

Unresectable malignant gallbladder neoplasm

Juliana Sofia Silva Vieira¹, Paula Karolyne Simões Mello².

INTRODUCTION

Gallbladder cancer often originates from somatic mutations in cells of the biliary epithelium, which can be precipitated by chronic inflammation (as in chronic cholelithiasis and cholecystitis), infections (e.g., by *Salmonella* spp. or *Helicobacter* spp.), and exposure to carcinogens. Adenocarcinomas account for more than 90% of gallbladder cancer cases, with other histological subtypes including squamous cell carcinomas, adenosquamous cells, and neuroendocrine tumors.

CLINICAL CASE

Female patient, 50 years old, born and from Pilar/AL, 03 children, housewife, denies alcoholism and smoking, hypertensive using hydrochlorothiazide le amlodipine, morphine 30mg every 6h/6h, domperidone 12h/12h, simethicone 8h/8h, pantoprazole 40mg fasting, ondansertron 4mg 8h/8h, buscoduo8h/8h, muvinlax 12h/12h, minilax once a day, denies allergies and other comorbidities. She is admitted to the office in a lucid and oriented wheelchair, accompanied by her sister with a report of inappetence and asthenia for a week, informs that she has already been admitted to the emergency room of the Santa Casa de Maceió Hospital four days ago with the same symptoms, without improvement, denies pain, VAS: 0.

On a CT scan of the total abdomen performed on December 29, 2023, it shows expansive formation in the right hyochondrium with gallbladder origin 15 x 3 x 9.7 cm with close contact with the hepatic parenchyma with compression of the duodenum. On the following day, a patient with a significant worsening of the general condition was found, pyloric syndrome was found and an emergency laparotomy was performed on (12/30/23) evidence of gastroenteroanastomosis, invasion of the duodenum and a large liver tumor. Gentamicin 1,000 mg/m² IV was evaluated, infused in 30 minutes on days D1 and D8 and cisplatin 25 mg/m² EV, on days D1 and D8. Laboratory tests of 02/23/24: Hb: 7.0, Leuco: 21.8000, Bast: 2%, Segm: 90%, Platelets: 746.000, EAS: negative nitrite, bacteria present, Leuco of 10 per field and red blood cells of 3 per field.

¹ 6th year medical student at Cesmac University Center – Maceió Alagoas

² Oncologist at Santa Casa de Misericórdia de Maceió



No day 28/02/24: Hb 8.2, Ht: 25.9%, Leucus: 22,300, Platelets 370,000, Bacterioscopic urine absent, Partial hemoculture negative, CRP: 32.70, Na: 130, K: 5.2, Urea: 60, Creatinine: 0.69, Mg: 1.9, Calcium: 6.9, TGO:41, TGP: 18, Alkaline phosphatase: 1,069, GGT: 294, Total bilirubin: 0.57, Bilirubin direta: 0.55, Bilirubin indireta: 0.02, Albumin: 2.0

In the evolution carried out on 2/29/24, the patient in an emergency bed accompanied by her sister, sleepy, but responds to a call, confused, afebrile, with evacuations absent for five days, the companion denies vomiting, fever and reports that the patient complained of pain in the gluteal region and upper abdomen at her awake moment, and continues on an enteral diet. On physical examination PS4, eupneic on room air, acyanotic, afebrile, pale (2+/4+), emaciated (2+/4+), dehydrated (2+/4+), abdomen with hepatomegaly - hardened tumor 5cm from the right costal margin, no signs of peritonitis, warmed and well-perfused extremities, edema in lower limbs (4+/4+) and bilateral upper limbs (1+/4+), ACV and AP without significant changes. Active problems: asthenia and inappetence. The patient was two days later, with relative improvement in the general clinical picture and was aware of the palliative care of the case. The family was discharged with biweekly outpatient follow-up and informed about palliative care and the severity of the situation.

DISCUSSION

Genomic studies have identified several molecular alterations associated with gallbladder cancer. Mutation, amplification, or overexpression of oncogenes such as KRAS, ERBB2 (HER2/neu), and alterations in tumor suppressor genes such as TP53 and SMAD4 are common. These alterations contribute to carcinogenesis through several pathways, including the MAP kinase pathway, the PI3K/AKT/mTOR signaling pathway, and the TGF-beta signaling pathway.

Surgical resection in this case will not have any benefit since the severity and invasion of the neoplasm into other adjacent tissues and adjuvant chemotherapy and radiotherapy were recommended. Patient remains in outpatient follow-up with clinical oncologist.

CONCLUSION

The prognosis varies significantly with the stage of the cancer at the time of diagnosis. Tumors diagnosed early have a significantly higher chance of curative treatment compared to those in advanced stages, where therapeutic options are mostly palliative.

Research continues to advance the understanding of the molecular mechanisms underlying gallbladder cancer in hopes of developing more effective targeted therapies and prevention strategies.

Keywords: Gallbladder cancer, Chronic inflammation, Neoplasm.



REFERENCES

- Arminski, T. C. (1949). Primary carcinoma of the gallbladder: A collective review with the addition of twenty-five cases from the Grace Hospital, Detroit, Michigan. *Cancer*, 2(4), 379-398.
- Strauch, G. O. (1960). Primary carcinoma of the gallbladder: Presentation of seventy cases from the Rhode Island Hospital and accumulative review of the last ten years of the American literature. *Surgery*, 47(3), 368-383.
- Carneiro, P. C. A. (1991). Tumores malignos primários da vesícula biliar: Estudo de 40 casos (Unpublished doctoral thesis). UNI-Rio, Rio de Janeiro, RJ.
- Kowalewsky, K., & Todd, E. F. (1971). Carcinoma of the gallbladder induced in hamsters by insertion of cholesterol pellets and feeding dimethylaminoanthracene. *Proceedings of the Society for Experimental Biology and Medicine*, 136(2), 482-489.