Annals of Clinical Case Reports

Case Summary
Published: 09 Apr, 2019

Iatrogenic Anemia and Liver Injury after Myocardial Infarction

*Ekknoor Sahota**

509 South Highland Avenue, USA

Abstract

Drug-Induced Liver Injury (DILI) is gaining recognition as a cause of acute and chronic liver disease, with statins being commonly implicated agents. Though a relatively rare presentation of DILI, there has been growing evidence of statin induced Autoimmune Hepatitis (AIH)-like liver injury which alters the management and prognosis of DILI. We present a case of DILI unique for the presence of anemia in the setting of abnormal hemolytic markers. The initial workup of hemolytic anemia led to the diagnosis of DILI and recognition of chronic blood loss exacerbated by medications as the etiology of the anemia instead.

Case Presentation

A 48 yr old female presented to the emergency department for evaluation of jaundice, abdominal pain, and generalized fatigue for one week.

Past medical history was significant for uterine fibroids and ST-segment elevation myocardial infarction four months ago, and she was prescribed aspirin, ticagrelor, and atorvastatin at that time.

On physical examination, patient was noted to have pallor and scleral icterus. Laboratory investigations were notable for hemoglobin 8.3 gm/dL, marked elevation of ALT 1100 IU/L, AST 1529 IU/L. Anti-nuclear antibody (ANA) was positive (1:160) and Anti-Smooth Muscle Antibody (ASMA) was positive (1:320) as well. Antibody testing for hepatitis viruses (A, B, C, D, E), Cytomegalovirus and Epstein-Barr virus was negative.

CT scan of the abdomen revealed cholelithiasis, but MRCP was negative for cholestasis.

Liver biopsy showed-moderately severe predominantly lobular hepatitis, mild portal fibrosis, consistent with Statin induced AIH-like liver injury.

Anemia was microcytic and further investigation revealed elevation of Lactate Dehydrogenase (LDH) at 489 IU/L and decrease in haptoglobin at 10 mg/dL. Hemolysis was suspected, however, direct antiglobulin test was negative and reticulocyte count was low, favoring a non-hemolytic process. Additionally, peripheral blood smear was negative for schistocytes, spherocytes or bite cells. It came to light that the patient had been experiencing heavy menstrual bleeding over the last four months since initiation of aspirin and ticagrelor, after she was diagnosed with STEMI and a drug eluting stent was placed during previous admission. Iron studies were ordered and were

Table 1: On physical examination, patient was noted to have pallor and scleral icterus.

Laboratory markers	Results	Reference range	Laboratory markers	Results	Reference range
WBC	9	4.40-11.30 k/mcl	Transferrin	394.9	170-370 mg/dl
Hemoglobin	8.3	12.3-15.3 g/dl	TIBC	565	240-450 mcg/dL
MCV	74	80-96 fL	Total bilirubin	4.1	0.2-1.2 mg/dL
Platelets	199	145-445 k/mcl	Direct bilirubin	2.6	<0.3 mg/dL
AST	1529	17-59 U/L	G6PD	265	5.5-20.5 U/gm of hb
ALT	ALT	ALT	ALT	ALT	ALT
Alkaline- phosphatase	515	35-140 U/L	ANA	0.152777778	Negative
LDH	489	110-216 U/L	Direct Coombs	Negative	Negative
Haptoglobin	<10	16-200 mg/dl	AMA	Negative	Negative
Ferritin	90	20.0-300.0 ng/ml	ASMA	0.263888889	Negative
Serum Iron	59	60-170 mcg/dL	Ceruloplasmin	30.6	20-35 mg/dL

OPEN ACCESS

Correspondence:
Ekknoor Sahota, 509 South Highland Avenue, USA, Tel: 4124037174; E-mail: ek.sahota@gmail.com

Received Date: *20 Mar 2019*
Accepted Date: *05 Apr 2019*
Published Date: *09 Apr 2019*

Citation:
Sahota E. Iatrogenic Anemia and Liver Injury after Myocardial Infarction. Ann Clin Case Rep. 2019; 4: 1645.
ISSN: 2474-1655

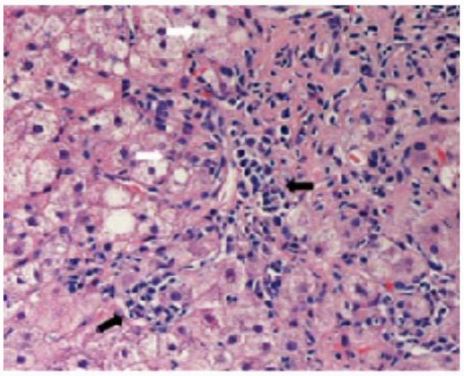

Figure 1: Autoimmune hepatitis: marked interface activity with disruption of the limiting plate (middle, white arrows). Adjacent hepatocytes are swollen, suggesting hepatocyte injury. Interface activity is dominated by plasma cells (black arrows).

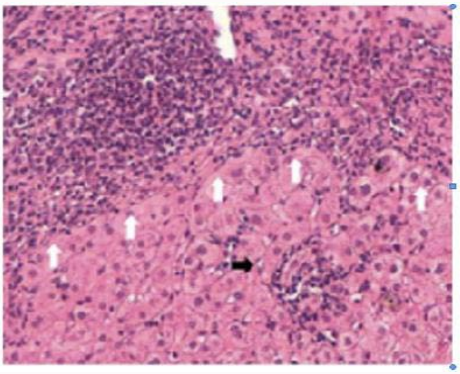

Figure 2: Drug induced liver injury: markedly expanded portal tract with marked, plasma cell-rich inflammation and interface activity (white arrows). There are scattered foci of inflammation in the lobules (black arrows).

consistent with iron deficiency anemia. The anemia improved with iron replacement therapy. With cessation of anti-platelet therapy and atorvastatin, her menorrhagia improved and her LFTs returned to baseline within four weeks.

Discussion

This case highlights the presence of two coexisting pathological processes-Iron deficiency Anemia and Drug induced liver injury, and the diagnostic challenges associated with both. A holistic approach, including detailed history and investigations is necessary for an accurate diagnosis. As seen in this case, etiologies other than hemolysis may be responsible for abnormal hemolytic markers. LDH is non-specific, unless it is fractionated, and low haptoglobin has been associated with other conditions, including liver fibrosis and severe liver disease. A negative DAT, low reticulocyte count and normal peripheral blood smear point towards a diagnosis other than hemolysis.

Statins have been implicated as a cause of DILI with a wide spectrum of histological presentations. Autoimmune hepatitis-like phenotype is a rare in this spectrum and one that needs close monitoring. Immunotherapy may be required if LFTs do not improve after cessation of the drug.

Notably, both pathological processes were in part, if not entirely, iatrogenic and caused by two different classes of drugs-emphasizing the importance of understanding the interplay of multiple factors in one patient.

References

1. Verma S, Kaplowitz N. Diagnosis, management and prevention of drug-induced liver injury. Gut. 2009;58(11):1555-64.
2. Shinton NK, Richardson RW, Williams JDF. Diagnostic value of serum haptoglobin. J Clin Pathol. 1965;18(1):114-8.
3. Suzuki A, Brunt EM, Kleiner DE, Miquel R, Smyrk TC, Andrade RJ, et al. The use of liver biopsy evaluation in discrimination of idiopathic autoimmune hepatitis versus drug-induced liver injury. Hepatology. 2011;54(3):931-9.
4. Cole. Autoimmune hepatitis.