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Effects of percutaneous transhepatic biliary drainage on renal function in patients with obstructive jaundice

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PURPOSE
We assessed the effects of percutaneous transhepatic biliary drainage on renal function in patients with obstructive jaundice using the estimated glomerular filtration rate (eGFR) and evaluated the factors associated with renal dysfunction.

MATERIALS AND METHODS
Between July 2007 and September 2009, 108 consecutive patients (69 men 39 women; median age, 59 years; range, 29–87 years) with obstructive jaundice (20 benign, 88 malignant) that were unsuitable for endoscopic retrograde cholangiopancreticography were evaluated at admission and at follow-up exams five and thirty days after percutaneous transhepatic biliary drainage. Two patients with suspected contrast-induced nephropathy were excluded. Renal function was assessed by measuring levels of urea, creatinine and electrolytes and evaluating the modification of diet in the renal disease formula for eGFR.

RESULTS
eGFR was <60 mL/min/1.73 m^2 before percutaneous transhepatic biliary drainage in 27 patients (25%) and increased significantly 30 days after percutaneous transhepatic biliary drainage ($P = 0.008$). In the malignant external drainage subgroup, there was a significant increase in eGFR on the fifth day after percutaneous transhepatic biliary drainage ($P = 0.038$). The procedure-related mortality rate was zero. Nine malignant patients (8.49%) died within thirty days due to underlying diseases. On the fifth day, eGFR was significantly lower in these patients than in surviving patients ($P = 0.049$), and bilirubin levels were significantly higher before the intervention than in surviving patients ($P = 0.04$). Multiple logistic regression analysis showed that serum direct bilirubin is a significant predictor of renal function ($P = 0.049$).

CONCLUSION
Obstructive jaundice is associated with renal dysfunction, and serum direct bilirubin is a significant predictor of renal function. Percutaneous transhepatic biliary drainage improves renal function and is crucial for prognosis of obstructive jaundice.

Key words: • *jaundice, obstructive* • *glomerular filtration rate* • *renal insufficiency* • *drainage*

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Percutaneous transhepatic biliary drainage (PTBD) has become a safe and effective technique. It has been employed for decompression of the obstructed biliary tract to palliate jaundice and pruritus as well as for the management of cholangitis. In recent years, PTBD has increasingly been employed in the management of benign biliary diseases. There are many complications, but most can be treated conservatively. The procedure-related mortality is <2% in most studies (1).

Subtle renal abnormalities have been known to occur in patients with obstructive jaundice for many years, and these abnormalities predispose patients to acute renal failure when challenged with infection, hemorrhage or surgery (2). It is known that hepatobiliary surgery for obstructive jaundice is associated with high morbidity and mortality rates. The association between obstructive jaundice and perioperative renal failure has been recognized since 1910 when Clairmont and von Haberer (3) reported the development of acute renal failure and subsequent death in five patients following surgery for obstructive jaundice.

As a therapeutic method, biliary drainage has been shown in multiple experimental models to improve liver function, nutritional status, cell-mediated immune function and renal function (4–7).

Most, if not all, investigations of the effects of jaundice on renal function and its treatments have been performed in experimental animals. To our knowledge, there is no report in the literature concerning the effect of PTBD on renal function with the exception of a few reports regarding contrast-induced nephropathy after PTBD (8).

In this retrospective study, we assessed the effects of PTBD on renal function using eGFR (modification of diet in the renal disease, MDRD, formula) and evaluated the factors associated with renal dysfunction in patients with obstructive jaundice.

Materials and methods

Between July 2007 and September 2009, 108 consecutive patients with obstructive jaundice (69 men 39 women; median age, 59 years; range, 29–87 years) were evaluated at admission and at five and thirty days after PTBD.

Twenty patients had benign conditions, and eighty-eight patients had malignant tumors. Our study group included all patients with high bile duct obstructions and patients with low bile duct obstructions that could not be drained endoscopically for technical reasons.

Inclusion criteria were having a serum total bilirubin level higher than 5 mg/dL and echographic evidence of extrahepatic and intrahepatic bile duct dilations of greater than 8 and 4 mm, respectively. Patients with gastrointestinal bleeding, parenchymal liver disease, heart failure, chronic renal failure, coagulation disorders or those who used diuret-

ics were excluded. Two patients with suspected contrast-induced nephropathy were also excluded. These patients had increases of more than 2 mg/dL in serum creatinine on the fifth day after drainage, but their serum creatinine levels returned to predrainage levels on the thirtieth day.

During the study period, all patients received the same medical therapy protocol [24 hour hydration before PTBD, antibiotic prophylaxis and IV sedation (nidazolam, fentanyl citrate) during the procedure]. After the procedure, fluid was replaced as appropriate.

Urea and creatinine concentrations (mg/dL), electrolyte measurements (sodium and potassium, mEq/L) and MDRD formula, given below, for estimating the glomerular filtration rate (GFR) (mL/min/1.73 m^2),

eGFR = 175 x (standardized S_{Cr})$^{-1.154}$ x (age)$^{-0.203}$ x (0.742 if female) x (1.210 if African American) (9)

were used to assess renal function. Liver function was assessed by measuring levels of aspartate transaminase (AST), alanine transaminase (ALT), alkaline phosphatase (ALP), gamma glutamyl transpeptidase (U/L) (GGT), and total and direct bilirubin (mg/dL).

All measurements were done in serum using the Modular System (Roche Modular PP System, Roche Diagnostics GmbH, Mannheim, Germany).

Techniques

After routine checks of blood parameters, antibiotic prophylaxis, analgesic/sedative premedication, sterile precautions and informed consent, transhepatic cholangiography was carried out using a conventional diagnostic fine needle (21-G Chiba needle). We modified the techniques and instrumentation for percutaneous transhepatic biliary drainage as described by Goodwin et al. (10). The fine needle was advanced by ultrasound guidance into the dilated right or left hepatic bile ducts through the liver parenchyma. In all procedures, patients were in the supine position. A midaxillary intercostal route was preferred for decompression of the right and epigastric insertion of a fine needle into the left bile ducts. After a duct was punctured, the peripheral biliary radicle was opacified by injection of 5 mL diluted, low osmolarity, non-ionic contrast medium. After opacification of the bile ducts, a 0.018-inch, 60 cm guidewire was inserted through the lumen of a Chiba needle for access to the punctured bile duct. The guidewire was advanced through the bile ducts toward the liver hilum and into the common bile duct. The needle was then removed and a triaxial catheter set (Accustick II introducer systems, Boston Scientific, USA) was advanced over the guidewire. After removal of the inner stylet, the 0.018-inch wire was replaced by a 0.035-inch hydrophilic J-tip guidewire. First, we attempted to recanalize the bile duct obstruction with this guidewire in combination with various catheters. When recanalization was successful, a specially designed biliary ring tip catheter with multiple side holes (Uresil LP, Skokie, Illinois, USA, or Skater, PBN Medicals, Denmark) was advanced over the guidewire to achieve internal-external biliary drainage. If the first attempt to recanalize the obstructed segment was not successful, an external drainage catheter was used. After removing a minimum of 20 mL bile, a cholangiography was obtained by injection of 10 mL non-ionic contrast medium to visualize the obstructed segment and verify the catheter position. External drainage was converted to internal-external drainage within five days after decompression of the bile ducts and resolution of the edema. 8F-sized catheters were used for drainage.

Approximately 10 days after PTBD, all inoperable malignant structures were dilated with metal self-expandable stents, and all benign strictures were dilated using appropriately sized (6–10 mm) balloon catheters. After establishment of adequate internal drainage, all catheters were withdrawn.

For statistical analysis, the results were analyzed using nonparametric tests within SPSS software (SPSS, Chicago, USA) for Windows (version 10.0; Microsoft, Redmond, Washington, USA). The Wilcoxon matched-pairs signed-rank test was used for the paired comparisons. Multiple logistic regression analysis was performed to identify factors associated with renal dysfunction. Statistical analysis was carried out using the Mann–Whitney U test for unpaired data. Data are expressed as medians (1st–3rd quartiles). The statistical significance was set at $P < 0.05$.

Our institutions do not require institutional review board approval for retrospective studies.

Results

The mean ± SD duration time of biliary obstruction was 1.7 ± 3.8 months for the malignant group and 3.75 ± 6.9 months for the benign group. There was no correlation between the duration of biliary obstruction and eGFR values before drainage.

Twenty-two patients with malignant obstructions underwent surgery after PTBD. One patient had postoperative acute renal failure, and one patient had increased serum creatinine despite preoperative biliary drainage.

Four patients did not show decreased bilirubin levels due to noneffective drainage. These patients were classified as Bismuth Type 4. Non-drained segments were found in these patients. One patient who did not show decreased bilirubin levels despite effective drainage had a malignant distal choledochal obstruction. Although we effectively drained and dilated this segment with a metallic stent, the patient's bilirubin levels did not decrease, most likely because of severe hepatic insufficiency.

eGFR was <60 mL/min/1.73 m^2 before PTBD in twenty-seven patients (25%) (median, 1st–3rd quartile; 58 [44–60] mL/min/1.73 m^2) and increased significantly 30 days after PTBD (median, 1st–3rd quartile; 69 [46–95] mL/min/1.73 m^2) ($P = 0.008$).

Pretreatment values for liver and renal function were compared with the values observed on the 30th day after PTBD. Tables 1 and 2 compare variables after biliary drainage in 20 patients with benign obstructive jaundice and 86 patients with malignant obstructive jaundice, respectively. Significant decreases in liver enzymes and bilirubin levels were observed in both groups. Furthermore, although statistically insignificant ($P = 0.081$), there was an increase in eGFR values compared with pretreatment values in the malignant patients.

Patients were divided into external (ED) and internal/external (IED) drainage subgroups based on the drainage route used during the first five days after PTBD. In the malignant group, 60 patients received IED and 26 patients received ED due to failure to recanalize the obstruction. In the benign group, 7 patients received ED and 13 patients received IED. The variables in the benign and malignant ED and IED subgroups are compared in Tables 3 and 4,

respectively. In the malignant ED subgroup, there was a significant increase in eGFR on the fifth day after PTBD (P = 0.038). In the benign group, there were increases in eGFR values in both the external and internal/external drainage subgroups, although these values were statistically insignificant.

Among the 106 patients, the procedure-related mortality was zero. Nine patients with malignant obstructive jaundice died within thirty days after drainage (8.49%) due to their underlying diseases. On the fifth day, eGFR (median, 1st–3rd quartile; 59 [32–103.5] mL/min/1.73 m^2) was significantly low in these patients compared with surviving patients (86 [67–121.5] mL/min/1.73 m^2) (P = 0.049). Furthermore, total bilirubin (23.7 [21.3–28.3] mg/dL) and direct bilirubin (18.23 [9.6–23.6] mg/dL) levels were significantly higher before the intervention than in surviving patients (14.93 [6.8–24] and 9.1 [3.7–16.3] mg/dL, respectively) (P = 0.04).

Multiple logistic regression analysis showed that serum direct bilirubin is a significant predictor of renal function in patients with obstructive jaundice (P = 0.049).

Table 1. Comparison of variables after PTBD in 20 patients with benign obstructive jaundice (age range, 42.3–67.5 years; mean 55 years)

	Before PTBD	30th day after PTBD	Significance[a]
AST (U/L)	67.0 (52.5–130.7)	38.0 (21.5–61.5)	P = 0.084
ALT (U/L)	84.5 (47.5–148.2)	41.0 (25.0–77.0)[b]	P = 0.046
ALP (U/L)	517.5 (370.5–691.5)	266.0 (159.5–426.5)[b]	P = 0.009
GGT (U/L)	349.5 (203.5–414.7)	124.5 (72.7–233.0)	P = 0.084
Total bilirubin (mg/dL)	7.3 (5.3–13.5)	2.0 (1.5–4.9)[b]	P = 0.018
Direct bilirubin (mg/dL)	5.6 (5.0–11.5)	1.2 (0.6-3.5)[b]	P = 0.028
Glucose (mg/dL)	101.0(89.0–121.7)	112.0 (86.0–135.0)	P > 0.05
Urea (mg/dL)	35.5 (23.7–46.2)	36.0 (23.7–47.7)	P > 0.05
Creatinine (mg/dL)	0.87 (0.55–1.03)	0.95(0.76–1.16)	P > 0.05
GFR–MDRD (mL/min/1.73 m^2)	87.5 (73.2–123.0)	84.0 (64.7–94.5)	P > 0.05
Sodium (mEq/L)	136.5 (133.2–139.0)	140.0 (137.5–142.5)	P > 0.05
Potassium (mEq/L)	4.0 (3.1–5.9)	4.3 (3.45–4.7)	P > 0.05

PTBD, percutaneous transhepatic biliary drainage
Values are median (1st–3rd quartile)
[a]Wilcoxon signed ranks test
[b]P < 0.05 versus before PTBD

Table 2. Comparison of variables after PTBD in 86 patients with malign obstructive jaundice (age range, 49.7–73 years; mean, 59 years)

	Before PTBD	30th day after PTBD	Significance[a]
AST (U/L)	114.5 (67.5–177.0)	57.0 (35.5–129.0)[b]	P = 0.001
ALT (U/L)	89.5(55.7–196.5)	47.0 (31.0–84.5)[b]	P = 0.0001
ALP (U/L)	720.5(459.7–1441.5)	476.5 (330.0–875.0)[b]	P = 0.0001
GGT (U/L)	419.0 (262.7–854.2)	235.5 (109.0–391.2)[b]	P = 0.0001
Total bilirubin (mg/dL)	19.94 (11.01–26.82)	5.46 (2.40–10.64)[b]	P = 0.0001
Direct bilirubin (mg/dL)	13.50 (6.10–17.70)	3.28 (1.09–6.47)[b]	P = 0.0001
Glucose (mg/dL)	94.0 (81.0–119.5)	100.0 (84.0–123.5)	P > 0.05
Urea (mg/dL)	30.0 (21.0–40.0)	31.0 (22.7–52.5)[b]	P = 0.033
Creatinine (mg/dL)	0.87 (0.73–1.07)	0.81 (0.63–1.07)	P > 0.05
GFR–MDRD (mL/min/1.73 m^2)	84.5(60.0–106.7)	101.0 (69.5–123.5)	P = 0.081
Sodium (mEq/L)	135 (132–137)	135 (132–139)	P > 0.05
Potassium (mEq/L)	4.0 (3.7–4.3)	4.2 (3.7–4.7)	P > 0.05

PTBD, percutaneous transhepatic biliary drainage
Values are median (1st–3rd quartile)
[a]Wilcoxon signed ranks test
[b]P < 0.05 versus before PTBD

Table 3. Comparison of variables after PTBD in 7 patients with benign obstructive jaundice (age range, 42–62 years; mean 49 years) who received external drainage and 13 patients (age range, 42.5–69 years; mean 65 years) who received internal and external drainage

	External drainage (n = 7)			Internal/external drainage (n = 13)		
	Before PTBD	5th day after PTBD	Intragroup significance[a]	Before PTBD	5th day after PTBD	Intragroup significance[a]
AST (U/L)	73.0 (52.0–133.0)	57.0 (16.3–92.8)	$P > 0.05$	66.0 (45.0–109.5)	40.0 (31.5–53.5)[b]	$P = 0.25$
ALT (U/L)	86(70–175)	112 (26–148)	$P > 0.05$	79 (39–137)	46 (32–67)[b]	$P = 0.25$
ALP (U/L)	543 (451–707)	402 (238–741)	$P > 0.05$	468 (296–655)	342 (230–482)[b]	$P = 0.23$
GGT (U/L)	373 (255–408)	343 (283–533)	$P > 0.05$	325 (196–450)	168 (119–365)	$P > 0.05$
Total bilirubin (mg/dL)	6.41 (5.42–22.91)	4.12 (2.52–13.33)	$P > 0.05$	8.33(5.97–13.52)	7.55 (5.18–10.82)	$P > 0.05$
Direct bilirubin (mg/dL)	5.49 (5.21–16.55)	3.65 (2.48–7.21)	$P > 0.05$	6.55 (5.11–11.52)	6.68 (5.01–9.47)	$P > 0.05$
Glucose (mg/dL)	91 (89–117)	111 (84–138)	$P > 0.05$	106 (90–131)	119 (99–161)	$P > 0.05$
Urea (mg/dL)	33.0 (25.0–47.0)	38.0 (17.0–64.0)	$P > 0.05$	37.0 (22.7–47.2)	30.5 (24.0–99.0)	$P > 0.05$
Creatinine (mg/dL)	0.85 (0.55–0.93)	0.68 (0.66–0.95)	$P > 0.05$	0.97 (0.54–1.08)	0.87 (0.47–1.19)	$P > 0.05$
GFR–MDRD (mL/min/1.73 m^2)	86.0 (74.0–131.0)	96.0 (89.0–128.0)	$P > 0.05$	89.0 (65.5–119.0)	95.0 (73.0–140.5)	$P > 0.05$
Sodium (mEq/L)	134 (132–139)	137 (133–142)	$P > 0.05$	137 (135–139)	134 (131–138)	$P > 0.05$
Potassium (mEq/L)	4.4 (3.7–4.8)	4.4 (3.6–4.8)	$P > 0.05$	4.2 (4.0–4.5)	4.3 (4.1–4.4)	$P > 0.05$

PTBD, percutaneous transhepatic biliary drainage
Values are medians (1st–3rd quartile)
[a]Wilcoxon signed ranks test
[b]$P < 0.05$ versus before PTBD

Table 4. Comparison of variables after PTBD in 26 patients with malign obstructive jaundice (age range, 46.5–73.2 years; mean, 59 years) who received external drainage and 60 patients (age range, 50.2–72.5 years; mean 59 years) who received internal and external drainage

	External drainage (n = 26)			Internal/external drainage (n = 60)		
	Before PTBD	5th day after PTBD	Intragroup significance[a]	Before PTBD	5th day after PTBD	Intragroup significance[a]
AST (U/L)	114.5 (67.5–177.0)	72.0 (51.5–117.5)[b]	$P = 0.005$	115.5 (61.2–188.7)	63.0 (42.0–106.0)[b]	$P = 0.0001$
ALT (U/L)	86.5 (68.5–178.5)	70.0 (36.7–95.2)[b]	$P = 0.0001$	90.0 (51.2–199.5)	59.0 (37.0–87.0)[b]	$P = 0.0001$
ALP (U/L)	765 (576–1019)	621 (403–944)[b]	$P = 0.0001$	702 (409–1604)	562 (338–860)[b]	$P = 0.0001$
GGT (U/L)	449 (214–924)	278 (206–493)[b]	$P = 0.004$	384 (266–844)	192 (122–460)[b]	$P = 0.0001$
Total bilirubin (mg/dL)	20.70 (12.64–28.72)	13.25 (7.59–20.96)[b]	$P = 0.0001$	19.50 (10.85–26.45)	13.98 (5.98–20.30)[b]	$P = 0.0001$
Direct bilirubin (mg/dL)	13.10 (6.80–17.89)	7.64 (5.10–14.92)[b]	$P = 0.005$	13.54 (6.06–17.58)	7.32 (3.10–14.51)[b]	$P = 0.0001$
Glucose (mg/dL)	94 (80–115)	96 (79–113)	$P > 0.05$	94 (81–125)	102 (83–135)	$P > 0.05$
Urea (mg/dL)	27 (21–33)	27 (21–40.2)	$P > 0.05$	31 (19–41)	36 (24–66)	$P > 0.05$
Creatinine (mg/dL)	0.81 (0.64–1.03)	0.77 (0.64–0.96)	$P > 0.05$	0.90 (0.74–1.17)	0.95 (0.71–1.28)[c]	$P > 0.05$
GFR-MDRD (mL/min/1.73 m^2)	89.0 (63.7–109.7)	105.5 (71.2–127.2)[b]	$P = 0.038$	82.5 (60.0–105.7)	82.5 (51.5–106.0)[c]	$P > 0.05$
Sodium (mEq/L)	135 (131–136)	133 (131–136)	$P > 0.05$	134 (132–138)	135 (131–137)	$P > 0.05$
Potassium (mEq/L)	3.8 (3.1–4.2)	3.7 (3.2–4.6)	$P > 0.05$	4.1 (3.7–4.4)	4.0 (3.5–4.3)	$P > 0.05$

PTBD, percutaneous transhepatic biliary drainage
Values are medians (1st–3rd quartile)
[a]Wilcoxon signed ranks test
[b]$P < 0.05$ versus before PTBD
[c]$P < 0.05$ versus external drainage

Discussion

Obstructive jaundice has been associated with renal dysfunction and its severity depends on the intensity of biliary obstruction (11, 12). We found renal dysfunction in 25% of our patients before PTBD.

The renal dysfunction associated with obstructive jaundice may be related to either altered systemic hemodynamics or a direct nephrotoxic effect of bile. Extracellular volume depletion was suggested as an important factor influencing renal function in obstructive jaundice. Due to the volume depletion in patients with jaundice, increased endothelin-1 activity is often observed, which leads to renal vasoconstruction and a reduction of GFR (13).

Furthermore, obstructive jaundice is associated with impaired cardiac function. Retained bile acids and liver damage may contribute to negative chronotropic and inotropic effects in an independent manner (14). This, in turn, may play a role in the pathogenesis of "underfilling" of the circulation and susceptibility to acute renal failure in patients with obstructive jaundice.

The effects of obstructive jaundice on the peripheral vasculature of humans and animals include decreased vascular resistance with normal or low blood pressure and an exaggerated hypotensive response to volume depletion. These changes may, in part, be secondary to changes in vascular reactivity (15).

It has been shown that decreased renal perfusion exists either in the presence of normal systemic blood pressure or in the presence of altered systemic hemodynamics. Bile duct ligation (BDL) has been shown to result in an exaggerated vasoconstrictor response of cerebral and renal blood vessels (16). Reduced renal blood flow (RBF), which is common to all BDL species, refers mainly to cortical perfusion. The compromised cortical perfusion observed in animals with BDL was related to the susceptibility of these animals to acute renal failure during anoxia or hypotension (15).

Based on the above-mentioned pathogenetic mechanisms, it appears that the high prevalence of acute renal failure and mortality in patients with obstructive jaundice after surgery, hemorrhage or infection originates extrarenally. Thus, "arterial underfilling" due to reduced peripheral vascular resistance and impaired cardiac function is related to compromised kidney function after these events. It has been reported that with vigilant control of fluid and electrolyte balance, intravenous volume expansion and, if necessary, cardiac evaluation is crucial before PTBD to prevent renal and cardiac complications during and immediately after the procedure.

When the natural excretory route of bile is blocked, the kidney becomes the main excretory organ for the retained bile substances. Given the multiple deleterious effects of bilirubin and bile salts on cell integrity and cell function (17), it is conceivable that the prolonged exposure of the kidney to bile constituents may affect kidney function. Interpretation of the various studies related to changes in GFR or RBF in patients and animals with obstructive jaundice is fraught with major difficulties due to conflicting results (18, 19).

Most studies were unable to demonstrate major renal dysfunction in response to exposure to bile, bile salts or bilirubin. However, it has been reported in animal models that GFR is relatively preserved in spite of reduced renal perfusion, suggesting that obstructive jaundice exerts a modulatory role at the level of the efferent arteriole to increase intraglomerular hydrostatic pressure (20).

Although bile and bilirubin may not be directly toxic, jaundice has been implicated in ischemic injury to the kidney. This evidence incriminates conjugated bilirubin rather than bile acids as the substance that potentiates the anoxic damage to the kidney (21). In our study, multiple logistic regression analysis showed that serum direct bilirubin level is a significant predictor of renal function in patients with obstructive jaundice.

We used MDRD eGFR calculations for renal functional assessment. GFR is the most important clinical function to monitor in renal health and disease. In clinical practice, serum creatinine is the most widely used index for the noninvasive assessment of GFR. Despite its specificity, serum creatinine demonstrates an inadequate sensitivity, particularly in the early stages of renal impairment. It has other significant disadvantages, such as inability to measure renal function impairments of 50% or less (22). Moreover, creatinine is secreted by the proximal tubules, elevating the true GFR by up to 30%. Furthermore, creatinine clearance measurements are of limited value due to inaccurately timed urine collections (23).

The US K/DOQI guidelines and European Best Practice Guidelines state that kidney function should be assessed with GFR estimating formulas such as the Cockcroft–Gault or the MDRD study formula instead of relying on serum creatinine alone (24, 25). Although the MDRD formula was derived from chronic kidney disease patients, Halan et al. concluded that the MDRD formula was the best formula available for estimating GFR in the general population (26).

Several treatment modalities [endoscopic biliary stenting, percutaneous biliary drainage and stenting (PTBD) and surgical biliary bypass] exist to obtain adequate bile duct drainage. Each has its specific merits and drawbacks. The technical success rates of the percutaneous and endoscopic treatments were similar, but therapeutic success was higher in the percutaneous group (27). Endoscopic retrograde cholangiopancreticography (ERCP) is widely used as the primary tool for drainage of distal obstructions, and PTBD is usually reserved for cases where ERCP fails or is not possible. For treatment of patients with proximal obstructions, both ERCP and PTBD are currently used as the primary drainage technique. The choice of technique in these patients depends on specific patient circumstances and on local availability and expertise.

With respect to the preferred route of drainage, internal biliary drainage was found to be superior to external biliary drainage in the reduction of endotoxemia and mortality in some studies. However, other studies demonstrated that external drainage might lead to better recovery of cellular immunity in the short term than internal drainage (4, 28).

Restoration of bile into the normal enterohepatic circulation resulted in reduced rates of endotoxemia (4) and renal impairment (7), and more rapid recovery of cell-mediated immunity (29).

We reviewed patients' data on the fifth and thirtieth days after PTBD. We attempted to determine changes in the short term and after complete decompression. It has been suggested that adequate recovery of hepatic function depends on the duration of obstructive jaundice before decompression (30). A minimum of four to six weeks of drainage was advised. One study showed

that decompression is necessary for at least three weeks before coagulation and hepatic and RES functions begin to improve (31).

In 25 of our patients with initially low eGFR, there were significant increases in eGFR 30 days after PTBD. Although these values were statistically insignificant, such increases were also observed in the overall group, indicating an improvement in renal function after PTBD. This improvement may be due to restoration of bile into the normal enterohepatic circulation as previously suggested (7). Furthermore, we found a significant increase in GFR values estimated on the fifth day in patients in the malignant group who were externally drained due to failure to recanalize the obstruction. There were also insignificant increases in the benign PTBD groups. These findings suggest that the improvement is better with ED in the short term.

In a prospective study, Dawson (32) measured creatinine clearances in 15 jaundiced patients both preoperatively and postoperatively and compared these results with the clearances from 12 nonjaundiced patients undergoing similar operations. In agreement with our results, decreases in creatinine clearance were noted in all of the jaundiced patients and in 10 of the 12 control patients. However, decreases in creatinine clearance were significantly greater in the jaundiced patients and correlated directly with serum bilirubin levels. In a study of nine patients with obstructive jaundice, Evans et al. (33) reported a decrease in postoperative creatinine clearance from a mean of 85 mL/min to 55 mL/min.

Thirty days after PTBD, mortality is >10% in many malignant obstruction studies, but this is largely due to underlying diseases (1). In our study, mortality was 8.49% thirty days after PTBD, and bilirubin levels before PTBD and the estimated GFR value on the fifth day appeared to have a prognostic value.

In conclusion, obstructive jaundice is associated with renal dysfunction, and serum direct bilirubin is a significant predictor of renal function in these patients. Renal function is crucial for prognosis in these patients and is not influenced by the etiology of obstructive jaundice. PTBD results in an improvement in renal function in obstructive jaundice.

References

1. van Delden OM, Laméris JS. Percutaneous drainage and stenting for palliation of malignant bile duct obstruction. Eur Radiol 2008; 18:448–456.
2. Sitges-Serra A. Renal dysfunction and postoperative renal failure in obstructive jaundice. In: Arroyo V, Gine´s P, Schrier P, Rode´s J, eds. Liver and kidney. London: Blackwell, 1999; 79–98.
3. Clairmont P, von Haberer H. Ueber Anurie nach Gallensteinoperatoinen. Mitteilunger aus den Grenzgebieten der Medizen und Chirurgie 1910; 22:159–172.
4. Gouma DJ, Coelho JC, Fisher JD, Schlegel JF, Li YF, Moody FG. Endotoxemia after relief of biliary obstruction by internal and external drainage in rats. Am J Surg 1986; 151:476–479.
5. Gouma DJ, Roughneen PT, Kumar S, et al. Changes in nutritional status associated with obstructive jaundice and biliary drainage in rats. Am J Clin Nutr 1986; 44:362–369.
6. Megison SM, Dunn CW, Horton JW, Chao H. Effects of relief of biliary obstruction on mononuclear phagocyte system function and cell mediated immunity. Br J Surg 1991; 78:568–571.
7. Greve JW, Maessen JG, Tiebosch T, Burman WA, Gouma DJ. Prevention of postoperative complications in jaundiced rats. Internal biliary drainage versus oral lactulose. Ann Surg 1990; 212:221–227.
8. Kone BC, Watson AJ, Gimenez LF, Kadir S. Acute renal failure following percutaneous transhepatic cholangiography. A retrospective study. Arch Intern Med 1986; 146:1405–1407.
9. Levey AS, Coresh J, Greene T, et al. Using standardized serum creatinine values in the modification of diet in renal disease study equation for estimating glomerular filtration rate. Ann Intern Med 2006; 145:247–254.
10. Goodwin SC, Bansal V, Greaser LE III, Stainken BF, McNamara TO, Yoon HC. Prevention of hemobilia during percutaneous biliary drainage: long term follow-up. J Vasc Interv Radiol 1997; 8:881–883.
11. Greve JW, Gouma DJ, Buurman WA. Complications in obstructive jaundice: role of endotoxins. Scand J Gastroenterol Suppl 1992; 194:8–12.
12. Padillo FJ, Cruz A, Briceño J, Martin-Malo A, Pera-Madrazo C, Sitges-Serra A. Multivariate analysis of factors associated with renal dysfunction in patients with obstructive jaundice. Br J Surg 2005; 92:1388–1392.
13. Sarac AM, Aktan AO, Moini H, Bilsel S, Scapa E. Role of endothelin in obstructive jaundice. Dig Dis Sci 1999; 44:356–363.
14. Green J, Beyar R, Sideman S, Mordechovitz D, Better OS. The "jaundiced heart": a possible explanation for postoperative shock in obstructive jaundice. Surgery 1986; 100:14–20.
15. Green J, Better OS. Systemic hypotension and renal failure in obstructive jaundice–mechanistic and therapeutic aspects. J Am Soc Nephrol 1995; 5:1853–1871.
16. Bomzon L, Weinbroum A, Kamenetz L. Systemic hypotension and pressor responsiveness in cholestasis: a study in conscious 3-day bile duct ligated rats. J Hepatol 1990; 11:70–76.
17. Better OS, Berl T. Jaundice and the kidney. In: Suki WN, Eknoyan G, eds. The kidney in systemic disease. 2nd ed. New York: John Wiley & Sons, 1981; 521–537.
18. Finberg JP, Syrop HA, Better OS. Blunted pressor response to anglotensin and sympathomimetic amines in bile duct ligated dogs. Clin Sci 1981; 61:535–539.
19. Hishida A, Honda N, Sudo M, Nagase M. Mechanisms of altered renal perfusion in the early stage of obstructive jaundice. Kidney Int 1980; 17:223–230.
20. Rodrigo Rodrigo R, Avalos N, Orellana M, Bosco C, Thielemann L. Renal effects of experimental obstructive jaundice: morphological and functional assessment. Arch Med Res 1999; 30:275–285.
21. Baum M, Stirling GA, Dawson JL. Further study into obstructive Jaundice and ischemic renal damage. BMJ 1969; 2:229–231.
22. Perrone RD, Madias NE, Levey AS. Serum creatinine as an index of renal function: new insights into old concepts. Clin Chem 1992; 38:1933–1953.
23. Payne RB. Creatinine clearance: a redundant clinical investigation. Ann Clin Biochem 1986; 23:243–250.
24. K/DOQI Clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Am J Kid Dis 2002; 39(Supplement1):S17–S31.
25. European Best Practice Guidelines: Measurement of renal function. Nephrol Dial Transplant 2002; 17(Suppl 7):7–15.
26. Halan S, Astor B, Lydersen S. Estimating glomerular filtration rate in the general population: the second Health Survey of Nord–Trondelag (HUNT II). Nephrol Dial Transplant 2006; 21:1525–1533.
27. Pinol V, Castelis A, Bordas JM, et al. Percutaneous selfexpanding metal stents versus endoscopic polyethylene endoprostheses for treating malignant biliary obstruction: randomized clinical trial. Radiology 2002; 225:27–34.
28. Mizuguchi K, Ajiki T, Onoyama H, Tomita M, Kuroda Y. Short-term effects of external and internal biliary drainage on liver and cellular immunity in experimental obstructive jaundice. J Hepatobiliary Pancreat Surg 2004; 11:176–180.
29. Povoski SP, Karpeh MS, Conlon KC, Blumgart LH, Brennan MF. Association of preoperative biliary drainage with postoperative outcome following pancreaticoduodenectomy. Ann Surg 1999; 230:131–142.
30. Koyama K, Takagi Y, Ito K, Sato T. Experimental and clinical studies on the effect of biliary drainage in obstructive jaundice. Am J Surg 1981; 142:293–299.
31. Kawarada Y, Higashiguchi T, Yokoi H, Vaidya P, Mizumoto R. Preoperative biliary drainage in obstructive jaundice. Hepatogastroenterology 1995; 42:300–307.
32. Dawson JL. Post-operative renal function in obstructive jaundice: effect of mannitol diuresis. BMJ 1965; 1:82–86.
33. Evans HJR, Torrealba V, Hudd C, Knight M. The effect of properative bile salt administration on postoperative renal function in patients with obstructive jaundice. Br J Surg 1982; 69:706–708.