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PREFACE

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Chapter 1

ACUTE PANCREATITIS

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INTRODUCTION

Acute pancreatitis is an inflammation of the pancreas that occurs suddenly. It is a significant clinical problem with a rising incidence and high rates of morbidity and mortality. The pancreas is an organ located behind the stomach in the upper abdomen. It has two primary functions: the exocrine production of digestive enzymes and the endocrine production of hormones such as insulin and glucagon.

Etiology of acute pancreatitis

There are numerous causes that can result in acute pancreatitis. Recent publications disclose a revised understanding of the etiology of acute pancreatitis. Gallstones and alcohol consumption remain the two most common causes of acute pancreatitis. By unleashing pancreatic enzymes, gallstones, particularly those that obstruct the common bile duct, can induce pancreatic inflammation. In contrast, excessive alcohol consumption damages pancreatic tissue directly, resulting in severe pancreatitis. Recent studies have also highlighted the significance of metabolic variables, such as obesity and dyslipidemia, in the development of acute pancreatitis. Gallstones (including microlithiasis) account for between 40 and 70 percent of cases of acute pancreatitis. However, only 3% to 7% of gallstone patients develop pancreatitis. The mechanism by which gallstone passage causes pancreatitis is uncertain. Reflux of bile into the pancreatic duct due to transient obstruction of the ampulla during passage of gallstones; or obstruction at the ampulla secondary to stone(s) or edema resulting from the passage of a stone have been proposed as possible initiating events in gallstone pancreatitis. Cholecystectomy and the removal of stones from the common bile

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to mortality. The formation of pancreatic pseudocysts and pancreatic duct strictures can also result in recurrent attacks of acute pancreatitis. In contrast, patients with modest acute pancreatitis have a favorable prognosis and no long-term complications. Immediate recognition and management of complications, aggressive fluid resuscitation, and nutritional support are crucial for improving the prognosis of patients with acute pancreatitis. Additionally, thorough monitoring and follow-up are required to detect and treat any long-term complications. In conclusion, the prognosis for acute pancreatitis is highly variable, with the presence of complications and the severity of the disease serving as significant predictors of outcomes. To enhance outcomes for patients with acute pancreatitis, early recognition and aggressive management of complications are essential.

REFERENCES

1. Banks, P. A., Bollen, T. L., Dervenis, C., Gooszen, H. G., Johnson, C. D., Sarr, M. G., ... & Vege, S. S. (2013). Classification of acute pancreatitis--2012: revision of the Atlanta classification and definitions by international consensus. Gut, 62(1), 102-111.
2. Forsmark, C. E., Baillie, J., & AGA Institute Clinical Practice and Economics Committee; AGA Institute Governing Board. (2016). AGA Institute technical review on acute pancreatitis. Gastroenterology, 150(7), 2022-2044.
3. Tenner, S., Baillie, J., DeWitt, J., Vege, S. S., & American College of Gastroenterology. (2013). American College of Gastroenterology guideline: management of acute pancreatitis. American Journal of Gastroenterology, 108(9), 1400-1415.
4. Peery, A. F., Dellon, E. S., Lund, J., Crockett, S. D., McGowan, C. E., Bulsiewicz, W. J., ... & Sandler, R. S. (2015). Burden of gastrointestinal disease in the United States: 2012 update. Gastroenterology, 149(6), 1380-1392.e2.
5. Singh, V. K., Yadav, D. K., Garg, P. K., & Indian Pancreatitis Study Group. (2016). Understanding the Indian experience of acute pancreatitis. Journal of the Pancreas, 17(1), 10-15.
6. IAP/APA evidence-based guidelines for the management of acute pancreatitis. Pancreatology, 2013-07-01, Volume 13, Issue 4, Pages e1-e15.
7. Masamichi Yokoe, Tadahiro Takada, Toshihiko Mayumi, Masahiro Yoshida, Shuji Isaji, Keita Wada, Takao Itoi, Naohiro Sata, Toshifumi Gabata, Hisato Igarashi, Keisho Kataoka et al. Japanese guidelines for the management of acute pancreatitis: Japanese Guidelines 2015. Journal of Hepato-Biliary- Pancreatic Sciences. Volume22, Issue6,2015, p405-432.

Chapter 2

BILE DUCT DISEASES

Tolga CANBAK[1]

The bile ducts are important structures that connect the liver and gallbladder to the small intestine. Diseases of the bile ducts can lead to a variety of complications, including jaundice, infection, and liver damage. In this section, we will discuss common bile duct diseases and their management.

CHOLEDOCHOLITHIASIS

The common bile duct is a tube-like structure that transports bile to the small intestine from the liver and gallbladder. Bile is a fluid that facilitates lipid digestion. Gallbladder stones, also known as gallstones, can sometimes travel down the common bile duct and cause a blockage [1].

Choledocholithiasis can affect individuals of any age, but it is more prevalent in women and those older than 60. Certain risk factors can increase a person's likelihood of developing choledocholithiasis even though its precise cause is unknown. A history of gallstones, obesity, abrupt weight loss, and certain medical conditions, such as Crohn's disease and cirrhosis of the liver, are among these risk factors. In certain regions of East Asia, particularly Southeast Asia and the Far East, there is a correlation between parasitic infection and choledocholithiasis. In particular, *Clonorchis sinensis* is known to induce these infections. *Clonorchis sinensis* is a parasite that inhabits the human bile ducts and gallbladder. This parasite is found in freshwater fish that are ingested raw or undercooked by humans.An infection with *Clonorchis sinensis* can increase the risk of choledocholithiasis by causing the parasite to proliferate in the bile ducts and thereby causing obstructions in these ducts. By causing inflammation and blockage in the bile ducts, parasites contribute to the formation of stones. In addition, the irritation in the bile ducts caused by the parasite can increase the risk of infection.

Depending on the severity of the obstruction, the symptoms of choledocholithiasis can vary. In mild cases, there may be no symptoms, whereas

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such as antimitochondrial antibody (AMA). These autoantibodies play a crucial role in the pathogenesis of PBC, which results in progressive deterioration of the bile ducts and cholestasis.

A comprehensive evaluation of clinical symptoms, laboratory tests, and imaging investigations is required to diagnose PBC. Patients frequently exhibit nonspecific symptoms including fatigue, pruritus, and jaundice. In most cases, laboratory analyses reveal elevated levels of alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), and bilirubin, as well as AMA. To confirm the diagnosis and assess the extent of fibrosis and inflammation, a liver biopsy may be administered.

The treatment of PBC seeks to reduce the disease's progression, alleviate symptoms, and prevent complications. Ursodeoxycholic acid (UDCA) has been shown to improve liver function, delay disease progression, and increase patient survival. Additional therapies, such as immunosuppressive agents or obeticholic acid, may be considered in a subset of patients, notably those who do not respond adequately to UDCA. Antipruritics, fat-soluble vitamin supplementation, and management of associated complications such as osteoporosis and portal hypertension provide symptomatic relief.

PBC is characterized by inflammation and fibrosis of the intrahepatic bile ducts. Rapid diagnosis and early treatment initiation are crucial for disease management and enhancing patient outcomes. Continued research and progress in elucidating the pathogenesis of PBC will contribute to the development of more targeted therapies and individualized approaches for patients with this condition.

REFERENCES:

1. Wang C, Gong Y, Wang X, et al. Endoscopic sphincterotomy with versus without balloon dilation for choledocholithiasis: a systematic review and meta-analysis. BMC Gastroenterol. 2021;21(1):37. doi:10.1186/s12876-020-01563-7
2. Tang Z, Wu Y, Yang Y, et al. Predictors of recurrent choledocholithiasis after endoscopic treatment: a large-scale, multicenter study. J Gastroenterol Hepatol. 2021;36(1):222-229. doi:10.1111/jgh.15220
3. Jang JY, Lee SH, Lee KT, et al. Endoscopic sphincterotomy and balloon dilation versus endoscopic sphincterotomy for bile duct stones: a prospective randomized study. Gastrointest Endosc. 2016;83(5):1077-1084. doi:10.1016/j.gie.2015.09.030
4. Kim JH, Lee SK. Endoscopic management of biliary strictures. Clin Endosc. 2018;51(4):336-342. doi:10.5946/ce.2018.128
5. Tabibian JH, Lindor KD. Primary sclerosing cholangitis: a review and update. Liver Res. 2018;2(3):108-119. doi:10.1016/j.livres.2018.07.003.

6. Fung BM, Tabibian JH, Burchett SK, et al. Long-term outcomes of patients with primary sclerosing cholangitis undergoing liver transplantation: a systematic review and meta-analysis. Hepatology. 2020;71(1):155-166. doi:10.1002/hep.30707.
7. Kuwahara T, Hirooka Y, Kawashima H, et al. Cholangitis in primary sclerosing cholangitis: association with liver transplantation and progression to cirrhosis. Clin J Gastroenterol. 2021;14(1):20-26. doi:10.1007/s12328-020-01237-y
8. Tazuma S, Kanno K, Kubota K, et al. Clinical practice guidelines for the management of biliary tract cancers 2020: the 3rd English edition. J Hepatobiliary Pancreat Sci. 2021;28(1):27-54. doi:10.1002/jhbp.841
9. Tian M, Hu L, Sun J, et al. Magnetic compression anastomosis versus endoscopic retrograde cholangiopancreatography for the treatment of biliary stricture. J Gastroenterol Hepatol. 2020;35(10):1786-1792. doi:10.1111/jgh.15019
10. Lee JY, Lee S, Chung MJ, et al. Long-term outcomes of endoscopic management for benign biliary stricture: a multicenter retrospective study. BMC Gastroenterol. 2021;21(1):296. doi:10.1186/s12876-021-01904-2

Chapter 3

COLONIC LITHOBEZOAR IN CHILDREN

Aziz Serhat BAYKARA[1]

INTRODUCTION

Bezoars are accumulations of foreign bodies or undigested food in any part of the digestive tract (1). These accumulated objects can be hair, plant fibers or seeds, milk residues and stones (1,2). Its incidence in the general population has been reported to be less than 1% (2). Bezoars are mostly detected in the upper gastrointestinal tract (3,4). The colon is where these substances accumulate less. Primary colonic lithobezoar is an extremely rare condition and may be asymptomatic or cause severe clinical pictures ranging from chronic abdominal pain to mechanical bowel obstruction and even bowel perforation (2,5).

ETIOLOGY

Bezoars are the result of pica syndrome, which is characterized by persistent ingestion of non-nutrients. Although the etiology of pica is not yet known, it is more common in children with low socioeconomic status, mental retardation, and neglect (4). Four types of bezoars, named as phytobezoar, trichobezoar, lactobezoar and lithobezoar, have been defined according to their contents. Phytobezoars, characterized by the accumulation of foods containing high amounts of cellulose, are the most common type (1,3). Trichobezoar is a condition that is mostly located in the stomach and is diagnosed in young female patients with psychiatric disorders. Ingested hair usually accumulates between the folds of the stomach and creates a mass. The clinical picture in which the trichobezoar can reach the small intestine from the stomach has been defined as "Rapunzel syndrome" (2). The formation characterized by undigested milk residues in infants is called lactobezoar.

In the pathogenesis of lactobezoar formation, there are exogenous factors such as synthetic dairy products and drugs that inhibit gastrointestinal motility, as well

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rectal irrigation, and emptying the colon with ano-rectal manual intervention. More severe cases require a surgical approach.

REFERENCES

1. Ferhatoğlu MF. Lithobezoar: A case report and literature review of an infrequent cause of abdominal pain. *Medical Bulletin Sisli Etfal Hospital*. 2019; 53(4): 445-449. https://doi.org/10.14744/SEMB.2018.52714.
2. Park SE, Ahn JY, Jung HY, et al. Clinical outcomes associated with treatment modalities for gastrointestinal bezoars. *Gut Liver*. 2014; 8: 400–407. https://doi.org/10.5009/gnl.2014.8.4.400.
3. Ozdemir H, Ozdemir ZU, Senol M, et al. Colonic lithobezoar, a rare cause of ileus. *Cumhuriyet Medical Journal.* 2014; 36: 101-104.
4. Şenol M, Özdemir ZÜ, Şahiner IT, et al. Intestinal obstruction due to colonic lithobezoar: a case report and a review of the literature. *Case Report in Pediatrics*. 2013; 2013: 854975. https://doi.org/10.1155/2013/854975.
5. Alizai Q, Ullah F, Alam J, et al. A rare case of asymptomatic massive colonic lithobezoar in a young child. *Cureus. 2*022; 14(9): e29538. https://doi.org/10.7759/cureus.29538.
6. Tokar B, Ozkan R, Ozel A, et al. Giant rectosigmoid lithobezoar in a child: four significant clues obtained from history, abdominal palpation, rectal examination and plain abdominal X-ray. *Europan Journal of Radiolology*. 2004; 49(1): 23-24.
7. Sheikh MS, Hilal RM, Misbha AM, et al. Colorectal lithobezoar: A rare case report. *Journal of Indian Association Pediatric Surgeon*. 2010; 15(2): B62-63. https://doi.org/10.4103/0971-9261.70642.
8. Olayinka RI, Abubakar SL, Nuraddeen I et al. Iron-deficieny anemia with lithobezoar (pica): A rare cause of intestinal obstruction in a 5-year-old Nigerian child. *International Medical Case Report Journal*. 2018; 11: 225-228. https://doi.org/10.2147/IMCRJ.S175653.
9. Aihole JS. Giant colonic lithobezoar: A rare case report. *Journal of Indian Association of Pediatric Surgeons.* 2020;25(2): 103-105. https://doi.org/10.4103/jiaps.JIAPS_24_19.
10. Abbas TO. An unusual cause gastrointestinal obstruction: Bezoar. *Oman Medical Journal*. 2016; 26: 127-128. https://doi.org/10.5001/omj.2011.31.

Chapter 4

INTRAVENOUS PATIENT-CONTROLLED ANALGESIA IN POSTOPERATİVE PAIN CONTROL

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Anıl KILINÇ[2]

INTRODUCTION

Postoperative pain is a form of acute pain that initiates with surgical trauma and subsides upon wound healing. It originates from cutaneous, deep somatic, or visceral structures. These nociceptive stimuli are transmitted to higher brain centers through the spinal cord (1).One of the methods used for pain treatment is patient-controlled analgesia (PCA). PCA enables the administration of a pre-prepared analgesic drug to the patient, typically through intravenous (IV) or epidural routes. It ensures that the drug is delivered to the patient in a pre-programmed dose by pressing a button. Infusion is performed using a special pump(2).

PCA was first described by Sechzer in 1968 using intravenous opioid administration. After demonstrating that small doses of intravenous (IV) opioid administration are more effective than traditional methods, a system has been developed in which the patient can control the dose of analgesic medication. This system has been defined as "the patient directly controlling their own pain using certain doses of analgesics." PCA has begun to be used in pain management following various surgeries, including major surgeries, thanks to advancements in microchip technology after the mid-1980s (3). PCA means more patient satisfaction, less sedation, and fewer post-operative complications. Patient-controlled analgesia is often preferred due to its positive contribution to the healing process of patients. Patient-controlled analgesia operates on the principle that the patient is responsible for their own pain management (4). PCA is mostly administered intravenously or epidurally, but it can also be administered by

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CONCLUSION

Intravenous PCA method has a long-established safety history. Although postoperative pain management can be achieved with developing regional anesthesia techniques and peripheral nerve blocks that are constantly developing and newly discovered, IV PCA remains a timeless treatment regimen. In cases where regional anesthesia or analgesia is relatively and/or strictly contraindicated, for example, in patients with reluctance, coagulopathy, and a history of blood thinning medication, IV PCA is still a good choice for pain relief. Successful postoperative pain management also contributes to the national economy with secondary benefits such as early mobilization, early discharge, early oral intake, and reduction of thromboembolic events.

REFERENCES

1. Yücel A. Postoperatif ağrı, nörofizyolojisi ve stres yanıt. In: (eds), Postoperatif Analjezi (ed) Mavimer Matbacılık, İstanbul 2004. s. 7-18.
2. PE., Macintyre. Safety and efficacy of patient-controlled analgesia. Br J Anaesth. 2001, 87 1, s. 36-46.
3. A Method of Pain Control: Patient Controlled Analgesia. Eti Aslan, F. ve Türkmen, E. 1, İstanbul : SANERC, Temmuz 2005, Hemşirelikte Eğitim ve Araştırma Dergisi, Cilt 2, s. 6-11. ISSN:2618-5741. s. 6-11.
4. Erdine S. PCA (Patient Controlled Analgesia). Ağrı. 3. Baskı. İstanbul: Nobel Tıp Kitabevleri; 2007. s. 188-97.
5. Atim A, Deniz S, Orhan ME, Sizlan A, Kurt E. A clinic's experiences in postoperative patient controlled analgesia. Agri. 2009;21;155-60. 21(4):155- 160
6. Çilingir D,Uzun Şahin C. Cerrahi Hastasında Hasta Kontrollü Analjezi Kullanımı Hacettepe Üniversitesi Hemşirelik Fakültesi Dergisi 2016, 3(3), 56-69
7. Grass JA. Patient-controlled analgesia. Anesth Analg. 2005; 101(5): 44-61.
8. Erdine S. Ağrı. İstanbul: Nobel Matbaacılık; 2002. s. 144-53.

Chapter 5

LIVER CYSTS

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Aylin ACAR[2]

Liver cysts are fluid-filled sacs that can form in the liver. They ca be small and 1harmless, or they can grow larger and cause symptoms.

SIMPLE CYSTS: DIAGNOSIS AND MANAGEMENT

Simple hepatic cysts are fluid-filled cavities that are typically discovered incidentally during radiological examinations. The majority of these cysts are benign and asymptomatic. However, they can occasionally produce symptoms and complications.

Several imaging studies, including ultrasound, CT scan, and MRI, can be utilized to diagnose hepatic lesions. These tests can assist in determining the lesions' location, size, and characteristics. Hepatic cysts must be distinguished from hemangiomas, focal nodular hyperplasia, and hepatocellular carcinoma during differential diagnosis. In some instances, a needle aspiration biopsy may be required for diagnosis confirmation (1).

The treatment of hepatic cysts is contingent on the presence of symptoms. Patients who are asymptomatic typically do not need treatment and can be monitored with periodic imaging studies. Symptomatic patients may require surgical or minimally invasive procedures such as laparoscopic deroofing, sclerotherapy, or percutaneous drainage to alleviate their symptoms. The vast majority of patients with hepatic simple cysts have an outstanding prognosis.

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* Enlarged liver
* Weight loss

Diagnosis

Cystic liver metastases are usually diagnosed using imaging tests such as CT scan, MRI, or ultrasound. A biopsy may also be performed to determine the type of cancer and guide treatment.

Treatment

The treatment for cystic liver metastases depends on the type of cancer, the size and location of the cysts, and whether the cancer has spread to other parts of the body. Treatment options may include surgery, chemotherapy, radiation therapy, or a combination of these therapies.

Surgical resection is the preferred treatment option for patients with cystic liver metastases. However, in some cases, surgery may not be possible due to the size or location of the cysts, or because the cancer has spread to other parts of the body. In these cases, chemotherapy and/or radiation therapy may be used to shrink the cysts and slow the growth of the cancer.

Prognosis

The prognosis for cystic liver metastases depends on several factors, including the type of cancer, the size and location of the cysts, and whether the cancer has spread to other parts of the body. Patients with cystic liver metastases typically have a poorer prognosis than those with primary liver cancer, but the prognosis can vary widely depending on the individual case.

In conclusion, cystic liver metastases are a rare form of liver cancer that can occur when cancer cells from a primary tumor in another part of the body spread to the liver and form cystic lesions. Early diagnosis and appropriate treatment are important for improving outcomes and managing symptoms.

REFERENCES:

1. Torres VE, Harris PC, Pirson Y. Autosomal dominant polycystic kidney disease. Lancet. 2007 May 12;369(9579):1287-301. doi: 10.1016/S0140-6736(07)60601-1. PMID: 17434405.
2. Kim JH, Lee JM, Han JK, et al. Hepatic cyst infection after percutaneous aspiration and ethanol sclerotherapy: frequency and risk factors. AJR Am J Roentgenol. 2006 Feb;186(2):449-56. doi: 10.2214/AJR.04.1957. PMID: 16423952.
3. Yoon JH, Lee JM. Imaging Diagnosis and Treatment of Hepatic Cystic Lesions. Clin Mol Hepatol. 2013 Sep;19(3):257-65. doi: 10.3350/cmh.2013.19.3.257. Epub 2013 Sep 25. PMID: 24133492; PMCID: PMC3798348.

4. Kim HJ, Lee JH, Park MS, Kim KW. Imaging Features of Simple Hepatic Cysts and Pseudocysts on MRI and CT. Korean J Radiol. 2015 Jan-Feb;16(1):31-42. doi: 10.3348/kjr.2015.16.1.31. Epub 2015 Jan 8. PMID: 25598671; PMCID: PMC4292094.
5. El-Serag HB, Tran T, Everhart JE. Diabetes increases the risk of chronic liver disease and hepatocellular carcinoma. Gastroenterology. 2004 Apr;126(4):460-8. doi: 10.1053/j.gastro.2003.10.065. PMID: 14988834.
6. Lee KF, Wong J, Li JC, Lai PB. Liver cysts: treatment with percutaneous ethanol sclerotherapy. J Vasc Interv Radiol. 2007 Jan;18(1 Pt 1):95-8. doi: 10.1016/j.jvir.2006.08.025. PMID: 17296711.
7. Keane MG, Horsfall L, Rait G, Pereira SP. Natural history of simple hepatic cysts detected on ultrasound. Int J Hepatol. 2014;2014:459068. doi: 10.1155/2014/459068. Epub 2014 May 15. PMID: 24926450; PMCID: PMC4043753.
8. Lai EC, Tomlinson JS, Fan ST. Polycystic liver disease: a comprehensive review. Surg Today. 2005;35(1):1-15. doi: 10.1007/s00595-004-2908-4. Epub 2005 Jan 6. PMID: 15640761.
9. Lantinga MA, Drenth JP. Genetic and Clinical Characteristics of Polycystic Liver Disease. Biomed Res Int. 2014;2014:141240. doi: 10.1155/2014/141240. Epub 2014 Jun 10. PMID: 24971313; PMCID: PMC4065022.
10. Qian LJ, Zhu J, Zhuang LP, et al. Hepatic Cyst Infection in Polycystic Liver Disease: Clinical Characteristics and Outcomes of Antibiotic Therapy. AJR Am J Roentgenol. 2016 Aug;207(2):W26-32. doi: 10.2214/AJR.15.15585. Epub 2016 May 18. PMID: 27193391.
11. Nishikawa H, Enomoto H, Iwata Y, et al. Clinical significance of simple hepatic cyst with respect to metabolic syndrome in Japanese patients: A retrospective cohort study. J Gastroenterol Hepatol. 2015 Nov;30(11):1615-21. doi: 10.1111/jgh.13015. PMID: 25864901.
12. Vicens RA, Scott R, Kronenberger B, et al. Magnetic resonance imaging is superior to computed tomography for detection and characterization of focal hepatic lesions in patients with normal or abnormal liver function. J Comput Assist Tomogr. 2013 Jul-Aug;37(4):545-50. doi: 10.1097/RCT.0b013e3182905a5c. PMID: 23863560.
13. European Association for the Study of the Liver (EASL). EASL clinical practice guidelines on the management of benign liver tumours. J Hepatol. 2016 Aug;65(2):386-98. doi: 10.1016/j.jhep.2016.04.006. Epub 2016 May 6. PMID: 27167036.
14. National Institute of Diabetes and Digestive and Kidney Diseases. Polycystic liver disease. Updated May 2017. Accessed April 24, 2023. https://www.niddk.nih.gov/health-information/liver-disease/polycystic-liver-disease
15. European Society of Gastrointestinal and Abdominal Radiology (ESGAR). ESGAR consensus statement on liver cystic lesions. Insights Imaging. 2013 Apr;4(2):231-43. doi: 10.1007/s13244-013-0230-0. Epub 2013 Feb 23. PMID: 23435609; PMCID: PMC3627841.
16. Pappas-Gogos G, Karavias D, Giannakopoulos I, Kesisoglou I, Kakaviatos N, Varnavas G. Polycystic Liver Disease: A Comprehensive Review of Pathogenesis, Diagnosis, and Management. Med Sci Monit Basic Res. 2019 Oct 30;25:256-266. doi: 10.12659/MSMBR.917401. PMID: 31667709; PMCID: PMC6832787.

Chapter 6

LIVER HEMANJIOMAS

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Aylin ACAR[2]

Hepatic hemangioma is the most prevalent benign liver malignancy. It originates in the vascular structures of the liver and is characterized by anomalous blood vessel growth. They are the most prevalent benign liver lesions, with a prevalence of 5 to 20% among the general population and women are more likely to have them. Hepatic hemangiomas are typically asymptomatic and are frequently discovered during unrelated imaging procedures. However, larger hemangiomas can cause abdominal pain, bloating, and a feeling of fullness, and can contribute to diagnostic uncertainty and treatment difficulties.

EPIDEMIOLOGY AND RISK FACTORS

Aynı olmasın The most prevalent benign liver tumors are hepatic hemangiomas, which are characterized by the proliferation of blood vessels within the liver tissue. They occur approximately five times more frequently in women than in males, with a ratio of approximately 3:1. The incidence of hepatic hemangiomas peaks between the ages of 30 and 50 in adults. Nevertheless, they may be present at birth or develop during childhood. Although the precise cause of hepatic hemangiomas is unknown, a number of risk factors have been identified. The increased prevalence of these tumors during pregnancy or in women taking oral contraceptives or hormone replacement therapy suggests that female reproductive hormones, particularly estrogen, play a significant role in their development and proliferation. Furthermore, genetic syndromes such as hereditary hemorrhagic telangiectasia (HHT) are associated with an increased risk of developing hepatic hemangiomas. Other risk factors, such as liver injury or inflammation, have also been implicated; however, their direct relationship with the development of

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of internal hemorrhage. Hemorrhage may necessitate intervention, such as embolization or surgical excision, in order to prevent anemia.

Compression of Adjacent Structures: Large hepatic hemangiomas can exert pressure on adjacent structures including the biliary ducts and hepatic veins. This compression may cause obstructive jaundice, obstruction of hepatic venous outflow, or portal hypertension. It may be necessary to intervene to mitigate the compression and associated symptoms.

Kasabach-Merritt Syndrome: This uncommon complication is distinguished by the consumption of platelets and the development of coagulopathy within the hepatic hemangioma. It can cause life-threatening thrombocytopenia, bleeding disorders, and other complications. There may be a need for prompt diagnosis and treatment, including surgical intervention.

Symptomatic Enlargement: While the majority of hepatic hemangiomas remain stable in size, some may enlarge and manifest symptoms over time. There may be discomfort, abdominal pain, or other symptoms associated with enlargement. Intervention or surgical excision may be contemplated in such situations.

REFERENCES:

1. Bajenaru N, Balaban V, Săvulescu F, et al. Clinical and morphological features of hepatic hemangiomas. J Gastrointestin Liver Dis. 2006;15(3):245-250. PMID: 17077973.
2. Chen S, Chen H, Xia H, et al. The etiology and pathogenesis of hepatic hemangioma: a review. Am J Med Sci. 2019;357(4):282-291. doi: 10.1016/j.amjms.2018.12.010. PMID: 30961814.
3. Yu X, Liu B, Jing X, et al. Diagnosis and management of hepatic hemangioma: a comprehensive review. Sci Prog. 2021;104(2):00368504211003938. doi: 10.1177/00368504211003938. PMID: 33929377.
4. Midorikawa Y, Takayama T. Hepatic hemangioma: current management strategy. World J Gastroenterol. 2014;20(24):7897-7903. doi: 10.3748/wjg.v20.i24.7897. PMID: 24976755.
5. Kamaleshwaran KK et al., "F-18 fluorodeoxyglucose PET/CT in evaluation of hepatic hemangiomas", Indian Journal of Nuclear Medicine, 2012.
6. Torzilli G, Palmisano A, Del Fabbro D, et al. Hepatic hemangioma: the surgeons' view. J Hepatobiliary Pancreat Surg. 2005;12(6):433-440. doi: 10.1007/s00534-005-0967-9. PMID: 16429231.
7. Zhu K, Meng X, Zhou X, et al. Ethanol ablation for hepatic hemangioma: a systematic review and pooled analysis. Int J Hyperthermia. 2021;38(1):100-109. doi: 10.1080/02656736.2020.1846693. PMID: 33257876.
8. Alper A., Ariogul O., Emre A., Uras A., Okten A. Treatment of liver hemangiomas by enucleation. Arch Surg. 1988 May;123:660–661. doi: 10.1001/archsurg.1988.01400290146027. PMID: 3358693.

9. İlgin Özden, Arzu Poyanlı, Yılmaz Önal, Ali Aslan Demir , Gültekin Hoş , Bülent Acunaş. Superselective Transarterial Chemoembolization as an Alternative to Surgery in Symptomatic/Enlarging Liver Hemangiomas. World J Surg. 2017 Nov;41(11):2796-2803. doi: 10.1007/s00268-017-4069-5.

Chapter 7

PREOPERATIVE ALBUMIN AND DEVELOPMENT ATRIAL FIBRILLATION IN HEART SURGERY

Selen ÖZTÜRK[1]

INTRODUCTION:

Atrial fibrillation is a common arrhythmia after cardiac surgery. Studies have shown that postoperative atrial fibrillation (POAF) increases long-term cardiovascular mortality and morbidity (ischemic stroke, heart failure, cerebrovascular diseases) (1,2).

Determining the risk of atrial fibrillation development in the postoperative period is effective in planning treatment and therefore reducing morbidity and mortality. For this reason, many biomarkers such as serum vitamin D levels has been investigated for the formation of POAF in patients undergoing heart surgery (3).

Inflammation, oxidative stress, and autonomic nervous system stimulation play a role in the pathogenesis of POAF, which is caused by many perioperative risk factors (age, gender, obesity, diabetes mellitus, duration of cardiopulmonary bypass (CPB), infection, bleeding and inotropic use) (4).

In this study, we aimed to investigate whether preoperative levels of albumin can predict POAF development after cardiac surgery.

METHOD:

Database screening was completed in accordance with the published guidelines (5). Aim of the review was to determine the possible role of preoperative serum albumin levels importance for the prediction of atrial fibrillation following cardiac surgery. And also we aimed

to determine the possible cut-off point for albumin level. We investigated the database between 01.01.2023 and 01.03.2023. No publication date was determined for the trials. Used electronic databases were Scopus, Web of Science, Ovid, and

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factor only in univariate logistic regression analysis. On the other hand, in an epidemiological study conducted by Liao et al. (29) in 2020, the connection between serum albumin level and atrial fibrillation in a total of 12833 patients were examined with the Mendelian randomization. According to this study, there was an inverse relationship between serum albumin level and the frequency of atrial fibrillation independently in a linear pattern; however, the mendelian randomization analysis showed that albumin did not have a causal role in the occurrence of atrial fibrillation. This result is consistent with the meta-analysis we have done.

Limitations:

Our analysis has two important limitations. The first is that the studies are not randomized controlled studies. The second is the low number of patients in individual studies. Although POAF is a complication with a high frequency and serum albumin level is an easily accessible marker, the small sample size is seen as an important limitation in terms of studies.

CONCLUSION:

Albumin level is generally used for prediction of nutritional status of intensive care patients. And it is also a biomarker of inflammation. Inflammation is one of the important cause of atrial fibrillation. Therefore, it is thought that it can be used to determine postoperative atrial fibrillation. However in contrast to literature our analysis could not determine that the preoperative serum albumin level as an inflammation marker has an effect on the development of POAF. Despite this result the larger randomized controlled studies are needed on this subject because of the high heterogeneity of the published studies.

REFERENCES

1. Thorén E, Wernroth ML, Christersson C, et al. Compared with matched controls, patients with postoperative atrial fibrillation (POAF) have increased long-term AF after CABG, and POAF is further associated with increased ischemic stroke, heart failure and mortality even after adjustment for AF. Clin Res Cardiol. 2020;109(10):1232-1242. doi:10.1007/s00392-020-01614-z
2. Thorén E, Hellgren L, Granath F, et al. Postoperative atrial fibrillation predicts cause-specific late mortality after coronary surgery. Scand Cardiovasc J. 2014;48(2):71-78. doi:10.3109/14017431.2014.880793
3. Öztürk S, Öztürk İ. Atrial fibrillation after cardiac surgery and preoperative vitamin D levels: A systematic review and meta-analysis. Turk Gogus Kalp Damar Cerrahisi Derg. 2020;28(1):101-107. Published 2020 Jan 23. doi:10.5606/tgkdc.dergisi.2020.18387

4. Rezaei Y, Peighambari MM, Naghshbandi S, et al. Postoperative Atrial Fibrillation Following Cardiac Surgery: From Pathogenesis to Potential Therapies. Am J Cardiovasc Drugs. 2020;20(1):19-49. doi:10.1007/s40256-019-00365-1
5. Moher D, Shamseer L, Clarke M, et al. Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015 statement. Syst Rev. 2015;4(1):1. Published 2015 Jan 1. doi:10.1186/2046-4053-4-1
6. Akgul E, Parlar AI, Erkul GSA, et al. Investigation of the Effect of Preoperative Hypoalbuminemia, Blood Urea Nitrogen and Creatinine Levels on Postoperative Atrial Fibrillation on Off-Pump Coronary Bypass Surgery Patients. Heart Surg Forum. 2020;23(5):E641-E646. Published 2020 Sep 8. doi:10.1532/hsf.3163
7. Aksoy F, Uysal D, Ibrişim E. Predictive values of C-reactive protein/albumin ratio in new-onset atrial fibrillation after coronary artery bypass grafting. Rev Assoc Med Bras (1992). 2020;66(8):1049-1056. doi:10.1590/1806-9282.66.8.1049.
8. Limite LR, Magnoni M, Berteotti M, et al. The predictive role of renal function and systemic inflammation on the onset of de novo atrial fibrillation after cardiac surgery. Eur J Prev Cardiol. 2016;23(2):206-213. doi:10.1177/2047487314564896.
9. Karabacak K, Kubat E, Akyol FB, et al. The C-reactive protein/albumin ratio as a new predictor for postoperative atrial fibrillation after coronary artery bypass graft surgery. J Card Surg. 2020;35(10):2747-2753. doi:10.1111/jocs.14898.
10. Bernstein LH, Leukhardt-Fairfield CJ, Pleban W, et al. Usefulness of data on albumin and prealbumin concentrations in determining effectiveness of nutritional support. Clin Chem. 1989;35(2):271-274. doi.org/10.1093/clinchem/35.2.271
11. Wada H, Dohi T, Miyauchi K, et al. Independent and Combined Effects of Serum Albumin and C-Reactive Protein on Long-Term Outcomes of Patients Undergoing Percutaneous Coronary Intervention. Circ J. 2017;81(9):1293-1300. doi:10.1253/circj.CJ-17-0124
12. Joles JA, Willekes-Koolschijn N, Koomans HA. Hypoalbuminemia causes high blood viscosity by increasing red cell lysophosphatidylcholine. Kidney Int. 1997;52(3):761-770. doi:10.1038/ki.1997.393
13. Laffey JG, Boylan JF, Cheng DC. The systemic inflammatory response to cardiac surgery: implications for the anesthesiologist. Anesthesiology. 2002;97(1):215-252. doi:10.1097/00000542-200207000-00030
14. Wan S, LeClerc JL, Vincent JL. Inflammatory response to cardiopulmonary bypass: mechanisms involved and possible therapeutic strategies. Chest. 1997;112(3):676-692. doi:10.1378/chest.112.3.676
15. Suakıtıcı S, Güven BB, Tanoglu A, et al. A combination of levosimendan and N-Acetylcysteine shows significant favorable efficacy on experimental liver ischemia/reperfusion injury. Levosimendan ve N-Asetilsistein kombinasyonu deneysel karaciğer iskemi/reperfüzyon hasarı üzerine olumlu etkiler göstermektedir. Ulus Travma Acil Cerrahi Derg. 2021;27(4):381-388. doi:10.14744/tjtes.2020.81782
16. Rimmelé T, Venkataraman R, Madden NJ, et al. Comparison of inflammatory response during on-pump and off-pump coronary artery bypass surgery. Int J Artif Organs. 2010;33(3):131-138. doi:10.1177/039139881003300301
17. Orhan G, Sargin M, Senay S, et al. Systemic and myocardial inflammation in traditional and off-pump cardiac surgery. Tex Heart Inst J. 2007;34(2):160-165. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1894692/

18. Lumeng CN, Bodzin JL, Saltiel AR. Obesity induces a phenotypic switch in adipose tissue macrophage polarization. J Clin Invest. 2007;117(1):175-184. doi:10.1172/JCI29881
19. Mazurek T, Kiliszek M, Kobylecka M, et al. Relation of proinflammatory activity of epicardial adipose tissue to the occurrence of atrial fibrillation. Am J Cardiol. 2014;113(9):1505-1508. doi:10.1016/j.amjcard.2014.02.005
20. Lau DH, Shipp NJ, Kelly DJ, et al. Atrial arrhythmia in ageing spontaneously hypertensive rats: unraveling the substrate in hypertension and ageing. PLoS One. 2013;8(8):e72416. doi:10.1371/journal.pone.0072416
21. Chen MC, Chang JP, Liu WH, et al. Increased inflammatory cell infiltration in the atrial myocardium of patients with atrial fibrillation. Am J Cardiol. 2008;102(7):861-865. doi:10.1016/j.amjcard.2008.05.038
22. Andrade J, Khairy P, Dobrev D, et al. The clinical profile and pathophysiology of atrial fibrillation: relationships among clinical features, epidemiology, and mechanisms. Circ Res. 2014;114(9):1453-1468. doi:10.1161/CIRCRESAHA.114.303211
23. Sinno H, Derakhchan K, Libersan D, et al. Atrial ischemia promotes atrial fibrillation in dogs. Circulation. 2003;107(14):1930-1936. doi:10.1161/01.CIR.0000058743.15215.03
24. Kornej J, Reinhardt C, Kosiuk J, et al. Response of circulating heat shock protein 70 and anti-heat shock protein 70 antibodies to catheter ablation of atrial fibrillation. J Transl Med. 2013;11:49. doi:10.1186/1479-5876-11-49
25. Baba A, Yoshikawa T, Fukuda Y, et al. Autoantibodies against M2-muscarinic acetylcholine receptors: new upstream targets in atrial fibrillation in patients with dilated cardiomyopathy. Eur Heart J. 2004;25(13):1108-1115. doi:10.1016/j.ehj.2004.05.012
26. De Jong AM, Maass AH, Oberdorf-Maass SU, et al. Mechanisms of atrial structural changes caused by stretch occurring before and during early atrial fibrillation. Cardiovasc Res. 2011;89(4):754-765. doi:10.1093/cvr/cvq357
27. Mukamal KJ, Tolstrup JS, Friberg J, et al. Fibrinogen and albumin levels and risk of atrial fibrillation in men and women (the Copenhagen City Heart Study). Am J Cardiol. 2006;98(1):75-81. doi:10.1016/j.amjcard.2006.01.067
28. van Beek DEC, Kuijpers YAM, Königs MHH, et al. Low serum albumin levels and new-onset atrial fibrillation in the ICU: a prospective cohort study. J Crit Care. 2020;56:26-30. doi:10.1016/j.jcrc.2019.11.011
29. Liao LZ, Zhang SZ, Li WD, et al. Serum albumin and atrial fibrillation: insights from epidemiological and mendelian randomization studies. Eur J Epidemiol. 2020;35(2):113-122. doi:10.1007/s10654-019-00583-6