Acute renal cortical necrosis (ARCN) is a rare cause of acute renal failure characterized by necrosis of the renal cortex with sparing of the renal medulla. Owing to poor clinical condition during the early period of ARCN in most cases, it does not allow renal biopsy which is a gold standard for diagnosis. Therefore, noninvasive diagnostic modalities have been tried and, among them, contrast-enhanced computed tomography (CT) is found to provide characteristic findings. We present a 31-year-old female of meningococcemia with septic shock, who developed oliguria and was diagnosed as ARCN by the specific CT findings without renal biopsy.

CASE REPORT

A 31-year-old female visited the emergency room due to fever and progressive epigastric pain in the past 24 hours. The patient was a hepatitis B carrier, a heavy smoker and an alcoholic. At emergency room, dyspnea and unstable hemodynamic condition (respiratory rate: 30/min, blood pressure: 70/40 mmHg) developed several hours later. Generalized petechiae with cyanotic change of skin turgor and oliguria were also noted. Laboratory data showed coagulopathy with thrombocytopenia (Fibrinogen: 92.1 mg/dL, FDP: 532.9 ug/mL, D-D diamer test: 85131.6 ng FEU/mL, PT >80 sec, aPTT >200 sec, INR>8.0, Platelet=18,000 / uL) and impaired renal function (BUN/Cr=14/2.1 mg/dL). Under the impression of septic shock with coagulopathy, contrast enhanced abdominal CT scan was performed to exclude the possibility of intraabdominal infection. CT scan incidentally showed nonenhancing renal cortex, enhancing medulla and no excretion of contrast medium, which were compatible with ARCN (Fig. 1a-1c). The patient’s condition remained unstable. Bradycardia and asystole occurred at night on the same day. Cardiopulmonary cerebral resuscitation (CPCR) was performed and spontaneous circulation recovered. However, bradycardia and asystole recurred again. Electrocardiogram did not recover despite CPCR. The patient’s family decided to discharge her from further medical treatments due to poor condition. Two samples of blood culture revealed Neisseria meningitides infection three days later.

DISCUSSION

ARCN is thought to result from arterial spasm of the cortical vessels. Generally, the corticomedullary junction and the subcapsular rim of cortex are spared. This is due to relative sparing of medullary arterioles and collateral circulation to the subcapsular cortex from capsular vessels [1-2]. The disease is frequently associated with obstetric complication, especially abruptio placentae.
several cases have been described unrelated to pregnancy, including sepsis with shock, snake bite, poisoning, etc. [3]. The pathophysiology of renal cortical necrosis may be related to ischemia due to constriction of small intracortical vessels, which may be related to toxins, circulating hormones or neural stimuli [4]. The Shwartzman reaction is regarded as a coagulopathy with massive intravascular coagulation and tissue necrosis and the kidney is the site of predilection [5]. Bacterial endotoxemia causing a generalized Shwartzman reaction has been described with E. Coli [6], S. aureus [7] and Neisseria meningitidis [5]. We postulated that our presented case developed gram-negative sepsis and endotoxemia due to Neisseria meningitides which led to Shwartzman reaction and subsequent ARCN. The prognosis of ARCN is mainly dependent on the underlying disease. Shwartzman reaction with disseminated intravascular coagulation may result from a wide variety of insults. Among them neoplasm, septic shock and severe trauma are lethal regardless of their effect on renal function. Other causes, such as obstetrical complications, carry a relatively good prognosis including recovery of renal function in some cases [8].

ARCN is known to have a specific appearance on contrast enhanced CT scan. It was initially described by Jordan et al. [2]. The three CT features diagnostic of this entity include (a) enhancement of the medulla, (b) nonenhancement of the renal cortex, and (c) lack of excretion of contrast medium into the collecting system. Cortical rim sign of enhanced subcapsular area on contrast-enhanced CT scan suggested the sparing of the most peripheral region of cortex, which was initially described by Goergon et al. [4]. Kim et al. [9] modified and combined those four characteristic findings to result in a diagnosis of ARCN. The CT imaging features of our presented case matched the findings described by Jordan et al. [2] but lack of enhancement in subcapsular rim. In the normal anatomy, the interlobular arteries have a radically oriented course toward the renal sur-

Figure 1. a. Axial non-contrast enhanced CT scan revealed no calcification of both kidneys. b. Axial and c. coronal reconstruction plans of contrast enhanced CT scan demonstrated bilateral renal medullary enhancement and lack of cortical enhancement. There were lack of peripheral rim enhancement in bilateral renal cortex.
face as perforating arteries. Three capsular arteries form a capsular network that anastomoses freely with perforating arteries and other retroperitoneal arteries [10]. The result of lack of subcapsular rim enhancement could be due to an anatomic variant of capsular artery, arterial spasm or massive intravascular coagulation in our presented case. The sonographic findings of ARCN have been described as hypoechoic cortical tissue adjacent to the renal capsule [11]. However, the sonographic appearance is variable and may be deceptively normal. The lack of a readily available renal contrast agent to evaluate renal perfusion and excretion is a major limitation of sonography at this time [2]. Magnetic resonance (MR) imaging findings (especially T2- and gadolinium enhanced T1 weighted images) are quite similar to those of contrast enhanced CT scan while most patients with such condition are usually critically ill, therefore difficult to be closely monitored during the procedure. Hence, MR may be unsuitable [9, 12-13]. The selective renal angiography defined a very unusual and diffuse abrupt termination of vascularity at the interlobar and arcuate artery levels, which resulted in irregular scalloped perfusion defects of the renal cortex [14]. The angiography is generally believed to be a more invasive procedure when compared to other imaging procedures.

The definite diagnosis of ARCN depends on histologic examination of the renal tissue. However, the unstable hemodynamic clinical status with coagulopathy always precludes renal biopsy, especially during the early period of renal cortical necrosis [3]. ARCN could be diagnosed in the initial period with its specific imaging findings [9, 15-16].

In conclusion, contrast-enhanced CT scan could be an important diagnostic modality in the initial phase of renal cortical necrosis. Three CT features of ARCN are (a) nonenhancement of the renal cortex, (b) enhancement of the renal medulla, and (c) lack of excretion of contrast medium to the collecting system.

REFERENCES

急性腎皮質壞死在電腦斷層之特異表現

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因急性腎臟皮質壞死而造成腎衰竭較少被描述及報告。急性腎臟皮質壞死的特徵為腎臟皮質壞死，但是腎臟髓質依舊完好。通常急性腎臟皮質壞死病人臨床情況都很差，腎臟切片證實有其困難性，因此其他非侵犯性檢查更顯得重要。注射顯影劑的電腦斷層檢查，對於急性腎臟皮質壞死有其特異性發現。我們報告一位31歲的女性，因腦膜炎雙球菌感染造成敗血病，並進而引發急性腎臟皮質壞死。