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Ischemic hepatitis after percutaneous nephrolitotomy: A case report

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ABSTRACT

INTRODUCTION: Ischemic hepatitis (IH) is the necrosis of the centrilobular hepatocytes of liver and is secondary to liver hypoperfusion in most of the cases. The diagnosis is usually based on biochemical findings due to the absence of symptoms and signs. Although the disease course is often mild, and sometimes is even not diagnosed, the outcome is poor if the etiology of hypotension and liver anoxia is not promptly corrected.

PRESENTATION OF CASE: A 64-year-old patient who underwent percutaneous nephrolithotomy (PNL) for right renal pelvic stone developed acute IH at first postoperative day as a result of hemorrhage related severe hypotension. After restoring hemodynamic parameters, she completely recovered 2 weeks after the operation.

DISCUSSION: IH is a frequent cause of marked serum aminotransferase elevation and most commonly occurs as a result of arterial hypoxemia and insufficient hepatic perfusion. Although no specific treatment of IH exists, stabilizing the hemodynamic parameters of the patient resolves the problem in most of the cases.

CONCLUSION: This case is presented to demonstrate that ischemic hepatitis should be kept in mind if severe hemorrhage occurs during PNL.

1. Introduction

Ischemic hepatitis (IH) term was first used by Bynum et al.[1] to refer to the liver injury characterized by centrilobular necrosis resulting from the failure of hepatic blood perfusion. They proposed the term "hepatitis" because the disease was presented with the clinical and laboratory manifestations that suggested viral hepatitis.

Although "ischemic hepatitis" is accepted worldwide, the term "hypoxic liver injury" would better fit the situation as it has been shown in several studies that the liver is not ischemic but rather hypoxic and the histological evidence of inflammation is absent.[2,3]

Despite several pathophysiological alterations contribute to the development of the process (systemic hypotension, low cardiac output state, toxins, sepsis, and respiratory distress), the final common end-point is decreased oxygen levels for metabolic processes and hepatocellular dysfunction.[4]

The clinical features of IH resemble acute viral or toxic hepatitis. A sharp but transient increase in serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels to at least 20 times the upper limits of normal was usually accompanied by increase in serum lactate dehydrogenase (LDH) levels. However, serum bilirubin and alkaline phosphatase (ALP) levels are usually normal or only mildly elevated. The elevated serum aminotransferases usually reach their peak 1–3 days after the onset and then gradually decrease to normal. However, prolonged elevations are associated with increased mortality risk.[5]

2. Case report

A 64-year-old woman presented with right flank pain, dysuria and pollakuria. Non-contrast abdominopelvic CT revealed a 41–40 mm right renal pelvic stone. She had co-existing diabetes, hypertension and hypothyroidism, all under control with medications. Preoperative evaluation consisted complete blood count, serum urea, creatinine, ALT, AST and coagulation parameters together with urine culture.

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Table 1
Laboratory investigations before and after surgery.

Investigations	Preop.	P1	P2	P3	P4	P5	P6	P7	P8	P13
LDH[a](IU)	185	2117	1757	1061	540	498	466	397	206	178
AST[b] (IU)	18	100	616	911	873	964	321	104	68	19
ALT[c] (IU)	17	83	392	878	1158	939	627	347	207	21
Cre[d] (mg/dL)	0.87	1.18	1.41	1.49	0.95	0.99	0.86			

[a]Lactic dehydrogenase.
[b]Aspartate aminotransferase.
[c]Alanine aminotransferase.
[d]Creatinine.

She underwent right PNL in prone position under general anesthesia. PNL was started in lithotomy position and a ureteral catheter was placed. After conversion of the patient to prone position, the collecting system was visualized with contrast and a posterior middle calyx puncture was performed between 11th and 12th ribs. After an uncomplicated procedure, a 16 Fr nephrostomy tube was placed at the end of the case. The initial 13 g/dl hemoglobin level slightly decreased to 11 g/dl at the end of the procedure. Because of the respiratory distress encountered during the recovery period, the patient was referred to intensive care unit. Just after 45 min, the patient experienced sudden and severe hypotension with 900cc hemorrhagic fluid in the drainage tube. Control hemoglobin was 8.1 g/dl and the postoperative thoraco-abdomino-pelvic tomography did not reveal any hematoma or adjacent organ injury. The nephrostomy tube was clamped and blood transfusion was initiated. After two units of erythrocyte suspension and a single unit of fresh frozen plasma transfusion, hemoglobin levels increased up to 10 g/dl. After the stabilization of respiratory and hemodynamic parameters, she was transferred to urology clinic.

The serum aminotransferases and LDH started to rise at the end of the first postoperative day reached to peak value after 96 h (AST > 20 times the upper limit of normal (ULN), ALT > 25 times ULN and LDH > 10 times ULN). All parameters were normalized within 2 weeks (Table 1). She was discharged from the hospital on postoperative 9th day.

3. Discussion

IH is liver necrosis, primarily of the centrilobular hepatocytes and it is secondary to hypoperfusion in most of the cases.[6] Unlike, hepatitis which is the inflammation of liver, the cells are damaged and cell death occurs. The diagnosis is usually based on biochemical findings due to the absence of symptoms and signs.[7] IH is generally defined in the literature by significantly elevated, transient (5–25 days) serum aminotransferases levels (greater than eight times the upper normal level) with or without LDH elevation, histological evidence of centrilobular necrosis, rapid resolution of pathology (within 7–10 days) and exclusion of other hepatic pathologies.[8]

The mainspring of IH is insufficient hepatic perfusion.[6] The most frequent cause of IH is left-sided heart failure because of coronary artery disease and/or cardiomyopathy when hypotension or reduced heart output develops.[7] Serum transaminases increase due to hepatic damage and serum lactate can also be elevated due to inadequate removal by the dysfunctional liver.[9] The prevalence of IH among admissions to hospital is around 1/1000. In intensive care units, the prevalence is 10 times higher and accounts for at least 1% of admissions.[10]

The outcome of the disease is poor if the cause of hypotension and liver anoxia is not corrected. The disease is often mild, and sometimes is not even diagnosed.[11]

Therapy of underlying diseases is, by far, the main part of the treatment of IH. Therapeutic goals are to increase oxygen delivery and to ease oxygen exchanges between blood and liver cells. There exists no specific treatment for IH and restoring systemic hemodynamics remains the main target.[10]

In present case while patient had no abdominal or other findings serum transaminases and LDH was started to elevate 24 h after systemic hypotension, while serum billuribin and international normalized ratio (INR) levels were normal. The patient was consulted to internal medicine and established the diagnosis of IH. The liver enzymes LDH, AST and ALT started to decrease on 2nd, 6th and 5th day respectively and normalized on 12th day. These findings were consistent with literature.

To the best of our knowledge our case is the first reported case after PNL. Patients undergoing PNL are at risk for significant intraoperative bleeding and subsequent blood transfusion. In this case, cause of IH was systemic hypotension secondary to hemorrhage after PNL.

In our case, the patient was clinically diagnosed to ischemic hepatitis and there was also massive bleeding due to PNL. Hemorrhage and hypotension could have contributed to ischemic hepatitis.

4. Conclusion

The mainspring of IH is insufficient hepatic perfusion. In this case, insufficient hepatic perfusion and IH caused by hypotension associated with hemorrhage. We conclude that routine serum biochemical parameters should evaluate after PNL if there is a suspicion of hemorrhage.

Conflict of interest

We have no financial and personal relationships with other people or organisations that could inappropriately influence(bias) this submission.

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Ethical approval

We have written and signed consent to publish the case report from the patient.

Author contributions

Mustafa Zafer Temiz was the major contributor in writing the case report and he was involved in analysis and interpretation of the data, and provided clinical care of the patient during her treatment. Emrah Yuruk and Engin Kandirali supervised the writing of the case report and were involved in the review and preparation of the manuscript. Kutlu Teberik, Burcu Kadriye Akbas, Mustafa Devrim Piroglu and Hande Selvi Oztorun were provided clinical care of the patient during her treatment. All authors read and approved the final manuscript for submission.

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