

CASE REPORTS

Liver infarction: To treat or not to treat?

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ABSTRACT

Liver infarctions are a very rare occurrence due to the multiple-source blood supply of the liver. The cause of hepatic infarction is unclear. When diagnosed, appropriate treatment of these infarctions is unknown. Herein we describe a case of acute liver infarctions, with no obvious cause, in a young woman. Work-up did not shed light on the underlying etiology. Treatment with anticoagulation was commenced in this case, with rapid improvement and a good clinical outcome. We suggest that anticoagulation can be administered to treat hepatic infarctions even in the absence of clear-cut thrombosis.

Key Words: Hepatic infarction, Liver infarction, Anticoagulation

1. INTRODUCTION

There are few descriptions of liver infarctions in the medical literature, and thus the mechanisms that bring about these infarctions are not fully known. Infarctions stemming from vascular abnormalities secondary to trauma or a surgical procedure, inflammatory processes, and the antiphospholipid syndrome have been described, though other factors may contribute in some cases. The ideal treatment approach is ambiguous, leaving the clinician with the dilemma of watchful waiting versus anticoagulant therapy, with the hope of improving hepatic blood flow with the latter option.

Herein we present a case of liver infarction of unknown etiology that was treated with anticoagulation, culminating in a good clinical outcome.

2. CASE PRESENTATION

A 26-year-old healthy young woman presented to the emergency department due to severe epigastric pain of a few hours

duration. History was positive for smoking and oral contraceptive use for the past five years. It should be noted that she had undergone a laparoscopic sleeve-gastrectomy two and a half months earlier, after which time she lost twenty kilograms. There were no symptoms of infectious disease.

Pre-operative abdominal ultrasound was normal. Peripheral blood tests were remarkable for slight leukocytosis with neutrophilia, elevated hepatocellular enzymes, and the absence of anemia or thrombocytopenia.

Abdominal ultrasonography demonstrated three new echogenic geographic lesions (see Figure 1).

Computed tomography revealed multiple peripheral wedge-shaped hypodensities consistent with hepatic infarctions, as demonstrated in Figure 2. No anatomic abnormalities or vessel occlusions were noted.

Clotting studies were normal. C-reactive protein was within normal range. Hypercoagulability testing – including pro-

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teins C and S, prothrombin mutation, activated protein C resistance, factor 8, homocysteine and thrombin time – were normal. Lupus anticoagulant studies were also normal, as were antiphospholipid antibodies. Pathergy testing for Bechet's disease was negative. Ocular funduscopy, performed to rule out uveitis, was unremarkable. Immune serologies, including anti-neutrophil cytoplasmic antibody studies, C3, C4 and anti-nuclear antibodies, were negative. Protein immune-electrophoresis showed no evidence of monoclonal gammopathy. Echocardiogram did not reveal ventricular dysfunction nor was there evidence of an intracardiac thrombus. Continuous monitoring was negative for arrhythmia.



Figure 1. Abdominal ultrasound demonstrating three echogenic lesions suspicious of infarction



Figure 2. CT scan demonstrating multiple peripheral wedge-shaped hypodensities consistent with hepatic infarctions

Anticoagulant therapy was chosen as the treatment strategy since no obvious treatable cause was found. Low molecular weight heparin (Enoxaparin) was initiated followed by a vitamin K antagonist (Warfarin). Swift resolution of the abdominal pain was noted within a few days, and elevated liver enzymes completely normalized. The patient continued treatment for six months thereafter. No additional symptoms pointing at a specific inflammatory disease developed during this time. On follow-up, the patient remains in good health, without clinical sequelae two years later.

3. DISCUSSION

Liver infarction is a rare occurrence, most likely due to the multiple-source blood supply of the liver through both the hepatic arteries and the portal vein. When these infarctions do occur, they are usually caused by surgical, traumatic, or septic etiologies.^[1-3] They have also been described in the medical literature as being secondary to vasculitis – specifically polyarteritis nodosa – via hepatic vessel inflammation and occlusion.^[1,4] Haratake et al. described a case of hepatic infarction diagnosed during the autopsy of a 44-year-old man suffering from polyarteritis nodosa. In this case, a thrombus was found in the right portal vein. This situation has also been described in a pregnant patient who was later diagnosed as suffering from ulcerative colitis.^[5] In addition, liver infarction has also been reported in hepatocellular enzymes, in the antiphospholipid antibody syndrome during pregnancy, in a hemodialysis patient with systemic lupus erythematosus, and in one additional non-pregnant patient.^[6-11] One case of embolization from an atherosclerotic lesion has also been reported.^[12] Although it seems like a logical conclusion, an etiological connection with thrombophilia – aside from the rare occurrence in antiphospholipid antibody syndrome – has not been reported.

Time-wise, a surgical etiology causing multiple infarctions in our patient seemed unlikely, since two and a half unremarkable months had elapsed since she had undergone the bariatric procedure. The onset of abdominal pain was sudden, without previous discomfort. No vascular anomaly was noted in this case, and extensive blood testing did not reveal any explanation.

Currently, there is no data concerning the appropriate management of these cases. Cases, for the most part, are treated according to etiology. In antiphospholipid antibody syndrome, treatment with anticoagulation is relevant since a thrombotic cause is obvious. In cases where an inflammatory cause is suspected, there may be a role for anti-inflammatory drugs, although no data regarding this issue has been published to date. The role of anticoagulant therapy, when there is no clear thrombotic etiology, is unknown.

We hypothesize that the combination of smoking, contraceptives, and obesity contributed to liver infarction in the case of our patient. We chose to treat the patient with anticoagulation, which was followed by swift clinical resolution.

4. CONCLUSION

Anticoagulant therapy can be considered in the treatment of liver infarction of unknown etiology.

CONFLICTS OF INTEREST DISCLOSURE

The authors declare no conflicts of interest.

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