<table>
<thead>
<tr>
<th>項目</th>
<th>内容</th>
</tr>
</thead>
</table>
| 症例報告 | 資料の詳細は次の通り。院内診療部における腎乳頭の組織の変化を報告し、その病理学的所見について考察。
| 共著者 | Takeshima, Yoshitaka; Nakachi, Atsushi; Shimoji, Hideaki; Miyazato, Hiroshi; Muto, Yoshihiro |
| 引用 | 琉球医学会誌 = Ryukyu Medical Journal, 20(3): 137-139 |
| 発行年 | 2001 |
| URL | http://hdl.handle.net/20.500.12001/3463 |
| 著作権 | 琉球医学会 |
Renal papillary necrosis: An autopsy case report and a brief review of the literature

Yoshitaka Takeshima, Atsushi Nakachi, Hideaki Shimoji, Hiroshi Miyazato and Yoshihiro Muto

The First Department of Surgery, Faculty of Medicine, University of the Ryukyus, Okinawa, Japan

(Received on November 22, 2000, accepted on April 24, 2001)

ABSTRACT

An 83-year-old Japanese woman was admitted to our surgical department for persistent rectal bleeding on July 11, 1997. The patient had received radiotherapy for uterine cervical cancer 3 years previously. Her performance status was grade 3, due to bilateral femoral fracture. Sixteen months after radiotherapy, the patient demonstrated slight rectal bleeding. Since her rectal bleeding was persistent and intractable, she underwent proctectomy with colostomy. The resected rectum histologically showed severe endoarteritis and she was diagnosed to have a radiation proctitis. She developed paralytic bowel obstruction on the 6th post operative day (POD) and renal dysfunction with signs of systemic inflammatory response syndrome on the 10th POD. On the 25th POD, she developed a sudden onset of hematemesis and hypovolemic shock. An emergency endoscopic examination showed bleeding from multiple shallow ulcers in the stomach (acute gastric mucosal lesion). Simultaneously the patient presented with clinical manifestations of sepsis. The patient finally expired due to multiple organ failure. The autopsy revealed clinically insidiously progressing bilateral renal papillary necrosis, which was considered to be the primary cause of her death. Microscopically multiple extensive necrosis areas were observed in the renal papillae. Ryukyu Med. J., 20(2)77–80, 2001

Key words: renal papillary necrosis, sepsis, radiation-associated proctocolitis

INTRODUCTION

The clinical spectrum of renal papillary necrosis (RPN) is wide and variable. The disease may follow a chronic smouldering course and behave much like pyelonephritis. On the other hand, an acute type of the disease may be characterized by a sudden onset, with fever, chills, pain in the renal areas, leukocytosis, and possibly hematuria. Malaise, prostration, uremia and shock may all ensue, eventually resulting in death.

This paper describes a case of acute renal papillary necrosis in the post operative course. Characteristically, the patient was a bed-ridden aged woman with a history of pelvic radiation for uterine cervical cancer. This report embodies our experience of an interesting RPN case.

CASE REPORT

An 83-year-old Japanese woman was admitted to our surgery department for further examination of an intractable rectal bleeding in July 11, 1997. Her past history revealed that she had received 56 Gy of radiotherapy for uterine cervical cancer (stage IIb) 3 years earlier at our hospital. Her performance status was grade 3, and she was almost completely bed-ridden due to the onset of a bilateral femoral fracture 2 years earlier. Sixteen months after radiotherapy, the patient demonstrated rectal bleeding. At a local hospital, a barium enema study revealed the affected rectum to have a lack of distensibility and haustration. Colonoscopy revealed diffuse oozing in the rectum. Based on these findings, she was diagnosed to most probably have radiation proctocolitis. Her rectal bleeding gradually worsened and subsequently she developed anemia (Hb 6.0 g/dl). Due to her deteriorating condition, she was referred to our department to undergo surgery. Since the rectal bleeding continued, the patient underwent proctectomy and colostomy. Grossly, the resected rectum was erosive and histologically revealed severe endoarteritis. As a result, she was diagnosed to have radiation proctocolitis.

On the sixth post operative day (POD), she developed paralytic bowel obstruction with sequential renal failure and a clinical manifestations of systemic inflammatory response syndrome (SIRS). She had taken analgesic including diclofenac sodium (Voltaren) daily 1-2 tablets for one week. Additional medications at the time of post operative course included antibiotics, diuretic and sedative. On the 25th POD, she developed a
sudden onset of hematemesis and subsequently hypovolemic shock. Emergency endoscopy revealed multiple erosion and trench ulcers in the stomach (acute gastric mucosal lesion: AGML). Two days later the bleeding stopped, but the renal function gradually worsened, and serum urea nitrogen and creatinine levels abnormally increased to 136 mg/dl and 3.34 mg/dl, respectively. Repeated urine and blood cultures grew Pseudomonas aeruginosa and she was also diagnosed to have sepsis. The patient was treated with antibiotics which proved to be ineffective. A computed tomography (CT) scan revealed low density areas in the renal medullae (Fig. 1). Unfortunately, we were not able to correctly diagnose the symptoms to be RPN. Moreover, ultrasonography revealed no obvious abnormal findings, a repeated urinalysis showed red blood cells that were too numerous to count. The patient's condition continued to deteriorate and she developed respiratory failure on the 65th POD and eventually expired.

An autopsy revealed bilateral RPN (Fig. 2) together with pneumonia and gastrointestinal bleeding, which was considered to be a result of the multiple organ failure (MOF). Microscopically multiple extensive necrosis areas were observed in the renal papillae (Fig. 3).

**DISCUSSION**

RPN results from ischemia of the renal papilla in a variety of diseases including diabetes mellitus and analgesic nephropathy. Most cases of RPN described in the literature are generally associated with diabetes mellitus. Other etiologic factors of this disease include obstructive uropathy, analgesic abuse, chronic alcoholism, pyelonephritis, sickle cell hemoglobinopathy, renal allografts, liver disease, and other causes. This case showed no medical evidence of diabetes mellitus, ureteral obstruction or analgetic abuse. She had taken analgesics and other medications in the post operative course, but their doses were within the normal range. As a result, pyelonephritis alone was considered to be the main contributory factor leading to RPN.

Although the exact mechanism of RPN development remains uncertain, the basic pathophysiologic process appears
to be ischemic necrosis as shown in various experimental models. In the clinical conditions associated with RPN, a vascular impairment is a prominent feature of the underlying disease, particularly since RPN tends to occur in older patients in which arteriosclerotic changes are more common. This patient was bed-ridden, elderly and had undergone a proctectomy and was urinating using a balloon urethral catheter. These factors taken together in our case seemed favorable for the development of pyelonephritis which eventually led to RPN.

A major radiological evidence for the diagnosis of RPN obtained from an intravenous urogram in certain situations may be diagnostic. Ultrasonography may also be suggestive of RPN. A CT scan demonstrates small kidney ring shadows in the medullae, contrast filled clefts in the renal parenchyma and renal pelvic filling defects. The most common findings on plain CT are a decreased length of the kidneys, bumpy contours and papillary calcification. Unfortunately, a radiologic diagnosis cannot be made until the changes in the papillary region have progressed to a point where the papillae either shrink or become sequestered in many cases. In our case, we carelessly did not identify RPN in the CT scan findings until the autopsy results revealed RPN.

AGML is induced by sepsis, resulting in hypotension and subsequently contributing to ischemia of gastric mucosa by producing splanchnic vasoconstriction. In our case, the patient initially developed intractable rectal bleeding which resulted in hypotension. Secondly she was urinating using a balloon urethral catheter so as to check the urinary volume postoperatively. The balloon catheter urination in our patient may have been caused by pyelonephritis which later led to SIRS. She then developed SIRS and AGML after the onset of renal failure. Unfortunately, we did not accurately diagnose RPN based on the clinical and image findings.

Regarding the treatment of RPN in this case, the treatment of choice remains urgent surgical intervention. Conservatively, an approach using a balloon ureteral catheter tamponades the papilla bleeding and necrosis. However, the outcome in such cases is often poor, and consequently the RPN still tends to show a poor prognosis. In conclusion, when patients present with smouldering septic disease, the presence of RPN should be included in the differential diagnosis because the clinical picture of RPN varies greatly. By diagnosing RPN in a timely manner, the optimal treatment regimen can thus be selected for such patients.

ACKNOWLEDGEMENTS

The authors thank Mr. Brian Quinn for his comments on this manuscript and also express our gratitude to the pathologists of our University Hospital for their excellent autopsy report.

REFERENCES