysis. J Transl Int Med. 2022;10(2):92-124. doi pubmed pmc
10. Seppa K, Sillanaukee P. Red cell distribution width in alcohol abuse and iron deficiency anemia. JAMA. 1992;267(8):1070; author reply 1071. pubmed
11. Shukla S, Sitrin M. Hemolysis in acute alcoholic hepatitis: Zieve's syndrome. ACG Case Rep J. 2015;2(4):250-251. doi pubmed pmc
12. Reyes JVM, Ahmad S, Majeed H, Kandoth E, Lieber JJ. Zieve syndrome: a clinical triad, or perchance a quartet? J Investig Med High Impact Case Rep. 2022;10:23247096221121393. doi pubmed pmc
13. Reuben A. True, true… but are they related? Hepatology. 2006;43(3):635-639. doi pubmed
14. Ballard HS. The hematological complications of alcoholism. Alcohol Health Res World. 1997;21(1):42-52. pubmed pmc
15. Heermans EH. Booze and blood: the effects of acute and chronic alcohol abuse on the hematopoietic system. Clin

Lab Sci. 1998;11(4):229-232. pubmed
16. Hashmi S, Allison MG, McCurdy MT, Reed RM. Hyperbilirubinaemia and haemolytic anaemia in acute alcoholic hepatitis: there's oil in them thar veins. BMJ Case Rep. 2014;2014:bcr2014203804. doi pubmed pmc
17. Sams LE, Krappe J, Czihal M, Hoppe JM. Zieve syndrome presenting with lipaemia and treated by plasmapheresis. BMJ Case Rep. 2022;15(4):e245257. doi pubmed pmc
18. Haider AA, Salabei JK, Hashmi M, Ismail Z, Zheng M, Iyer UG. Zieve syndrome in a patient with hepatitis C. Cureus. 2022;14(5):e25412. doi pubmed pmc
19. Senatore FJ, McDonald K. Pitfalls of treating alcoholic hepatitis: recognizing hemolytic anemia in Zieve's syndrome. Am J Gastroenterol. 2016;111(4):577-579. doi pubmed
20. Bishop CR, Athens JW, Boggs DR, Warner HR, Cartwright GE, Wintrobe MM. Leukokinetic studies. 13. A non-steady-state kinetic evaluation of the mechanism of cortisone-induced granulocytosis. J Clin Invest. 1968;47(2):249-260. doi pubmed pmc
21. Duru F, Gurgey A. Effect of corticosteroids in hereditary spherocytosis. Acta Paediatr Jpn. 1994;36(6):666-668. doi pubmed
22. Liu MX, Wen XY, Leung YK, Zheng YJ, Jin MS, Jin QL, Niu JQ. Hemolytic anemia in alcoholic liver disease: Zieve syndrome: A case report and literature review. Medicine (Baltimore). 2017;96(47):e8742. doi pubmed pmc
23. Gitlin N. Zieve's syndrome and porphyrinuria in an alcoholic. Br Med J. 1969;1(5636):96-98. doi pubmed pmc
24. Benraad HB, Penn HJ, Pieters GF, Tan HS. Zieve's syndrome. J R Coll Physicians Lond. 1977;12(1):42-52. pubmed pmc
25. Pickens S. Fatality after hepatic angiography in Zieve's syndrome. Postgrad Med J. 1979;55(654):903-904. doi pubmed pmc
26. Martin JC, Ross A, Watson D, O'Sullivan MM. A case of Zieve's syndrome presenting with myalgia: not to be confused with polymyalgia rheumatica. Br J Rheumatol. 1996;35(5):495-496. doi pubmed
27. Pilcher CR, Underwood RG, Smith HR. Zieve's syndrome a potential surgical pitfall? J R Army Med Corps. 1996;142(2):84. doi pubmed
28. Abughanimeh O, Kaur A, Numan L, Bahaj W, Madhusudhana S. Zieve's syndrome: an under-reported cause of anemia in alcoholics. Cureus. 2019;11(2):e4121. doi pubmed pmc
29. Achufusi TG, Sandhu J, Chahal J, Shepherd Z, Mirchia K. Zieve syndrome in acute alcoholic hepatitis. Proc (Bayl Univ Med Cent). 2020;33(3):380-381. doi pubmed pmc
30. Gosal K, Singh P, Westbrook K, Carter MK. Underrecognized Zieve's syndrome, A case report. Ann Med Surg (Lond). 2021;66:102464. doi pubmed pmc
31. Sivanandam LK, Kannan A, Srinivasan AA, Bammigatti C. A rare case of extensive cutaneous spider angioma in Zieve's syndrome: A case report and review of literature. J Family Med Prim Care. 2022;11(7):4036-4038. doi pubmed pmc

Articles © The authors | Journal compilation © J Med Cases and Elmer Press Inc™ | www.journalmc.org