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Review Article

# Nutritional Status of Patients with Malignant Obstructive Jaundice Evaluated by Nutritional Risk Screening and the Indocyanine Green Retention Test

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## Abstract

Malignant Obstructive Jaundice (MOJ) is typically caused by malignant tumors of hepatopancreatic biliary origin and less frequently by metastatic primary malignancies of non-hepatopancreatic biliary origin. MOJ is associated with an unusually high risk of malnutrition. Therefore, effective screening of nutritional status is especially important for early detection of MOJ. We recommend the indocyanine green retention test combined with the nutritional risk screening for assessment and dynamic real-time monitoring of nutritional status to improve the treatment outcomes of MOJ patients.

**Keywords:** Malignant obstructive jaundice; Dystrophy; Nutritional status; Liver reserve function; Indocyanine green

## Introduction

Jaundice is characterized by yellow discoloration of the skin, sclera, and mucous membranes resulting from the accumulation of excess bilirubin [1]. Bilirubin is formed by heme in the presence of heme oxygenase and biliverdin reductase [2,3]. Obstructive jaundice, also known as surgical jaundice, is a relatively common clinical condition usually caused by complete mechanical obstruction of the intra/extrahepatic bile ducts and obstruction of bile flow into the intestine, resulting in cholestasis or reverse flow of ester-type bilirubin into the blood, eventually leading to jaundice [4,5].

Malignant Obstructive Jaundice (MOJ) is caused by a primary malignancy or direct/indirect obstruction of the bile duct resulting from a distant malignancy with main clinical manifestations that include hyperbilirubinemia, yellow discoloration of tissues, and dilation of the bile duct [5,6]. Obstruction of the bile duct above the common hepatic duct is termed a high duct obstruction, which is usually caused by hilar cholangiocarcinoma, hepatocellular carcinoma, local infiltration and compression resulting from gallbladder carcinoma, and invasion of the hilar bile ducts [5-7]. Obstruction of the bile duct from the common hepatic duct to the duodenal papilla, known as low duct obstruction, is usually caused by malignant tumors of the ampulla and surrounding tissues of the pancreas, lower segment of the common bile duct, and duodenal papillae [8-10]. Previous studies have reported that MOJ in up to 14% of patients is due to metastatic cancers of the kidney, lung, and stomach [11,12]. MOJ is usually diagnosed in the late stage of disease because of the absence of symptoms. Progressive obstruction of the bile duct leads to complete obstruction of bile excretion. Therefore, most patients have relatively high bilirubin levels and abnormal liver function, requiring effective decoy therapy to create opportunities for surgery or to improve quality of life [13,14].

### Impact of MOJ on Liver Function

The liver, which is the largest organ of the digestive tract, is involved in various functions that support metabolism, immunity, digestion, detoxification, and vitamin storage, among others, including rapid elimination of free bilirubin to maintain normal serum levels [15,17]. The liver has a rich lymphatic network that is a major component of the hepatic microcirculation and produces 25%–50% of the lymph fluid in the thoracic ducts [18]. The lymphatic network circulates along the bile ducts and portal veins, and the larger portal veins form bundles composed of 6–10 lymphatic vessels [18-21]. When obstructed, there is




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a marked increase in pressure throughout the biliary system that can rupture the capillaries and ciliary ducts, which communicate directly with the lymphatic system [22,23], potentially causing the accumulation of bilirubin in the blood via the sinusoidal membranes or close junctions of the hepatocytes, resulting in hyperbilirubinemia [24,25]. MOJ can increase serum levels of ester-type bilirubin, which is not excreted in the feces, thereby reducing levels in the urine. Moreover, the lack of bilirubin entering the digestive tract results in pale, "putty" colored stools and an absence of urobilinogen in the urine [26-28]. Long-term MOJ can impair bilirubin uptake, transport, and esterification by the hepatocytes, and beta-glucuronidase in many tissues can promote the hydrolysis of ester-type bilirubin, resulting in increased serum concentrations of nonester-type bilirubin [27-30].

As a result of increased pressure in the bile ducts and capillaries, the portal veins within Greenson's sheath are compressed, resulting in increased pressure in the sinusoidal sinusoids, decreased hepatic blood flow, fibrotic changes to the hepatic lobes, and prolonged cholestasis, resulting in biliary cirrhosis [31-33]. In a state of MOJ, the hepatic arteries are dilated, distorted, and the vascular plexus surrounding the bile ducts is markedly hyperplastic with irregular thickening of the arterial walls, which can result in crescent-shaped changes and narrowing of the arterial lumen and rupture or disappearance of the internal arterial elastic lamina, causing loss of arterial blood flow, eventually resulting in decreased hepatic blood flow and portal hypertension [33,34]. Cirrhosis is characterized by increased pressure in the portal veins, hyperplasia of the hepatic lymphatic system, and reabsorption of excess fluids in the liver and internal organs resulting in increased flow of hepatic lymph (up to 30 times the normal volume), which promotes ascites formation. Meanwhile, portal hypertension leads to gastrointestinal congestion, impairment of the gastrointestinal mucosal barrier and intestinal vascular barrier, formation of stress ulcers, translocation of the intestinal bacteria through the portal vein to the liver, leading to biliary infection and massive endotoxin production, which can result in disseminated intravascular coagulation [34-36].

Increasing evidence suggests that severe obstruction of the biliary tract leads to impaired liver function, immune dysfunction, coagulopathy, significant reductions to the volume of bile in the intestinal tract, and impaired feedback to the bile acid intestinal-hepatic circuits,35 resulting in impaired absorption of lipolytic vitamin K, which leads to vitamin K deficiency, further affecting the synthesis of hepatic coagulation factors II, VII, IX, and X, resulting in inhibition of the coagulation process and a tendency for severe bleeding of the nasal cavity, gingiva, and internal organs [36-39].

**Effects of MOJ on Nutritional Status**

Malnutrition affects 15% to 60% of older adults, up to 30% of hospitalized patients [40,41], and to varying degrees in patients with MOJ, with an incidence of malignant dystrophy as high as 50% [42,43]. In addition, malnutrition is associated with diminished bile flow into the gut, impaired circulation of bile acid in the intestines and liver, dysregulation of the intestinal microbiota, and promotion of inflammatory responses in the gut and liver, which further inhibits secretion of bile, disrupts the integrity of the intestinal mucosal barrier, and impairs digestion and absorption in the intestines [35,42,44]. In addition, intrahepatic accumulation of bile salts increases oxidative stress and apoptosis of hepatocytes by inhibiting production of cytochrome P450 in addition to reductions in oxidative and aerobic metabolism. Inhibition of cytochrome P450 and microsomal mixed-function oxidase affects the metabolism of drugs and other substances [45,46].

MOJ leads to reduced liver function and subsequent decreased production of albumin, coagulation factors, and immunoglobulins, resulting in diminished humoral immunity [42,47-49]. As the most important plasma protein synthesized by the liver, albumin is an important biomarker of disease severity. Although changes to serum albumin are usually absent in the early stages of biliary obstruction [50], prolonged obstruction can lead to impaired hepatic production of albumin and poor protein intake due to intestinal bile deficiency and loss of appetite, resulting in decreased serum albumin levels to >30 g/L in 56% of patients. Hence, low serum albumin levels are an important predictor of severe impairment of liver function [37,51,52]. Even after the obstruction is resolved, improvement in serum albumin levels may require a relatively long period [53]. These findings indicate that MOJ adversely affects nutrient absorption and serum albumin levels, thereby highlighting the importance of monitoring nutritional status in clinical practice.

**Indocyanine Green (ICG) Retention Test in Combination with Nutritional Risk Screening to Assess Nutritional Status**

Rapid short-term (1–2 weeks) increases in bilirubin levels can lessen the effects of compromised nutrient absorption, although the risk of malnutrition may increase significantly over time. MOJ can severely impair nutrient absorption and adversely affect prognosis and prolong hospitalization [54,55]. In addition, percutaneous transhepatic common ductal drainage and endoscopic transnasal ductal drainage can improve patient survival but increases the length of hospitalization and associated costs [56-58]. The severity of malnutrition and serum bilirubin levels vary among patients, thus early detection and treatment of MOJ are particularly important [59].

Poor nutritional status is common in hospitalized patients, especially those with chronic diseases, such as cirrhosis, advanced malignancies, and end-stage liver disease [60]. Hence, monitoring of nutritional status in a timely manner may be useful to predict morbidity and mortality in clinical practice, particularly for critically ill patients [61].

The Nutrition Risk Assessment Scale (NRS-2002) is a relatively simple, noninvasive, and low-cost tool that is commonly used in clinical practice to assess nutritional status and prevent poor treatment outcomes [62,63]. Since the degree of obesity varies among patients, the Body-Mass Index (BMI) alone is insufficient to accurately assess nutritional status, which is influenced by numerous factors, including sodium retention, edema, and limb vein thrombosis [64,65]. The presence of dystrophy in MOJ cannot be ruled out by a high BMI [66]. In addition, BMI is not a reliable biomarker for children, adolescents, and the elderly [62]. The NRS-2002 is reportedly appropriate for assessment of patients with early and mild liver disease, but has limited use for those with more severe liver disease [67-69]. Hence, combinations of tools can more accurately assess the nutritional status of high-risk patients and those in the intensive care unit [70].

The NRS-2002 combined with the ICG retention test more accurately reflects the nutritional status of patients with MOJ. At present, three methods are commonly used to assess liver reserve: (1) biochemical tests of serum biomarkers (i.e., aminotransferases, bilirubin, alkaline phosphatase, albumin, and pro-

thrombin); (2) composite scoring systems; and (3) quantitative tests of liver function [15,71,72]. However, biochemical tests of serum biomarkers reflect liver function only in one dimension with no predictive value for postoperative liver failure. In addition, the degree of variability in biomarkers is not completely consistent with the severity of liver disease and does not permit precise assessment of liver reserve function [15,73,74]. Moreover, comprehensive scoring systems, such as the Child–Pugh score, is subjective with poor comparability and does not directly reflect perfusion conditions, which are more likely to be influenced by extrahepatic factors [71,75]. As a quantitative liver function test, the ICG retention test is used to assess the dynamics of hepatic function over time in addition to physiologic status (i.e., effective hepatic or hepatic reserve function) and physiologic function (i.e., hepatocyte volume and effective infusion of hepatic blood flow [76,77]. The ICG retention test is a well-established indicator of liver reserve function, which is the most important determinant of the outcome of hepatectomy [76,78,79].

MOJ leads to decreases in the number of functional hepatocytes and hepatic blood flow, which can affect hepatic synthesis and metabolism of biomolecules, resulting in nutritional impairment [74,75,80]. However, ICG uptake is strongly influenced by hepatic blood flow and the collateral circulation, which might be useful to assess the degree of liver dysfunction and cirrhosis rather than screening of liver diseases or as a predictor of mortality.[80] Hepatocellular carcinoma is frequently associated with different degrees of cirrhosis. A recent study reported ICG-R15 values of 10%–23.7% in 183 patients with moderate cirrhosis, 10%–19% in 91 patients with severe cirrhosis, and >20% in 6.2% patients with severe cirrhosis [80,81], indicating that the risk of malnutrition is similar for patients with moderate and severe disease. Therefore, the combination of the NRS-2002 with the ICG retention test could more accurately reflect liver function and the nutritional status of patients with MOJ. Therefore, further studies are warranted to dynamically assess the nutritional status in patients with MOJ and high bilirubin levels, to investigate the effects of different bilirubin levels on the severity of malnutrition, and to explore potential relationships between serum bilirubin levels and ICG retention rates.

## Summary

MOJ can lead to varying degrees of nutritional impairment, thus early detection and treatment could improve patient outcomes and lower hospital costs, while conforming to current healthcare policies. We suggest that the combination of the NRS-2002 with the ICG retention test to more accurately reflect liver function and the nutritional status of patients with MOJ as a guide to improve therapeutic outcomes.

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