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Methylprednisolone induced recurrent acute liver injury in a patient with multiple sclerosis

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Abstract

Background: Methylprednisolone (MP) induced acute liver injury has been reported in patients who received pulse steroid therapy. Several case reports and a case series of MP induced hepatotoxicity in patients with Multiple Sclerosis (MS) and Graves ophthalmopathy have been published. Most of the cases had a complete recovery, but few had suffered very severe disease that led to death, however no case fatality reported in patient with MS.

Case summary: we presented a case of recurrent acute liver injury in a patient with multiple sclerosis. She had three attacks of acute liver injuries with normal liver function tests in between the attacks and no signs of chronic liver disease either clinically or radiographically. 3-6 weeks before each episode, she had treatment of MS relapses with IV MP, dose range 1.5 to 2.5 gm. No clear cause was identified except for consistent association with IV methylprednisolone. RECAM score which is the electronic version of RUCAM (Roussel Uclaf Causality Assessment Method) used to assess the likelihood of drug-induced liver injury [1] was calculated and the result was the association between MP and acute liver injury in this patient is highly likely or highly probable (score of 12). With each episode of acute liver injury, the time to onset decreased, the attack became more severe, and the duration of illness was prolonged

Conclusion: Methylprednisolone induced liver injury has been reported. In patients who have indication for pulse methylprednisolone, liver function should be monitored carefully and for several weeks after the therapy is completed. As the patients who are required methylprednisolone may need the treatment again in their lifetime, change to other steroid treatment or other alternative treatments may be necessary to avoid severe hepatotoxicity that may be catastrophic.

Keywords: Methylprednisolone; Liver injury; Hepatitis; Case report; Hepatotoxicity; MS; Multiple sclerosis; Drug induced liver injury; DILI.

Introduction

Acute liver injury can be significantly caused by medications, a condition referred to Drug-Induced Liver Injury (DILI). It is estimated that DILI occurs in approximately 14 to 19 cases per 100,000 individuals. This condition is associated with considerable morbidity and mortality. Around 53% of affected patients need hospitalization, 2% may require a liver transplant, and the mortality rate is estimated to be about 5% [2]. DILI can be categorized into intrinsic and idiosyncratic. Intrinsic DILI is dose-dependent, while idiosyncratic DILI is unpredictable and not linked to dosage. The latter often involves a latent period between the initiation of the causative agent and the onset of hepatotoxicity [3]. Corticosteroids are effective in managing a wide range of medical conditions, including but not limited to multiple sclerosis, Graves' ophthalmopathy, transplant rejection, bronchial asthma, adrenal insufficiency, inflammatory diseases, and certain autoimmune disorders [4]. Conversely,

corticosteroids can lead to significant side effects that impact multiple organs. Hepatotoxicity associated with corticosteroids may manifest as hepatic steatosis, liver enlargement, the onset or worsening of steatohepatitis, hepatic glycogenosis, and the reactivation of hepatitis B and C. Additionally, there is a risk of severe autoimmune hepatitis following the cessation of steroid therapy. The severity of these effects often correlates with the treatment duration and dosage [4].

Methylprednisolone (MP) is a synthetic, intermediate acting glucocorticoid. MP induced direct hepatotoxicity is rare but have been reported in case series and case reports [5]. This adverse drug reaction is especially reported in patients with Graves ophthalmology and Multiple Sclerosis (MS) relapses [5].

Case presentation

A 39-year-old female presented with a three-days history of upper abdominal pain, yellowish discoloration of the eyes, and dark urine. Also reported nausea and episodes of non-bloody vomiting. She denied any fever, mouth ulcers, diarrhea or weight loss. Her medical history includes relapsing-remitting multiple sclerosis, hypothyroidism, and migraine. There was no family history of thromboembolic or rheumatological illnesses. She is non-smoker and does not consume alcohol. Notably, 20 days prior to her presentation, she experienced an MS relapse for which she was treated with intravenous methylprednisolone 500 mg for 5 days. At presentation, her initial vital signs were: temperature: 37.5°C, blood pressure: 130/76 mmHg, pulse rate: 71/min, respiratory rate: 19/min, and oxygen saturation: 100% on room air. Physical examination revealed a slight tinge of jaundice, but was otherwise unremarkable, with normal abdominal and neurological assessments. Laboratory results indicated a normal CBC, renal function tests, and electrolyte levels. However, liver function tests showed markedly elevated ALT (653 U/L), AST (783 U/L), and ALP (182 U/L), while total bilirubin was 26 µmol/L and INR was 1.4. The patient was admitted for further evaluation and management, with daily monitoring of Liver Function Tests (LFTs) and INR.

Over the course of hospitalization, ALT levels rose to 1324 U/L before beginning to decline, while AST peaked at 1856 U/L. INR increased to 2, and ALP reached a maximum of 184 U/L. all autoimmune markers were unremarkable Doppler ultrasound of the hepatic veins and arteries, along with MRI of the liver, returned negative results. Subsequently, Liver biopsy was performed and showed moderate balloon degeneration associated with portal and lobular inflammation comprising lymphocytes, scattered neutrophils and some eosinophils. Few plasma cells are identified. Acidophil bodies are noted. A few areas show bridging and zone 3 necrosis. Impression was moderate to severe acute hepatitis pattern. Possible causes include medication and infection induced liver injury. The features are not characteristic of autoimmune hepatitis though it cannot be totally excluded.

Upon reviewing her past medical history, the patient experienced two episodes of acute liver injury, both of which were preceded by relapses of Multiple Sclerosis (MS) treated with Intravenous Methylprednisolone (IVMP), followed by complete recovery.

The patient had the first episode of acute liver injury in 2016, she was 30 weeks pregnant at that time. She had been admitted on 31/08/2016 with vaginal bleeding diagnosed as placenta previa. 5 days before admission on 27/08/2016, she was diagnosed with MS relapse and was given 500 mg IV methylprednisolone for 3 days. On 04/10/2016 (39 days after the first dose of MP) and while the patient was observed in the hospital for placenta previa, she developed generalized itching and dark urine, LFT showed ALT was 349 U/L, AST 296 U/L, ALP 188 U/L, Total bilirubin of 99 umol/L, direct bilirubin 72 umol/L and INR was 1.4, In keeping with hepatocellular injury. Workup for liver disease was done, viral hepatitis, Ceruloplasmin, alpha 1 antitrypsin were negative. US liver was normal except for a hemangioma of 0.9*0.9 cm. ANA and anti-smooth muscle antibody were positive at level 1:80. Autoimmune hepatitis was suspected; however, no treatment was given and her LFT was improving daily. Her LFT was normalized over a 10-day period. Patient followed in the hepatology clinic, US elastography done later which was negative for fibrosis, repeated Autoimmune hepatitis panel was negative.

The second episode of acute liver injury occurred in 2020, when the patient presented with yellowish discoloration of the sclera and dark urine lasting for 3 days. Notably, 31 days prior to her presentation, she experienced a relapse of multiple sclerosis and was treated with 500 mg of intravenous methylprednisolone for 5 days. All hepatitis workup, including autoimmune tests, returned negative results. Imaging studies, including a liver ultrasound and MRCP, were unremarkable. Her liver function tests showed daily improvement, and she fully recovered over the course of 6 weeks.

Upon the last admission patient received supportive treatment, including IV fluids. Throughout her hospitalization, there were no signs of hepatic encephalopathy or bleeding. Her liver function tests continued to improve and discharged on day 20 in good condition, diagnosed with drug-induced acute liver injury.

She returned for follow-up in the hepatology clinic, where her liver function tests had normalized, and her symptoms had completely resolved since discharge.

Table 1: Show the characteristics and comparison of each episode of acute liver injury.

Parameter/Year	2016	2020	2024
Time to onset(days)	39	31	20
MP total dose(grams)	1.5	1.5	2.5
Highest ALT	349	1190	1324
Highest AST	296	1146	1856
Highest ALP	188	108	182
Highest total Bilirubin	99	229	286
Highest direct Bilirubin	72	204	237
Highest INR	1.5	1.9	2
Recovery duration(days)	10	16	40

Table 2: Showed some reports of Methylprednisolone induced acute liver injury, autoimmune hepatitis aggravated by methylprednisolone. IVMP: Intravenous Methylprednisolone; ALT: Alanine Transferase; AST: Aspartate Transferase; HC: Hepatocellular.

Author	Age/Ge r	Indi on	IVMP t dos	Time t onset	Symptoms	Concomitant medication	Total bilirub	ALT/A (IU/l	ALP/G (IU/l	Type liv	Antibodie	Time recov	Outco
Hofstee[9]	46/F	MS	1 (3)	6	-	None	-	1095/755	-/156	HC	None	16	Recovary
Oliveira[10]	33/F	MS	1 (-) + oral maintenan ce	-	Jaundice	Cyclophosphamide , Glatiramer Acetate	16	2308/710	92/247	HC	ANA (weakly +)	-	Recovary
D'Agnolo[11]	48/F	MS	1 (3)	3	Abdominal pain, nausea	None	29	3028/2384	- /182	HC	None	-	Recovary
Furutama[12]	11/F	MS	3 (3)	6	Low-grade fever, mild general fatigue	None	0.8	428/278	-/36	-	ANA (weakly +)	2	Recovary
Reuß[13]	42/F	MS	5 (-)	3	-	None	-	1082/485	-/170	HC	-	-	-
Bresteau[2]	35/F	MS	5 (5)	8	Asymptomatic	None	49	1512/778	86/109	HC	ANA	6	Recovary
Takahashi[14]	43F	MS	3 (3) + oral 50mg/day	4	Abdominal discomfort, nausea, vomiting	IFN-β	3.4	1067/1102	377/26	HC	ANA/ASMA	-	Recovary
Grilli[15]	35/F	MS	5 (5)	4	Jaundice	None	23.9	2000/1104	114/232	HC	-	11	Recovary
Davidov[16]	23/F	MS	3 (3)	3	Jaundice, malaise, abdominal pain	None	6.79	2011/1515	148/121	HC	ASMA	12	Recovary

Discussion

We presented a case of a young lady known to have multiple sclerosis who had three episodes of acute liver injury. All three episodes were preceded by IV methylprednisolone with a dose ranging from 1.5 to 2.5 gm. RECAM score which is the electronic version of RUCAM (Roussel Uclaf Causality Assessment Method) used to assess the likelihood of drug-induced liver injury was calculated in this case and the result was the association between MP and acute liver injury in this patient is highly likely or highly probable (score of 12).

With each attack of liver injury, the time from starting IV MP to onset of acute liver injury was decreasing and severity increases with prolonged recovery time (Table 1). The time to onset ranged from 3 to 6 weeks as was documented in previous case reports (Table 2). In all three attacks, the type of hepatic injury is hepatocellular, similar to what was reported in the literature [6].

The adverse reaction related to methylprednisolone in this case could be idiosyncratic as evident by latent period of 3-6 weeks. However, she received a higher dose of 2.5 gm before the last episode of acute liver injury which was more severe and more prolonged. Higher doses are associated with more severe hepatotoxicity as reported in a case series of methylprednisolone induced hepatotoxicity in Graves ophthalmopathy [7].

Kimura et al has reported eight cases of Methylprednisolone induced hepatotoxicity, all of them are female, with hepatocellular type of liver injury. Time to onset range from 30 to 60 days for 6 cases and only two cases the onset was 4 and 12 days [6].

A prospective observational single-center study on patients with MS treated with IV methylprednisolone 1,000 mg/day for 5 days to assess liver injury was carried out on 175 patients, showed that liver injury prevalence was 8.6%, 2.5% of them had severe liver injury [8].

Conclusion

Methylprednisolone induced liver injury has been reported. In patients who have indication for pulse methylprednisolone, liver function should be monitored carefully and for several weeks after the therapy is completed. As the patient who is required to have methylprednisolone, may need the treatment again in their lifetime, change to other steroid treatment or other alternative may be necessary to avoid severe hepatotoxicity that may be catastrophic.

Declarations

Disclosure: We Declare that no conflict of interest for any author of this case report.

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