A Comparative Analysis of Nasogastric and Intravenous Fluid Resuscitation in Patients with Malignant Obstructive Jaundice Prior to Endoscopic Biliary Drainage

Kavita Baghel, Saloni Raj, Induja Awasthi, Vishal Gupta, Abhijit Chandra, Rajeshwar Nath Srivastava

Department of Surgical Gastroenterology, 1Physical Medicine and Rehabilitation Center, King George’s Medical University, Lucknow, India

Abstract

Background: An alternative to intravenous is nasogastric fluid administration through normal functioning gut. Though not common, this practice has significance in mass causalties and elective situations. Aim: The study was designed to compare nasogastric and intravenous fluid resuscitation in malignant obstructive jaundice (OJ) and their effect on endotoxemia. Materials and Methods: Sixty patients with malignant OJ undergoing endoscopic biliary drainage were randomized into two groups. A total of 4 l of fluid (Ringer's lactate) was administered to Group A through nasogastric tube and to Group B through intravenous route for 48 h. Vital parameters, serum bilirubin, serum creatinine, creatinine clearance rate, electrolytes, and endotoxinemia were monitored. Results: Significant improvement in blood pressure (Group A, \( P = 0.014 \); Group B, \( P = 0.020 \)) and significant decrease in serum bilirubin level (Group A, \( P = 0.001 \); Group B, \( P < 0.0001 \)) was observed in both groups after resuscitation. Significantly decreased \((P = 0.036)\) post hydration endotoxin level was observed in Group A as compared to Group B. Febrile events were significantly higher \((P = 0.023)\) in Group B as compared to Group A \((6 \text{ vs } 0)\). Electrolyte abnormalities were found more in Group B, however statistically insignificant. Conclusion: In OJ patient undergoing biliary drainage, preoperative fluid resuscitation through nasogastric tube may be helpful in reducing postoperative septic complications and endotoxinemia.

Keywords: Endotoxemia, Fluid therapy, Obstructive jaundice, Parenteral infusion, Resuscitation

Address for correspondence: Prof. Rajeshwar Nath Srivastava, Department of Physical Medicine and Rehabilitation Center, King George’s Medical University, Lucknow - 226 003, Uttar Pradesh, India. E-mail: drrnsrivastava@yahoo.com

Introduction

A typical patient with obstructive jaundice (OJ) is often subjected to an extensive preoperative preparation in an effort to reduce the number of complications encountered in the postoperative period. Obstruction to the flow of bile is due to tumor, stone, stricture, parasites, or other causes showing various alterations in systemic physiology. These changes occur in various body fluid compartments affecting renal hemodynamics, hemostatic mechanism, gastrointestinal barrier, and hepatic and immune function. Renal failure often seen in such patients is of major concern. These patients have significant alterations in the hormonal mechanisms of water and sodium regulation accompanied by a marked depletion of extracellular volume.[3] Uslu et al., have stressed the importance of adequate preoperative hydration for acute renal failure prophylaxis in patients with OJ.[3] Another key event in the pathophysiology of OJ associated complications is endotoxemia of gut origin because of failure of intestinal barrier. This breakage of the gut barrier in OJ is multifactorial involving disruption of the immunological, biological, and mechanical barrier.[3] The mechanisms implicated
Several endotoxins exist in the gastrointestinal tract in which there are abundant gram negative bacteria. Endotoxin in the intestine is not absorbed into the blood circulation normally. Absence of bile in the gut following obstruction promotes the overgrowth and translocation of bacteria and failure to neutralize or degrade endotoxin which leads to endotoxemia.[4] Feeding through enteral route has shown to reduce gut-induced apoptosis, thus maintains gut mucosal integrity during endotoxemia.[9]

Preoperative fluid expansion and careful postoperative fluid balance is needed to correctly deplete extracellular fluid compartment and is the most important factor to prevent renal failure. Patients are also subjected for preoperative biliary decompression that improves postoperative morbidity and septic events. Traditionally, 3-4 l of fluid is administered intravenously per day to maintain good hydration. In a randomized control trial (RCT) by Padillo et al.,[6] 3 l of fluid in a period of 24 h was administered intravenously in the patients with OJ undergoing endoscopic drainage. Fewer surgeons rely on the enteral route alone for adequate hydration probably for the fear that it may not meet the patient’s requirement. The use of gut for fluid resuscitation has thus far not been examined in this group of patients.

As early as 1950, the Surgery Study Section of the National Institutes of Health published a strong recommendation for the use of oral saline solution as standard procedure in the treatment of shock due to burns and other serious injuries in the event of large-scale civilian catastrophe.[7] The development, commercialization, and acceptance of simple-to-place plastic IV catheters along with the rise of new specialties of emergency medicine and trauma surgery led to a disappearance of scientific background supporting enteral resuscitation from contemporary medical consciousness. Apart from mass causalties there are many elective situations, which could serve as the basis for enteral resuscitation, thus far not explored as an option. Preoperative enteral resuscitation in patients with OJ may reduce the risks of renal failure besides decreasing the incidence of gut-induced sepsis. Eventually this could be applicable to all major surgical interventions expected to last for over 3 h to reduce overall postoperative mortality and morbidity. Enteral resuscitation has been evaluated in animal experiments[8] as well as in reasonable large scale trials of burn injured children and adults.[9] These studies appear to have established effectiveness and value of enteral resuscitation using balanced saline solution. Whether the use of gut as a route for resuscitation have role in reducing the number of events of gut-induced sepsis remains unclear. This study has been done largely to compare nasogastric vs intravenous fluid resuscitation in patients with malignant OJ planned for endoscopic biliary drainage.

**Materials and Methods**

The study was approved by institutional ethics committee and was in accordance with Declaration of Helsinki, 1975. All patients fulfilled the following inclusion criteria: Age over 18 years, diagnosed with malignant OJ, serum bilirubin level >10 mg/dl, and fit for endoscopic biliary stent placement and drainage. Critically ill patients (severe cholangitis and acute renal failure) were excluded from the study. Informed consent was obtained from all patients enrolled in the study. Ringer’s lactate (Hartman solution or balanced salt solution) was used for hydration, as it is an isotonic and iso-osmolar solution containing essential additives like calcium, potassium, lactate, sodium, and chloride. It also benefits to maintain the electrolyte balance and expand the plasma volume.

The patients were investigated with complete hemogram, renal and liver function tests, and coagulation parameters. Ultrasound of abdomen was done in all cases. Computed tomography (CT) or magnetic resonance imaging (MRI) of the abdomen was also performed as per the complexity of the case. After confirming malignant OJ, all patients were listed for endoscopic biliary stent placement and drainage either to reduce the bilirubin levels prior to definitive surgery or as a palliative procedure in unresectable cases. Those with severe cholangitis were excluded from the study. The standard orders on admission were vitamin K, fluid intake, urine output and vital charting. The patients were posted at the next available date for stenting. All patients had a single dose of intravenous third generation cephalosporin 1 h prior to the procedure. The procedure was performed under intravenous sedation. One day prior to the procedure, patients were randomized in one of the two groups through computer generated random allocation table; Group A received hydration through nasogastric tube (polyvinyl chloride Ryle’s tube, Size: 16 FG, length: 105 cm) and Group B received through intravenous route. Patients were allocated into the respective groups before the tube placement to avoid the selection bias. We enrolled 30 patients in each group in the duration of 2 years starting from 1st January 2009-1st January 2011. Current study included only patients who had successful endoscopic biliary drainage. Those with failed attempts were referred for percutaneous biliary drainage and were excluded from the study. All the patients of each groups had a suspected malignant obstruction of the biliary tract.
(carcinoma gallbladder: Eighteen, periampullary cancer: Eight, cholangiocarcinoma: Four). All patients in the study group had a normal functioning gut as assessed by the history of normal bowel movement and presence of bowel sounds. Gastric outlet obstruction was ruled out prior to this procedure clinically, or if suspected by doing an upper gastrointestinal endoscopy. All patients had normal renal parameters and lacked symptoms or signs of cholangitis.

All 30 patients of Group A received preprocedure hydration through a nasogastric tube inserted 1 day prior to the day of procedure and continued on the day of procedure (total 48 h). Complete hemogram, renal function, electrolytes, serum bilirubin, and plasma oxygen saturation (SpO₂) were obtained just before and then after the enteral fluid resuscitation [Table 1]. Pulse rate, blood pressure, temperature, and urine output were recorded every 2 h during the fluid administration. Febrile events were defined as oral temperature >98.4°F with chills and shivering. Any nausea or intolerance to nasogastric tube was recorded.

The other group, Group B had same number of patients with the similar disease (malignant obstruction), severity, and age groups. In this group, IV resuscitation was carried out instead of enteral resuscitation.

We defined enteral resuscitation as gastric infusion though a nasogastric tube in patients who tolerated it without discomfort or significant vomiting. The patients were given Ringer’s lactate solution through the nasogastric tube connected to an intravenous infusion set. A total of 4 l of fluid was given through the enteral route. The feeding was withheld 4 h prior to the procedure and 4-6 h postprocedure till clear bowel sounds could be heard or the patient had passed flatus. In Group B, similarly the total 4 l Ringer’s lactate fluid was given using intravenous route. Peripheral blood was sampled before fluid administration and 72 h after the procedure (stenting) for the measurement of serum endotoxin level (Toxin Sensor TM Chromogenic LAL Endotoxin Assay Kit; GenScript USA).

### Statistical analysis

Paired or unpaired Student’s t-test and chi-square test were used to compare within and between groups and P-values <0.05 were taken as significant. Statistical Package for Social Science (SPSS) version 13.0 (SPSS Inc., Chicago, IL) was used to analyze the data.

### Results

There were a total number of 60 patients, equally randomized in two Groups; Group A and Group B. The mean age of the patients enrolled in Group A was 48.24 ± 9.33 years (11 male:19 female) and Group B was 50.83 ± 9.46 years (12 male:18 female).

None of the patients enrolled in this study had any evidence of renal failure prior to intervention (starting hydration) and the renal functions remained within normal limits post hydration. No significant difference in urine output was observed between both the groups [Table 2].

During the 48 h of observation no febrile event was observed in Group A, whereas six patients in Group B had febrile events with leukocytosis (15,000-22,000/mm³) although blood culture was negative. All patients with febrile events were provided intravenous broad spectrum antibiotic. Serum level of all the electrolytes were within normal range before starting hydration, whereas postintervention electrolyte derangement was observed in two patients in Group A (K⁺ levels below normal range 3.4 and 3.2 mmol/l) and five patients in Group B (three patients had K⁺ levels below normal range 3.4, 3.2, and 3.1 mmol/l and two patients had altered Na levels 156 and 128 mmol/l).

In both the groups, statistically significant improvement in blood pressure (Group A, P = 0.014; Group B, P = 0.020) and significant decrease in serum bilirubin level (Group A, P = 0.001; Group B, P < 0.0001) was observed after resuscitation. Statistically significant decrease (P = 0.001) in serum endotoxin levels was observed post hydration in Group A. However, no significant difference in the pulse rate, serum creatinine, creatinine clearance rate, serum K⁺ level, and SpO₂ was observed in both the groups [Table 2].

### Table 1: Parameters used as end points to determine the adequacy of resuscitation

| A. Clinical markers of resuscitation                  |
|------------------------------------------------------|
| 1. Heart rate                                       |
| 2. Rate of urine formation                           |
| 3. Blood pressure                                    |
| B. Biochemical markers of resuscitation               |
| 1. Hb, PCV                                           |
| 2. S. urea/creatinine/electrolytes                   |
| 3. Tissue perfusion SpO₂                              |
| 4. Creatinine clearance                              |
| C. Clinical markers of sepsis                        |
| 1. Temperature                                       |
| 2. Pulse, BP, respiratory rate                       |
| 3. Oxygen saturation                                 |
| D. Biochemical markers of sepsis                     |
| 1. Total, differential counts                         |
| 2. Blood culture                                     |

Hb: Hemoglobin; PCV: Packed cell volume; BP: Blood pressure
No complaints of intolerance from the nasogastric tube or episodes of vomiting were observed in Group A; however, those cases that refused or were uncooperative during placement were excluded. There was no need for simultaneous prokinetic or antiemetic medication.

A comparison between the groups is also shown in Table 2. Significant differences were observed in terms of post hydration febrile events ($P = 0.023$) and endotoxin levels ($P = 0.036$). In Group B, endotoxemia was also found to be significantly associated ($P = 0.006$) with febrile events after resuscitation. The groups were otherwise comparable in terms of pulse rate, blood pressure, serum creatinine, creatinine clearance, serum bilirubin, urine output, and electrolyte abnormalities; and no significant difference was observed.

### Discussion

This study compares the outcomes of nasogastric vs intravenous fluid hydration in patients with malignant OJ undergoing endoscopic biliary drainage. Deterioration of renal function was not observed in any patient during the study period. There was significant improvement in blood pressure in all patients after resuscitation, which is an objective reflection of the restoration of extracellular plasma volume towards normal in patients with OJ. Pulse rate decreased following hydration, although not of statistical significance. There was a significant reduction in serum bilirubin levels at the end of study in both the groups. This can be ascribed to successful stenting and subsequent free flow of bile after endoscopic biliary drainage.

There were statistically significantly more febrile events ($P = 0.023$) in the control group undergoing intravenous hydration as compared to the enteral group. It is uncertain whether the reduction in febrile events was due to the use of gut for hydration. Several studies indicate that stimulation of the gastrointestinal tract may modulate immune function and prevent bacterial infection.\[^{5,10,11}\] The presence of leukocytosis in all six

---

**Table 2: A comparison within and between the groups; A: Enteral resuscitation, B: IV fluid resuscitation**

|                       | Group A          | Group B          | $P$-value |
|-----------------------|------------------|------------------|-----------|
| **Pulse (per min)**   |                  |                  |           |
| Pre                   | 96.8±13.86       | 92.4±13.56       | 0.218     |
| Post                  | 94.9±14.46       | 92.6±13.94       | 0.538     |
| $P$-value             | 0.279            | 0.864            |           |
| **BP (mean arterial pressure in mmHg)** |                  |                  |           |
| Pre                   | 83.2±4.91        | 83.1±5.39        | 0.952     |
| Post                  | 85.7±4.21        | 85.7±4.96        | 0.973     |
| $P$-value             | 0.014            | 0.020            |           |
| **Creatinine (mg/dl)**|                  |                  |           |
| Pre                   | 0.83±0.26        | 0.85±0.29        | 0.726     |
| Post                  | 0.86±0.20        | 0.89±0.23        | 0.591     |
| $P$-value             | 0.470            | 0.322            |           |
| **Creatinine CL (mg/dl)** |                |                  |           |
| Pre                   | 88.7±30.68       | 85.7±31.78       | 0.704     |
| Post                  | 82.8±26.45       | 78.3±24.49       | 0.494     |
| $P$-value             | 0.197            | 0.096            |           |
| **Bilirubin (mg/dl)** |                  |                  |           |
| Pre                   | 16.6±3.53        | 16.9±3.23        | 0.741     |
| Post                  | 14.9±2.61        | 14.8±2.45        | 0.866     |
| $P$-value             | 0.001            | <0.0001          |           |
| **Na$^+$ (mmol/l)**   |                  |                  |           |
| Pre                   | 138.4±3.18       | 138.4±3.13       | 1.000     |
| Post                  | 137.6±4.15       | 137.7±5.55       | 0.937     |
| $P$-value             | 0.279            | 0.465            |           |
| **K$^+$ (mmol/l)**    |                  |                  |           |
| Pre                   | 4.05±0.53        | 3.99±0.54        | 0.665     |
| Post                  | 3.96±0.36        | 3.89±0.45        | 0.508     |
| $P$-value             | 0.279            | 0.177            |           |
| **SpO$_2$ (mmHg)**    |                  |                  |           |
| Pre                   | 95.3±2.24        | 94.9±2.82        | 0.515     |
| Post                  | 95.1±2.84        | 95.1±2.58        | 0.898     |
| $P$-value             | 0.705            | 0.511            |           |
| **Endotoxin (EU/ml)** |                  |                  |           |
| Pre                   | 0.66±0.16        | 0.65±0.17        | 0.744     |
| Post                  | 0.59±0.14        | 0.62±0.35        | 0.036     |
| $P$-value             | 0.001            | 0.673            |           |
| **Urine output (ml/day)** | 2472.4±465.13    | 2384.4±521.00    | 0.535     |
| Pre                   | 0                | 6                | 0.023     |
| Post                  | 2                | 5                | 0.423     |

Student’s unpaired $t$-test and chi-square test was used to test the difference between and within the groups. $P$-value <0.05 is considered as statistically significant.

(Pre: Preprocedure; Post: Postprocedure). BP: Blood pressure; CL: Clearance
febrile cases of Group B and none in Group A may be related to the enteral resuscitation modulated immune function and prevention of bacterial infection.

The incidence of electrolyte abnormalities were least in the Group A (enteral resuscitation, 2) and most in Group B (intravenous resuscitation, 5), however statistical significance (P = 0.423) was not observed. An oral (gastric) bolus would enter the circulation over time, while an intravenous bolus might cause acute hemodynamic overload. Rosenthal and Tabor in 1943 reported that equal volumes of intravenous saline or plasma were often less effective than enteral administration of saline. Use of better fluid solutions through enteral route can minimize the acute electrolyte and volume changes, which are often seen with the use of intravenous fluids.

Patients with OJ, especially when exposed to the additional stress of an invasive diagnostic or therapeutic procedure, are prone to septic complications and renal dysfunction contributing to high morbidity and mortality. The key event in the pathophysiology of OJ-associated complications is endotoxemia of gut origin because of intestinal barrier failure. The common organisms found to be involved in gut related sepsis are Escherichia coli, Salmonella, Shigella, Enterobacteriaceae, and Pseudomonas. The levels of endotoxins are normally increased in OJ and the same was observed in our study in all the patients prior to randomization. After intervention, endotoxin levels decreased in both the groups, although the decrease was statistically significant (P = 0.001) in Group A (Table 2).

In elective situations also, resuscitation is often required and recommended before major surgery. Patients of OJ in particular are potentially hypovolemic and run an increased risk of postoperative renal failure. Also OJ promotes bacterial translocation in gut, leading to endotoxemia and increased postoperative septic complications. In fact the risk of resecting a cholestatic liver may be even greater than a cirrhotic liver for similar reasons. We encounter many patients of OJ requiring surgery where liver also has to be resected (as this is a high incidence belt for cancer gallbladder). Gut-induced sepsis may also be a problem for all major surgeries exceeding 3 h duration. Preoperative enteral fluid resuscitation may be potentially beneficial in all such patients in reducing postoperative septic complications and multiorgan failure and needs to be investigated further.

Further, we found no published clinical studies on enteral resuscitation in traumatic or hemorrhagic hypovolemia. Also there is no published data on the use of gut for preoperative resuscitation in an elective setting. Patients with burns are the only group where this form of resuscitation has been used clinically, though largely forgotten now. We have identified multiple reports in the preMedline literature prior to 1966, and non-English speaking literature that describe enteral resuscitation in animal and human experiments. Some recent reasonable large scale trials of burn injured children and adults have also established the effectiveness and value of enteral resuscitation using balanced saline solutions.

With established benefits of enteral feeding, nasogastric tube insertion should be preferred to provide nutrition in hospital wards and intensive care. Nasogastric tube is cheap, safe, and easily inserted by paramedical person. Enteral resuscitation can be applied by layperson without other specialized medical devices or vascular catheters. Specific solutions and administration regimens including pharmacological agents and perhaps special nasogastric tubes will permit safe implementation of enteral resuscitation in a controlled fashion. Though enteral hydration has shown better outcomes, its reflection in terms of postoperative antibiotic requirement and hospital stay needs to be investigated further.

Feeding by the enteral route is more physiologic than that by the parenteral route by maintaining gut mucosal integrity and reducing bacterial translocation, endotoxemia, and gut-induced sepsis. In contrast, enteral resuscitation has its own limitations. Several of the clinical trials state that enteral resuscitation was not effective when there is “centralization of the circulation” or “peripheral vascular collapse”. Enteral resuscitation is likely to be contraindicated in severe shock when intestinal blood flows as to prevent any significant fluid absorption. Also enteral resuscitation cannot be practiced in a nonfunctional gut or a vomiting patient with gastric outlet obstruction.

Conclusion

In patients with malignant OJ undergoing endoscopic biliary drainage, adequate preoperative fluid resuscitation using enteral route may be helpful in reducing postoperative septic complications and endotoxia. However, its role in the reduction of renal failure and the incidence of gut-induced sepsis remains uncertain.

Acknowledgement

Authors wish to thank Council of Science and Technology, UP, Govt. of Uttar Pradesh, Lucknow, India for financial assistance.

References

1. Sitges-Serra A, Carulla X, Piers C, Martinez-Rödenas F, Franch G, Pereira J, et al. Body water compartments in patients with obstructive jaundice. Br J Surg 1992;79:553-6.
2. Uslu A, Nart A, Colak T, Aykas A, Yüzbasioglu MF, Hidir K, et al. Predictors of mortality and morbidity in acute obstructive jaundice: Implication of preventive measures. Hepatogastroenterology 2007;54:1331-4.

3. Assimakopoulos SF, Scopa CD, Vagianos CE. Pathophysiology of increased intestinal permeability in obstructive jaundice. World J Gastroenterol 2007;13:6458-64.

4. Yang YJ, Shi JS, Xie SM, Zhang DT, Cui BS. Effect of different drainage procedures on levels of serum endotoxin and tumor necrosis factor in patients with malignant obstructive jaundice. Hepatobiliary Pancreat Dis Int 2003;2:426-30.

5. Alschter KT, Phang PT, McDonald TE, Walley KR. Enteral feeding decreases gut apoptosis, permeability, and lung inflammation during murine endotoxemia. Am J Physiol Gastrointest Liver Physiol 2001;281:G569-76.

6. Padillo FJ, Briceño J, Cruz A, Chicano M, Naranjo A, Vallejo J, et al. Randomized clinical trial of the effect of different drainage procedures on levels of serum endotoxin and tumor necrosis factor in patients with malignant obstructive jaundice. Hepatobiliary Pancreat Dis Int 2003;2:426-30.

7. Surgery Study Section of NIH. Saline solution in treatment of burn shock. Public Health Rep 1950;1317-20.

8. Rosenthal SM, Tabor H. Experimental chemotherapy of burns and shock. Public Health Rep 1943;58:1429-36.

9. Fox CL. Oral sodium lactate in treatment of burns and shock. JAMA 1944;124:207-12.

10. Welsh FK, Ramsden CW, MacLennan K, Sheridan MB, Barclay GR, Guillou PJ, et al. Increased intestinal permeability and altered mucosal immunity in cholestatic jaundice. Ann Surg 1998;227:205-12.

11. Pain JA, Cahill CJ, Bailey ME. Perioperative complications in obstructive jaundice: Therapeutic considerations. Br J Surg 1985;72:942-5.

12. Deitch EA, Sittig K, Li M, Berg R, Specian RD. Obstructive jaundice promotes bacterial translocation from the gut. Am J Surg 1990;159:79-84.

13. Scopa CD, Kourelas S, Tsamandas AC, Spiliopoulos T, Alexandrides T, Filos KS, et al. Beneficial effects of growth hormone and insulin-like growth factor I on intestinal bacterial translocation, endotoxemia, and apoptosis in experimentally jaundiced rats. J Am Coll Surg 2000;190:423-31.

14. von Eiff C, Jansen B, Kohnen W, Becker K. Infections associated with medical devices. Pathogenesis, management and prophylaxis. Drugs 2005;65:179-214.

15. Jiang KY. Evaluation of resuscitation of shock with oral fluids in dogs with 30% superficial second degree burn. Zhonghua Zheng Xing Shao Shang Wai Ke Za Zhi 1988;4:288-91.

16. Jackson D, Cason JS. The treatment of burns shock with oral hypotonic saline-bicarbonate solutions. In: Wallace AB, Wilkinson AW, eds. Research in Burns. London: E. Livingstone, Ltd; 1966;61-70.

17. Venter M, Rode H, Sive A, Visser M. Enteral resuscitation and early enteral feeding in children with major burns — effect on McFarlane response to stress. Burns 2007;33:464-71.

18. Scopa CD, Kourelas S, Tsamandas AC, Spiliopoulos T, Alexandrides T, Filos KS, et al. Beneficial effects of growth hormone and insulin-like growth factor I on intestinal bacterial translocation, endotoxemia, and apoptosis in experimentally jaundiced rats. J Am Coll Surg 2000;190:423-31.

19. Michell MW, Oliveira IM, Kinsky MP, Vaid SU, Herndon DN, Kramer GC. Enteral resuscitation of burn shock using World Health Organization oral rehydration solution: A potential solution for mass casualty care. J Burn Care Res 2006;27:29-35.

20. Ghatak T, Samanta S, Baronia AK. A new technique to insert nasogastric tube in an unconscious intubated patient. N Am J Med Sci 2013;5:68-70.

21. Wilson BJ, Stirman JA. Initial treatment of burns. J Am Med Assoc 1960;173:509-16.

How to cite this article: Baghel K, Raj S, Awasthi I, Gupta V, Chandra A, Srivastava RN. A comparative analysis of nasogastric and intravenous fluid resuscitation in patients with malignant obstructive jaundice prior to endoscopic biliary drainage. North Am J Med Sci 2013;5:523-5.

Source of Support: Council of Science and Technology, UP, Govt. of Uttar Pradesh, Lucknow, India. Conflict of Interest: None declared.

Announcement

“QUICK RESPONSE CODE” LINK FOR FULL TEXT ARTICLES

The journal issue has a unique new feature for reaching to the journal’s website without typing a single letter. Each article on its first page has a “Quick Response Code”. Using any mobile or other hand-held device with camera and GPRS/other internet source, one can reach to the full text of that particular article on the journal’s website. Start a QR-code reading software (see list of free applications from http://tinyurl.com/yzlh2tc) and point the camera to the QR-code printed in the journal. It will automatically take you to the HTML full text of that article. One can also use a desktop or laptop with web camera for similar functionality. See http://tinyurl.com/2bw7fn3 or http://tinyurl.com/3ysr3me for the free applications.