Pharmacophore

ISSN-2229-5402

Journal home page: http://www.pharmacophorejournal.com

ASSESSMENT OF DIAGNOSTIC VALUE OF CLINICAL AND LABORATORY SIGNS IN PATIENTS WITH OBSTRUCTIVE JAUNDICE

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ARTICLE INFO

Received:
03 Apr 2018
Received in revised form:
24 Nov2019
Accepted:
05 Dec 2019
Available online:
28 Feb 2020

Keywords: obstructive jaundice, cholestatic jaundice, cholelithiasis, bile formation, bile excretion, bile ducts, mechanical jaundice

ABSTRACT

In the case of complete or partial blockage of the passage of bile through the bile tree, a syndrome of obstructive jaundice develops. Obstructive jaundice is still a serious problem, despite the widespread adoption of auxiliary diagnostic methods. According to our observations, in 75-83% of cases, it is possible to diagnose obstructive jaundice according to clinical examination and laboratory diagnostics, but the "signs" of this syndrome have a different diagnostic value, so the purpose of the work is to determine the frequency of occurrence of certain clinical and laboratory symptoms and indicators. Materials: 427 patients aged 21-92 years with a diagnosis of "obstructive jaundice". All the patients were made a comprehensive diagnosis, including clinical and laboratory examinations. Biochemical study of blood and urine has great diagnostic value, it allows not only to assume the diagnosis but also to assess the degree of metabolic disorders due to the toxic effects of bile acids. The combination of increased total bilirubin, alkaline phosphatase activity, and gamma-glutamyltranspeptidase in our study proved to be the most sensitive for determining obstructive cholestasis and cytolysis. Nevertheless, we consider the problem of research of reliable laboratory markers of obstructive jaundice, and most importantly – markers of severity of the hepatocellular failure, to be relevant.

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To Cite This Article: Kulumbegov Georgii Rolandovich, Kulumbegova Ilana Rolandovna, Iraskhanov Atabi Shaikhaevich, (2020), "Assessment of Diagnostic Value of Clinical and Laboratory Signs in Patients with Obstructive Jaundice", ***Pharmacophore, 11(1),*** *43-46.*

Introduction

One of the functions of the liver is the formation and excretion of bile (from 800 to 2000 ml), which emulsifies lipids, aids the absorption of fat-soluble vitamins, excretes some endobiotic, etc. Bile is secreted by hepatocytes and passes out through the biliary tree. Intrahepatic bile ducts include bile capillaries, cholangioles, and interlobular bile ducts. Extrahepatic bile ducts include the right and the left hepatic ducts, the common hepatic duct, the cystic duct, coming from the gallbladder, and the common bile duct. Before entering the duodenum, the common bile duct joins the duct of Wirsung forming ampulla of Vater that opens at the tip of the major duodenal papilla.

In the case of complete or partial blockage of the passage of bile through the bile tree, a syndrome of obstructive jaundice develops. To understand the pathogenesis of this condition, we consider it important to focus on some biochemical aspects of pigment metabolism. The life span of red blood cells is 80-120 days, after which it undergoes degradation in the system of erythrodieresis. Old red blood cells are destroyed in the cells of the reticuloendothelial system, mainly in the spleen (less so in the bone marrow, liver Kupffer cells, lymph nodes). Hemoglobin of red blood cells undergoes catabolism with the formation of globin (protein part) and heme. The protein component is hydrolyzed to amino acids, heme iron replenishes the total pool of iron in the body and can be reutilized. The porphyrin part of the heme undergoes irreversible transformations: biliverdin, indirect bilirubin, direct (conjugated) bilirubin, excreted with bile. In the intestine, under the influence of microflora enzymes produced by intestinal bacteria, bilirubin is converted into urobilinogen (mesobilirubinogen and stercobilinogen). Most of the stercobilinogen is converted into stercobilin and excreted in the feces, a small part is absorbed through the system of hemorrhoidal veins and, bypassing the liver, enters the systemic circulation and then is excreted by the kidneys (along with urobilin). [1, 2]

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The patency of the biliary ducts is fundamentally important for the proper performance of the liver's functions. In the case of blockage of the passage of bile, many substances, in particular, bile acids, enter the bloodstream and cause systemic toxic effects. This is how the syndrome of obstructive jaundice develops, which is caused by a block at the level of the intrahepatic or extrahepatic biliary tract. Untimely diagnosis and treatment of mechanical jaundice can lead to infectious and hemorrhagic complications, hepatorenal syndrome, hepatic encephalopathy. [3, 4]

According to our observations, in 75-83% of cases, it is possible to diagnose cholestatic jaundice with the help of clinical examination and laboratory diagnostics, but the "signs" of this syndrome have a different diagnostic value, so we consider it necessary to determine the frequency of occurrence of certain clinical and laboratory symptoms and indicators. [5]

Materials and Methods

427 patients aged 21-92 years with diagnosed mechanical jaundice. All of the patients were made a comprehensive diagnosis, including clinical and laboratory examinations. The laboratory examination included blood tests and urine tests, also, the patients were examined feces. Statistical data analysis was performed using the software package “Statistica 10”.

Results and Discussion

Among the analyzed cases, women – 289 (67.7%), men – 138 (32.3%). The severity was assessed by the level of total bilirubin and the presence of complications. In 97 cases (22.7%) patients had mild jaundice, in 274 cases (64.2%) – average jaundice and in 56 cases (13.1%) jaundice was severe. In the vast majority of cases, the cause of mechanical jaundice was non-tumor diseases of the biliary tract, in particular, cholelithiasis (table 1).

Table 1. Frequency and structure of etiological factors of obstructive jaundice (n=427)

Causes	Number of patients	
	Absolute	Percentage
Congenital abnormality:	**9**	**2.1**
– choledochal cysts	4	0.9
–duodenal diverticula	5	1.2
Non-tumor diseases of the biliary tract:	**284**	**66.5**
–cholelithiasis	262	61.4
–biliary strictures	19	4.4
–papillary stenosis	3	0.7
Inflammatory diseases:	**42**	**9.8**
–cholecystitis	26	6.1
–cholangitis	7	1.6
–chronic pancreatitis	7	1.6
–duodenal papillitis	2	0.5
Malignant causes:	**86**	**20.1**
–cholangiocarcinoma	31	7.3
–pancreatic cancer	44	10.3
–ampullary carcinoma	9	2.1
–liver/lymph node metastasis	2	0.5
Parasitic infection:	**6**	**1.4**
–echinococcosis	4	0.9
–alveococcosis	1	0.2
–ascariasis	1	0.2

Pain syndrome, as well as a feeling of heaviness in the stomach, was found in 369 patients (86.4%), the pain was localized only in the right hypochondrium in 34.7%, only in the epigastrium in 17.3%, in the right hypochondrium, and the epigastrium in 48.0% (table 2). In some cases (13.6%) there was a pain-free form of jaundice; subsequent instrumental examination revealed a tumor cause of mechanical jaundice in three-quarters of these patients. In patients with jaundice caused by tumors, a positive Courvoisier sign was also determined – a palpably enlarged and painless gallbladder. Dyspeptic symptoms (nausea, vomiting, loss of appetite) were noted by 37.7% of patients.

Table 2. Frequency and structure of symptoms of obstructive jaundice (n=427)

Symptom	Number of patients	
	Absolute	Percentage
Pain and heaviness in the stomach:	**369**	**86.4**

– right hypochondrium	128	29.9
– epigastrium	64	15.0
– right hypochondrium and epigastrium	177	41.5
The dynamics of pain:		
– sudden appearance	311	84.3
– gradual development	58	15.7
Dark beer-colored urine	394	92.3
Acholic stool	356	83.4
Skin itch	238	55.7
Weakness, irritability, sleepiness	408	95.6
Icterus:		
– mucous membranes	46	10.8
– mucous membranes + skin	381	89.2
Bradycardia	331	77.5
Low blood pressure	254	59.5
Fever	233	54.6
Dyspeptic symptoms	161	37.7
Abdominal palpation:		
– Courvoisier sign	61	14.3
– abdominal guarding in the right hypochondrium	27	6.3

Typical for mechanical jaundice is dark beer-colored urine, this symptom was observed in 394 patients (92.3%). Patients also reported foamy urine, which is caused by changes in surface tension due to the increase of bilirubin in the urine. The acholic stool was found in 356 patients (83.4%).

The general toxic effect of bile acids was manifested as skin itching (238 – 55.7%), bradycardia (331 – 77.5%), low blood pressure (254 – 59.5%), weakness, irritability, sleepiness (408 – 95.6%).

Only scleral icterus was observed in 46 patients (10.8%), and in most cases, it occurred in patients with mild jaundice. The rest of the patients had also icteric skin and visible mucous membranes.

In blood tests, leukocytosis was observed in 304 patients (71.2%), while it was expressed in the case of severe jaundice and the addition of infectious complications: suppurative cholangitis, liver abscesses, bacterial toxic shock syndrome, abdominal sepsis, etc. (Table 3). Acceleration of the ESR was observed in 362 patients (84.8%). Severe intoxication in 83 patients (19.4%) was accompanied by the appearance of signs of anemia. In 14 cases (3.3%) there was eosinophilia; these same patients were made serological parasite tests (Echinococcus, Alveococcus, Ascaris), and in 9 patients, they were positive (as revealed in the process of further diagnosis and treatment, 3 cases of parasite tests were false positive). Changes in hemostasis were registered in 266 patients (62.3%): reduction of the prothrombin time, lengthening of activated partial thromboplastin time (usually by 2-3 days). In 70.7% of patients, total protein of serum was reduced, dysproteinemia was found in 58.1%, while three-quarters of the cases were in patients with average and severe jaundice. All patients had elevated levels of total bilirubin, mainly due to the direct fraction. Specific for mechanical jaundice is elevated alkaline phosphatase (96.5%) and gamma-glutamyltranspeptidase (94.6%). The increase in transaminase activity was not pronounced (as in hepatitis), but it was observed in 283 patients (66.3%). The biochemical study also showed an increase in the level of urea (29.0%) and creatinine (34.9%), and the degree of increase was correlated with the severity of jaundice, as well as it was an indicator of the development of the hepatorenal syndrome.

Table 3. The results of laboratory blood tests in patients with mechanical jaundice (n=427)

Causes	**Number of patients**	
	Absolute	**Percentage**
Leukocytosis	304	71.2
Acceleration of the ESR	362	84.8
Signs of anemia	83	19.4
Eosinophilia	14	3.3
Hemostasiological changes	266	62.3
Low total protein	302	70.7
Dysproteinemia	248	58.1
Bilirubinemia:		
– 1st degree (up to 100 µmol/L)	97	22.7
– 2nd degree (100 to 200 µmol/L)	274	64.2
– 3rd degree (over 200 µmol/L)	56	13.1
High total cholesterol	314	73.5
Elevated alkaline phosphatase	412	96.5
Elevated transaminases (ALT, AST)	283	66.3

Elevated GGT	404	94.6
Serological parasite tests	9	2.1
Elevated blood urea	124	29.0
Elevated blood creatinine	149	34.9
High blood amylase level	66	15.5

The urine of almost all the patients was dark beer-colored and foaming, biochemical research confirmed the presence of bilirubinuria (98.6%) and the absence of urobilinogen (75.6%). The study of feces was auxiliary for diagnosis, in 74.0% of cases, there was steatorrhea, in 68.4% – the absence of stercobilin (table 4).

Table 4. The results of the laboratory examination of urine and feces in patients with mechanical jaundice (n=427)

Causes	**Number of patients**	
	Absolute	**Percentage**
Urine:		
– dark beer-color	394	92.3
– foaming	373	87.4
– bilirubinuria	421	98.6
– the absence of urobilinogen	323	75.6
Feces:		
– steatorrhea	316	74.0
– the absence of stercobilin	292	68.4

Conclusion

Cholestatic jaundice continues to be a serious problem despite the widespread introduction of auxiliary diagnostic methods. Biochemical study of blood and urine has great diagnostic value, it allows not only to assume the diagnosis but also to assess the degree of metabolic disorders due to the toxic effects of bile acids. Thus, the combination of increased total bilirubin, elevated alkaline phosphatase and gamma-glutamyltranspeptidase in our study was the most sensitive for determining obstructive cholestasis and cytolysis. Today, it is not possible to determine the optimal set of clinical and laboratory studies that make it possible to diagnose mechanical jaundice in 100% of cases without usage of instrumental diagnostics (ultrasound, endoscopic, X-ray, etc.), so the problem of finding reliable laboratory markers of mechanical jaundice, and most importantly – markers of the severity of hepatocellular failure, continues to be relevant.

Conflict of Interest

The authors declare no conflict of interest.

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