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Morphin Colic Or Morphin-Induced Sphincter of Oddi Dysfunction Syndrome: Analytical Evidence

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1. Abstract

1.1. Introduction

Morphine-induced biliary colic is a complication that has been described for years. Morphine can cause dysfunction and spasms of the sphincter of Oddi, raising intraciliary pressure and increasing the risk of biliary symptoms and pancreatitis. Its exact incidence is not clearly established, but it is considered a very rare, though clinically relevant, adverse effect, especially in patients with biliary disease. In any case, its evolution is difficult to objectively assess through laboratory tests; a key aspect of this report.

1.2. Clinical Case

We present the case of a 47-year-old patient, cholecystectomy patient diagnosed with irritable bowel syndrome who, after administration of 5mg of morphine subcutaneously for lower back pain, develops intense epigastric pain. The tests revealed elevated transaminases and the scan showed a dilated common bile duct without evidence of lithiasis. Following symptomatic treatment, liver enzymes (which were elevated in the initial serial measurements) improved, normalizing at discharge. The diagnosis was biliary colic secondary to morphine administration.

1.3. Conclusion

Although rare, opioid-induced sphincter of Oddi dysfunction syndrome should be suspected when epigastric colicky pain and abnormal laboratory findings of hypertransaminasemia and cholestasis are observed after opioid administration, all of which are transient, and without detectable lithiasis or tumours. In these cases, morphine causes sustained contraction of the sphincter of Oddi, raising biliary pressure and generating transient cholestatic symptoms. Several factors increase this risk: individuals with known biliary pathology (gallstones or sphincter dyskinesia), those receiving high or repeated doses of morphine, and young women with a history of functional abdominal pain.

2. Introduction

Morphine-induced biliary colic, or Oddi Sphincter Dysfunction Syndrome, is a complication of opioid treatment described for years [1-4], and one of the most deeply ingrained in the subconscious of healthcare professionals. Its incidence is not clearly established [4-6], but it is considered very rare5,6. Clinically relevant, it is a transient and benign complication, demonstrated by manometry in the intramural sphincter of Oddi [2,4]. Although some authors have succeeded in doing so5-7, its evolution is difficult to objectively assess through laboratory tests, to the point that some authors doubt its existence5; a relevant aspect of this article.

3. Clinical Case

We present the case of a 47-year-old patient, cholecystectomy patient with a prior diagnosis of Irritable Bowel Syndrome (IBS), who, after being administered 5 milligrams of subcutaneous morphine for intense lower back pain in the Primary Care Emergency Department, within a few minutes began to experience intense epigastric pain, which is why she was referred to the Emergency Department of our centre. After evaluation, analytically identified figures of Alanine Aminotransferase (GPT/ALT) 47 units/litter (U/L) / Aspartate Aminotransferase (GOT/AST) 100 U/L / Gamma Glutamyl transferase (GGT), Alkaline Phosphatase (ALP) and total Bilirubin (BT) within normal range (Figure 1).

The patient underwent an abdominopelvic CT scan which ruled out a perforation of a hollow viscus and identified ectasia of the common bile duct without identifying a lithiasis cause.

After being treated with Paracetamol, Butyl scopolamine, Magaldrate and Omeprazole, with partial improvement of the pain, the patient underwent a new blood test where an increase in transaminases was observed: GPT/ALT 170 U/L, GOT/AST 341 U/L, GGT 48 U/L, ALP 110 U/L, and BT 0.2 milligrams/decilitre (mg/dL). The patient is progressing favourably and is therefore discharged with a follow-up appointment at 12 hours for clinical and analytical re-evaluation. Twelve hours later the patient is asymptomatic and the analytical evolution is favourable: GPT/ALT 80, GOT/AST 109 U/L, GGT 41 U/L, ALP 94 U/L, BT <0.1 mg/dL, so she is discharged with the diagnosis of morphine-induced biliary colic.

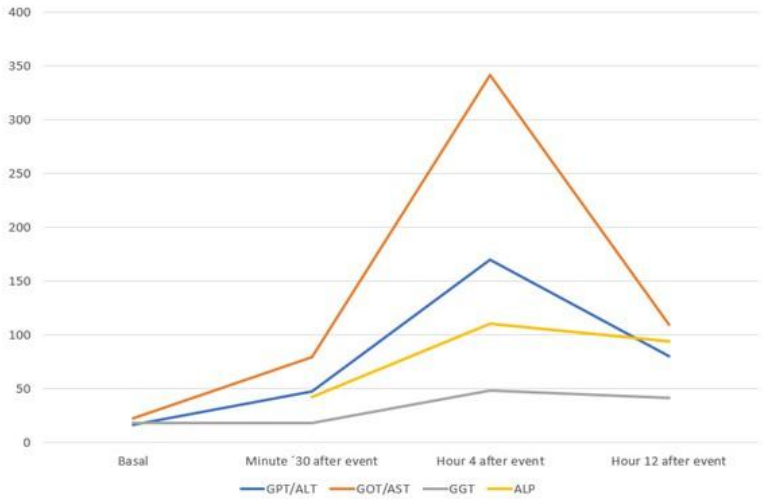

Figure 1: Evolution of transaminases over the hours following Morphine administration. GPT/ALT - Alanine Aminotransferase; GOT/AST - Aspartate Aminotransferase; GGT - Gamma-Glutamyltransferase; ALP - Alkaline Phosphatase; BT - Total Bilirubin. Range measured in units/liter for ALT, AST, GGT, and ALP. The evolution of total bilirubin is not included because it is expressed in different units of measurement.

4. Conclusion

The sphincter of Oddi regulates the flow of biliary and pancreatic secretions into the duodenum [4]. In all patients, opioids cause an increase in myogenic tone in the gastrointestinal tract, including the sphincters in all patients [1,2,5-7]: anal, leading to constipation; or of the sphincter of Oddi. It is not only a spasm of the sphincter, but a synergistic Oddi-duodenal effect [2]. As a result, the sphincters adopt a sustained hyperkinetic, clinic, and hypertonic rhythm from the first minutes after opioid administration2, and sometimes this hypertonicity manifests clinically as pain secondary to increased pressure in the biliary tract, and with elevated transaminases due to biliary congestion [1,2,4-7]. Occasionally, it also presents with pancreatitis, and even hepatocellular necrosis. These potential complications are addressed in the opioid prescribing information [8]. The clinical impact of sphincter hypertonicity, expressed as pain, is not present in all patients. This functional condition occurs especially in female patients5, those with a history of biliary disease, particularly those who have undergone cholecystectomy (since the gallbladder can act as a reservoir for refluxed bile secondary to the sudden increase in pressure resulting from the obstruction and compensate for the rise in pressure in the biliary tract [4-6], in patients with previous dyskinesia of the sphincter of Oddi, and in patients who may have a history of functional abdominal pain such as Irritable Bowel Syndrome (IBS) [5,6]. It also occurs in those receiving high or repeated doses of morphine.

The pain resulting from morphine-induced biliary colic can mimic typical biliary colic, with epigastric pain radiating to the back, nausea, and vomiting [5,7].

Morphine colic should be suspected in cases of colic-like pain following opioid treatment, with elevated transaminases, in which no lithiasis or tumours are observed with imaging tests [6,7].

This hypertonicity and the resulting clinical symptoms gradually decrease from minute 25 to 4 hours after opioid administration [2]. Nitro-glycerine, isosorbide dinitrate, and calcium channel blockers can all help relax the sphincter of Oddi [4,5], control pain, and prevent further elevation of transaminases secondary to subsequent tissue destruction. If dysfunction persists, sphincterotomy may be indicated [4,6,7].

Our case, in a woman who underwent cholecystectomy with a history of IBS, is a true reflection of the patient profile in which the side effect of morphine can have clinical repercussions.

The transient elevation of transaminases (GPT/ALT, 10 times the upper limit of normal; x10), and GOT/AST, x15), with a mild cholestatic profile (GGT, x2.5), and ALP, x3), and normal bilirubin, differs from that reported (Figure 1) by Berger5: GPT/ALT (x52), GOT/AST (x37), GGT (x9), and ALP and bilirubin within the normal range; by Sharma7, who presents mild, non-significant elevations of both cytolytic and cholestatic transaminases; or by Canullan6, in a report with gallstones and a marked cholestatic profile. Even so, they are clear evidence of the transient increase in intraciliary pressure and, therefore, of this syndrome. In this case, the indicated treatment was not used, but only symptomatic treatment until the opioid effect ceased.

References

1. Alvarez Y, Farré M. Farmacología de los opioides. Adicciones. 2005; 17(2): 21-40.
2. Cosco Montaldo H, Soto Carriquiry JP. Morphine-induced hepatic colic and its pathophysiology: Part 2. Action of morphine on the sphincter of Oddi and the biliary-duodenal junction. Electromanometry in biliary surgery. Cir. Urug. 1963; 34(5-6): 386-99.
3. Harkouk H, Pares F, Daoudi K, Fletcher D. Farmacología de los opioides. EMC-Anestesia-Reanimación. 2018; 44(2): 1-24.
4. Afghani E, Lo SK, Covington PS, Cash BD, Pandol SJ. Sphincter of Oddi Function and Risk Factors for Dysfunction. Front Nutr. 2017; 4: 1.
5. Berger Z, Arcos M, Matamala F, Rojas C. Morphine-induced biliary colic. Revista médica de Chile 2017; 145(3): 406-9.
6. Canullan C, Du Plessis MC, Merchan Del Hierro P, Quesada B, Petracchi E. An uncommon cause of extrahepatic cholestasis: morphine-induced cholestasis. Rev Fac Cien Med Univ Nac Cordoba. 2022; 79(3): 277-9.
7. Sharma V, Rana SS, Chaudhary V, Dhaka N, Manrai M. Opium-related sphincter of Oddi dysfunction causing double duct sign. Endosc Ultrasound. 2016; 5(4): 269-71.
8. Spanish Agency of Medicines and Medical Devices. Morphine B. Braun 10 mg/ml injectable solution technical data sheet [Internet]. Madrid: AEMPS; [publication date unknown. 2025.