

ABSTRACT

Background: Liver cirrhosis is among the most common causes of death in the United States. Cirrhosis can result from alcoholic liver disease, hepatitis, or non-alcoholic fatty liver disease. Advanced cases of liver cirrhosis may result in complications such as portal hypertension, hepatosplenomegaly, varices, and many others.

Case Presentation: This report describes a case of extensive liver cirrhosis found during cadaveric dissection of a 71-year-old male. Observation revealed a slightly enlarged, cirrhotic liver with recanalization of the umbilical vein (of the round ligament). The patient also had significant splenomegaly, indicative of portal hypertension, and dilation of the left ureter. In developing countries, the leading cause of liver cirrhosis is schistosomiasis. Schistosomiasis has been shown to cause both liver cirrhosis and hydroureter (Genitourinary schistosomiasis, 2012). While this infection cannot be completely ruled out, the likelihood that this was the case in this cadaver is unlikely.

Discussion: Non-invasive and cost-effective options such as serum and imaging tests can prove useful in detecting liver pathology. Detection of early liver disease and intervention can decrease the incidence of advanced complications and prolong life (Smith et al. 2019). In the case of hydroureter, a parasitic infection can be ruled out by laboratory analysis of stool or urine samples by detecting the presence of parasitic eggs. Histological specimens of the liver, ureter and urinary bladder can be taken to determine if parasitic eggs were present in either of these areas.

Conclusion: This cadaver reveals a classic presentation of portal hypertension that can lead to various secondary pathologies. This case study can be used as additional supporting evidence linking portal hypertension to splenomegaly, hydroureter and liver cirrhosis.

INTRODUCTION

Liver cirrhosis is a disease that results in scarring and damage to the liver that can manifest itself from a variety of causes. In the U.S., the leading causes of liver cirrhosis are alcohol use, hepatitis and non-alcohol related fatty liver disease (Cleveland Clinic, 2020). In non-western societies, a common cause of liver cirrhosis is Schistosomiasis. *Schistosoma spp.* is a larval fluke that uses snails as intermediate hosts and can transfer to humans in freshwater bodies such as rivers, lakes, ponds, et cetera. *Schistosoma spp.* is most prevalent in the Southeast Asia region (Nelwan, 2019). If left untreated, chronic schistosomiasis infection can lead to an increased risk of liver fibrosis and bladder cancer (CDC, 2019).

MATERIALS AND METHODS

During routine cadaveric dissection of the abdomen in the anatomy laboratory, various anomalies were found in one of the cadavers. Our cadaver was a 71-year-old Caucasian male that worked as a contractor in Savannah, GA, for most of his life. His cause of death was determined to be liver cirrhosis.

Tissue samples (1 cm²) were collected from the following organs:

Anterior right (quadrant V) and left (quadrant III) lobes of the liver
Left kidney
Left ureter
Spleen
Superior and medial portion of the bladder.

Samples were placed in a solution containing a 3:1 ratio of water to Maryland State Blend (embalming fluid), then transported to Colquitt Regional Hospital for detailed histological processing and staining.

RESULTS

The following anatomical variations were found in our cadaver: liver cirrhosis (Figure 1a), splenomegaly (Figure 1b), pyonephrosis of the left kidney (Figure 1c), a left hydroureter (Figure 1d), and calcifications on the superior aspect of the bladder (Figure 1e). Biopsy samples of our cadaver revealed the presence of Schistosome larvae eggs upon histopathological examination. Histopathological examination of the liver revealed the classic presentation of liver cirrhosis in which there were fibrotic bands surrounding regenerating hepatocytes. These fibrotic bands can be seen in Figure 2. Multiple calcified *Schistosoma mansoni* eggs were confirmed in the liver with its notable lateral spine as seen in Figure 3. Some areas of the liver showed malignant changes, which could be caused by both liver cirrhosis and schistosomiasis. The malignant hepatocytes appear to be darker stained, as seen in Figure 4. Nuclear atypia and pleomorphism were noted in the hepatocellular carcinoma tumor cells. Figure 5 demonstrates non-malignant hepatocytes (Figure 5a) as compared to malignant liver tissue (Figure 5b), both seen in the cadaver. Examination of the bladder showed an increased amount of granulomas, lymphocytes, macrophages, and fibrotic tissue which may be the inflammatory response associated with the parasitic infection. The superior wall of the bladder was noted to be much tougher, which is likely related to the increased amounts of granulomatous tissue. There was also a non-calcified *S. mansoni* ovum identified in the bladder as seen in Figure 6. Samples taken from the left ureter did not show any significant pathology. Examination of the other collected biopsies showed no other abnormalities apart from the classic presentation of portal hypertension and liver cirrhosis.



Figure 1. Gross anatomical specimens showing (a) liver with fibrosis and nodules diagnostic for cirrhosis, (b) enlarged spleen, (c) left kidney with pyogenic abscess, (d) dilated left ureter, and (e) hemisected bladder with hard nodule (tip of forceps).

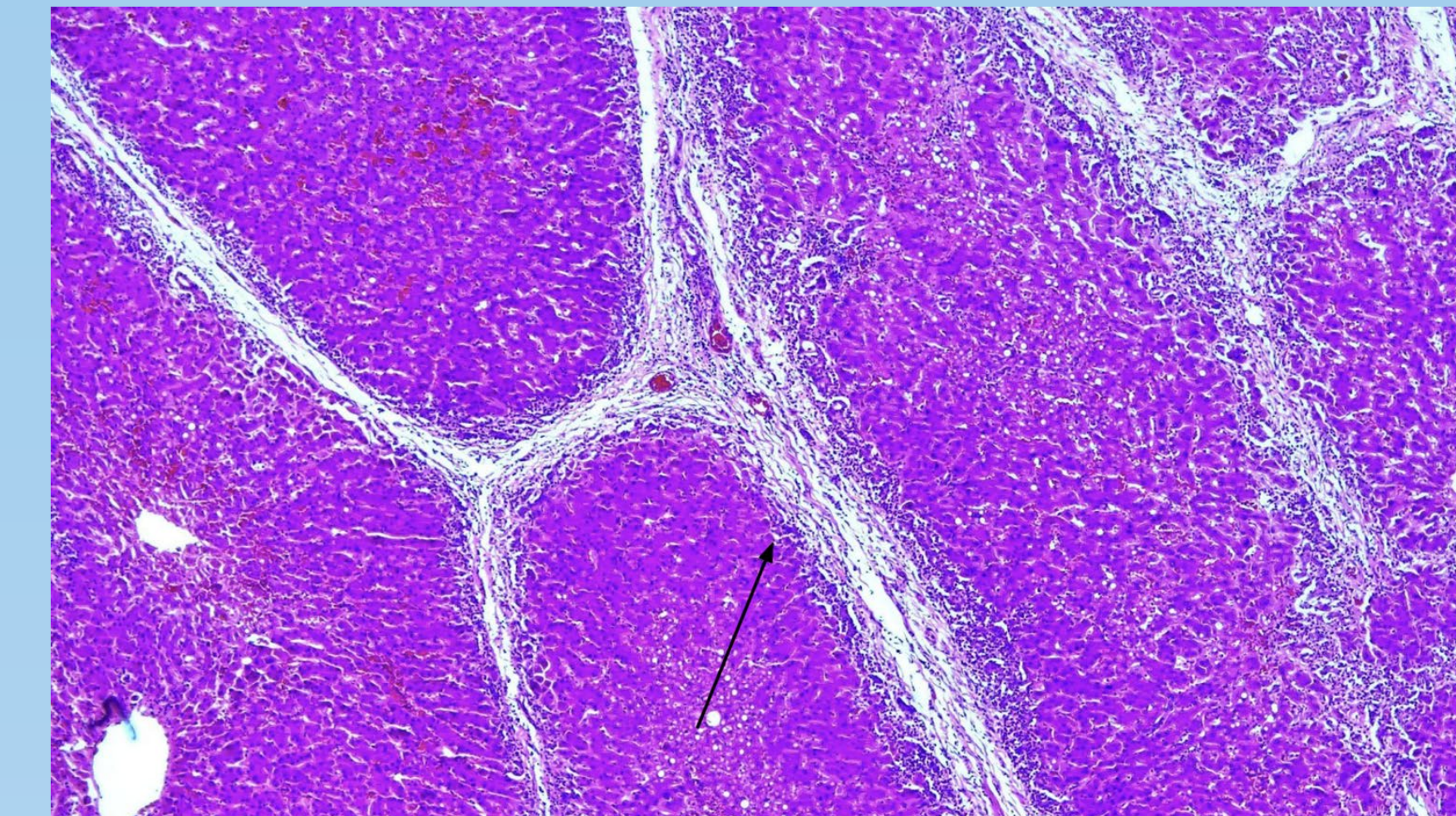


Figure 2. Liver section showing cirrhotic liver lobules and fibrotic bands (arrow).

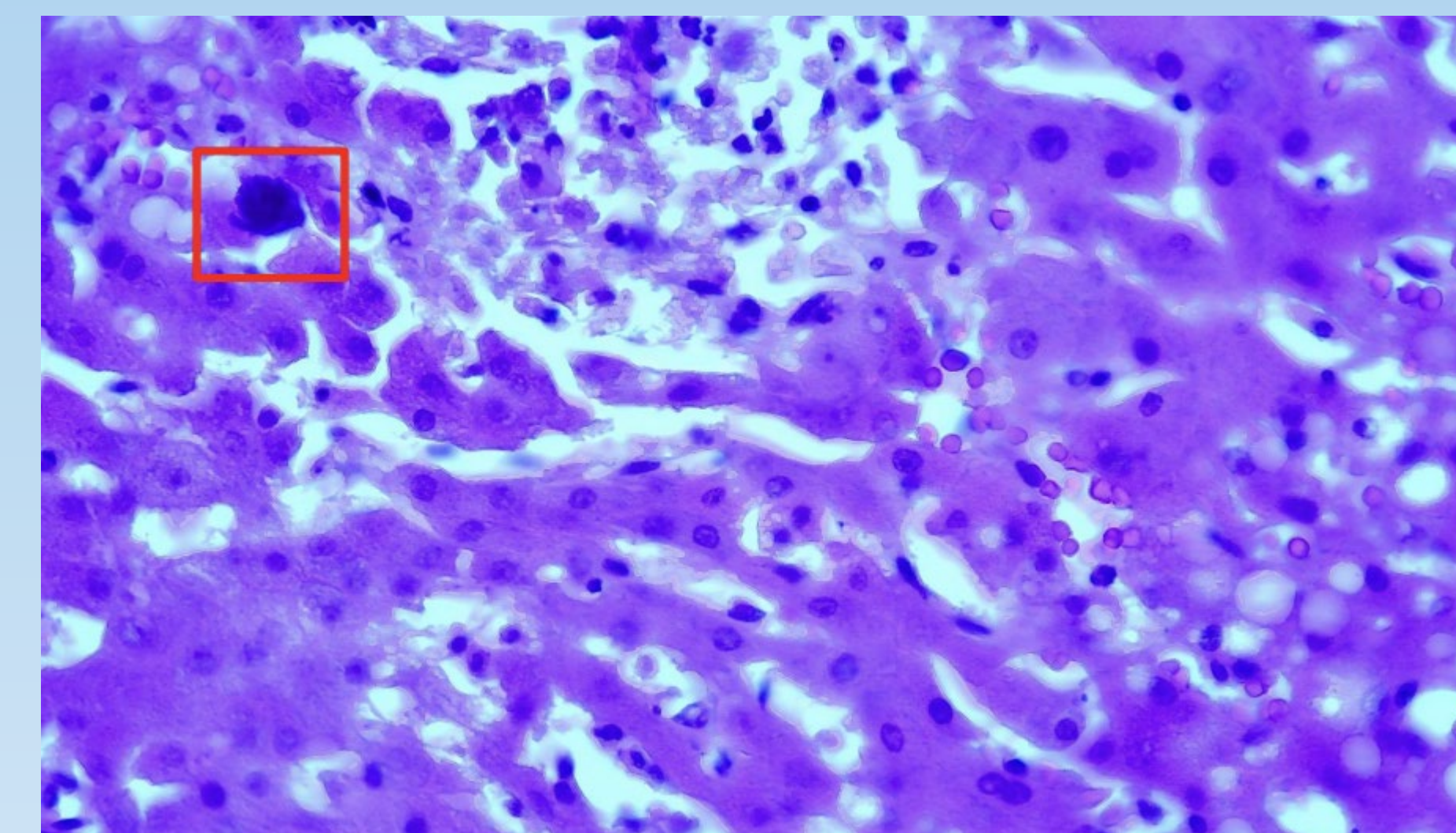


Figure 3. Liver section showing the large lateral spine diagnostic of *Schistosoma mansoni* (box).

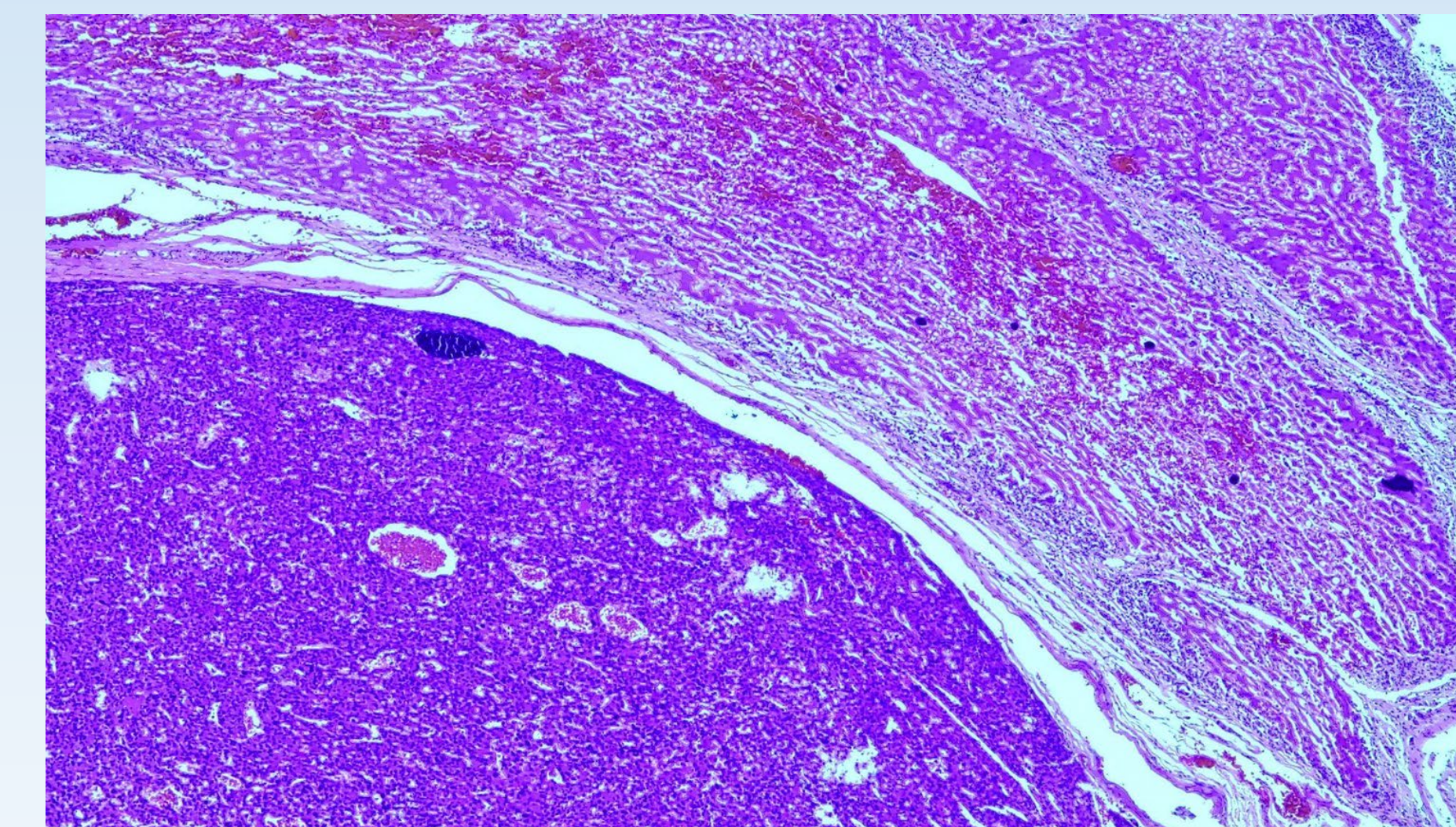


Figure 4. Liver section showing the separation between non-malignant liver tissue (above) and the darker-staining malignant liver tissue (below).

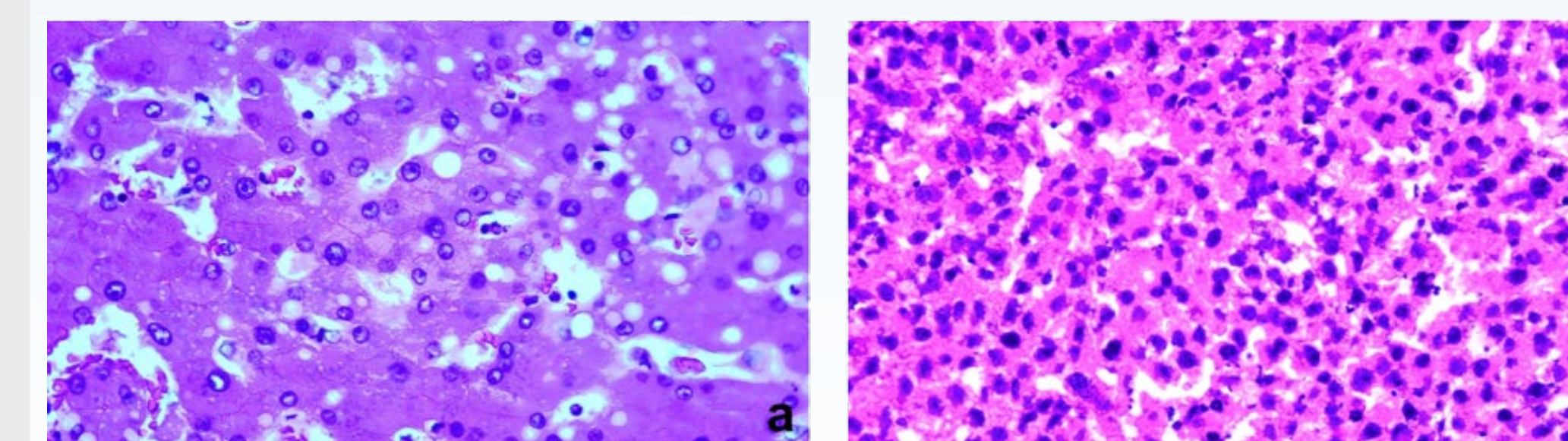


Figure 5. Liver sections comparing non-neoplastic liver tissue (a) to neoplastic liver tissue (b).

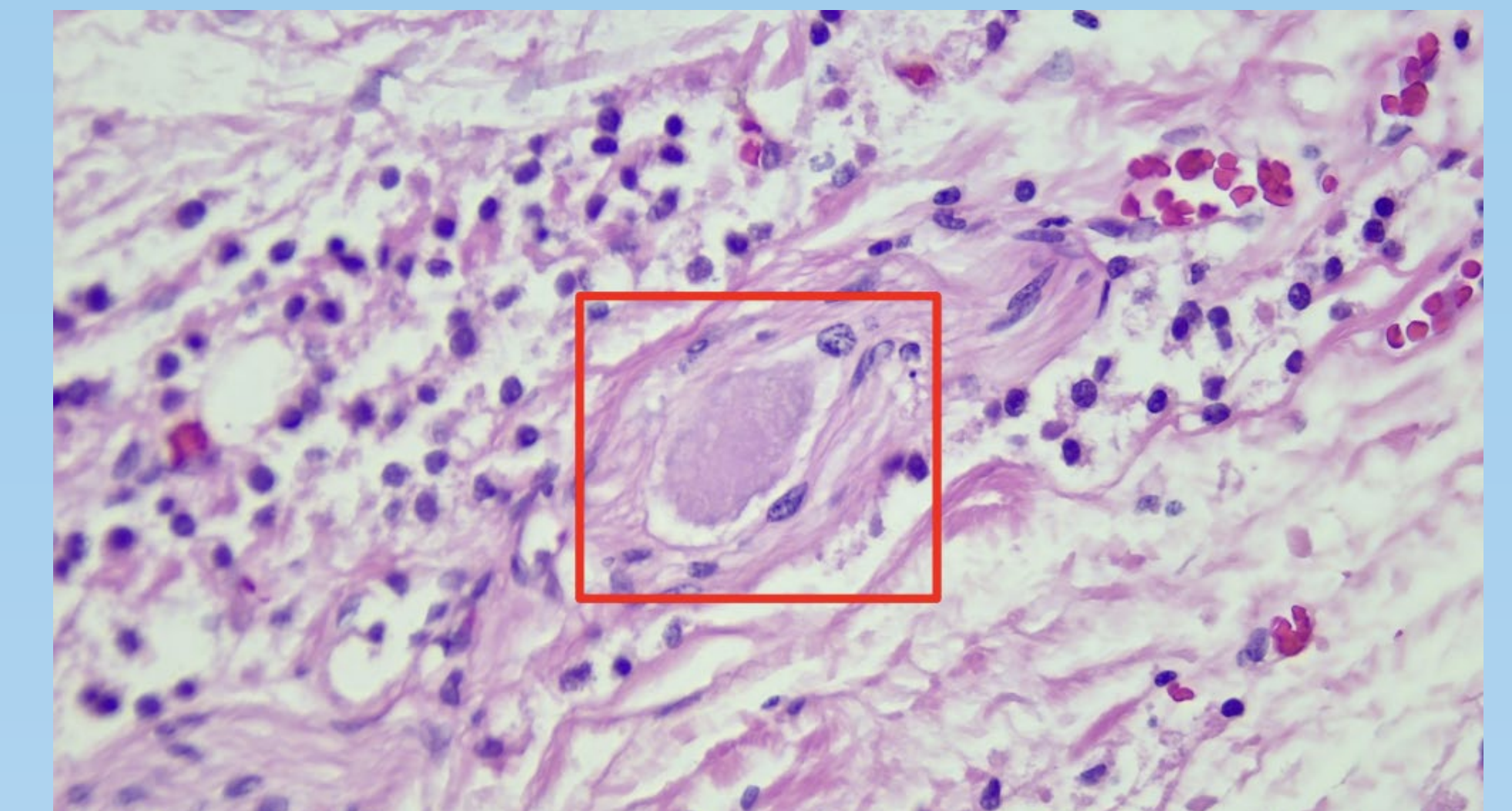


Figure 6. Bladder section showing ovum of *Schistosoma mansoni*.

CONCLUSION

Due to poor screening methods for *Schistosoma* in the U.S., it is doubtful that physicians associated the larvae with liver cirrhosis. The patient exhibited the characteristic signs of portal hypertension that accompany liver cirrhosis, including a large fibrotic liver and an enlarged spleen. *Schistosoma*-associated liver cirrhosis cases may display, in addition to these findings, squamous cell hepatocellular carcinoma and implantation of larvae in the hepatobiliary tracts (Shaker, et al., 2014).

Histological samples of the cadaver confirmed the presence of *Schistosoma mansoni* ova in both liver and urinary bladder biopsies. This cadaver reveals a classic presentation of portal hypertension that can lead to various secondary pathologies including splenomegaly, hydroureter, and liver cirrhosis as observed in this patient. This case study will aid in the diagnosis and treatment of portal hypertension caused by infection with schistosomiasis, and possibly prevent secondary pathologies due to chronic schistosomiasis infection. Our results further characterize this parasitic infection, supporting the limited literature investigating this parasite.

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