Acta Cardiol Sin 2016;32:502–505
Case Report
doi: 10.6515/ACS20151013C

A Case of Metanephric Adenoma and Acute Myocardial Infarction

Ruzicic Dusan,[1] Kovacevic Relja,[2] Mirkovic Marija,[1] Radovanovic Jelena,[3] Krstevska Vesna,[3] Terzic Milijana,[1] Pantelic Vladimir,[4] Matic Irena[1] and Hrncic Dragan[5]

Metanephric adenoma (MA) is a rare neoplasm that acounts for 0.2% of adult renal neoplasms. MAs are typically discover incidentally during detailed examinations for nonspecific symptoms such as abdominal or flank pain, hematuria, fever and palpable abdominal mass. Additionally, polycythemia has occasionally been reported as well. Herein we describe a case of metanephric adenoma which was an incidental finding in the course of a clinical autopsy in a patient with complete AV block and polycythemia. Histologically, the tumor was composed of small and uniform tubular structures reminiscent of renal tubuli, without signs of cellular atypia and pleomorphism. Such tumor histomorphology was consistent with the diagnosis of metanephric adenoma.

Thrombosis is a common complication of polycythemia that often causes death. Polycythemia with an increasing number of blood cells causes hyperviscosity and, in 20-40% of cases, lethal thrombosis or hemorrhage. Hyperviscosity and coronary artery disease in our patient caused acute myocardial infarction with the subsequent rupture of posterior left ventricle wall and hemopericardium.

Key Words: Acute myocardial infarction • Metanephric adenoma • Polycythemia

INTRODUCTION

Metanephric adenoma (MA) is a rare neoplasm accounting for 0.2% of adult renal neoplasms.[1,2] MA generally occurs in adults, particularly in middle-aged women (male:female 1:2-6).[3] Benign renal epithelial kidney tumors are subclassified into metanephric adenoma, cortical/papillary renal cell adenoma and renal oncocytoma. MA is the rarest renal tumor, however, and with growing awareness and recognition it is becoming increasingly more prevalent.[4,5] Nonetheless, it is not well-recognized by either clinicians or pathologists.[6] MAs are incidentally discovered during detailed examinations for nonspecific symptoms such as abdominal or flack pain, hematuria, and fever and palpable abdominal mass.[3] Polycythemia has occasionally been reported as well.[3,4,6] Sonographic or radiological features have no typical presentation in this lesion.[6] The association of hyperechogenicity on ultrasound with hyperdensity on unenhanced computed tomography has been reported as characteristic for benign adenomatous kidney tumors.[6]

We herein describe a case of metanephric adenoma which was an incidental finding in the course of a clinical autopsy.

Received: May 27, 2015 Accepted: October 13, 2015
[1]Department of Cardiology, General Hospital Valjevo; [2]Insitute of Pathology, Faculty of Medicine, Belgrade; [3]Department of Pathology; [4]Department of Surgery, General Hospital Valjevo; [5]Insitute of Physiology, Faculty of Medicine, Belgrade.
Address correspondence and reprint requests to: Dr. Dusan Ruzicic, Department of Cardiology, General Hospital Valjevo, Naselje Oslobodioci Valjeva 6/2, 14000 Valjevo, Serbia. Tel: +381652223006, +38114222300; E-mail: ducaruzicic@yahoo.com

CASE REPORT

A 64-year-old man was admitted to the Emergency Room of the Regional General Hospital in Valjevo complaining of fatigue and vertigo. His medical and family history were unremarkable. The patient's physical examination showed moderate arterial hypertension (160/80

mmHg). Electrocardiography (ECG) registered complete AV block with right bundle branch block (RBBB) and heart frequency of 45 beats per minute (Figure 1A). Thereafter, a temporary pacemaker was implanted. A subsequent blood workout showed a high red blood cell (RBC, 6.57 $\times$ 10^{12}/L, normal reference 3.60-6.0 $\times$ 10^{12}/L), white blood cell (WBC) count (15.1 $\times$ 10^{9}/L, normal reference 4.0-10.0 $\times$ 10^{9}/L), low platelet count (59 $\times$ 10^{9}/L, normal reference 150-450 $\times$ 10^{9}/L), and increased levels of hemoglobin (HGB, 205 g/L, normal reference 120-160 g/L), lactate dehydrogenase (LDH, 338 U/L, normal reference 125-243 U/L), and C-reactive protein (100.3 mg/L, normal reference 0.1-8.2 mg/L). Prothrombin time (PT, 23.2s, normal reference 11.1-16.1s) and activated partial thromboplastin time (APTT, 59.6s, normal reference 27-35 s) were prolonged and fibrinogen level was low (1.1 g/L, normal reference 2.0-4.8 g/L), and erythropoietin level was high 34 mU/ml (normal range 4 up to 24 mU/ml). Additionally, hematuria was further noted. Abdominal ultrasound failed to show any significant pathology. The day after admission, the patient suddenly lost consciousness; ECG showed ST elevation in the leads of the inferior myocardial wall (Figure 1B), while urgent heart ultrasound revealed pericardial effusion. Cardiopulmonary resuscitation to cardiopulmonary-cerebral resuscitation (CPCR) was done but without result, and clinical autopsy was indicated.

The patient's autopsy revealed that the cause of death was a massive pericardial hemorrhage and cardiac tamponade due to the acute myocardial infarction of the posterior/inferior left ventricle wall and its subsequent rupture, in the setting of severe coronary atherosclerosis (Figure 2A, B). The incidental finding was a tumor of the left kidney.

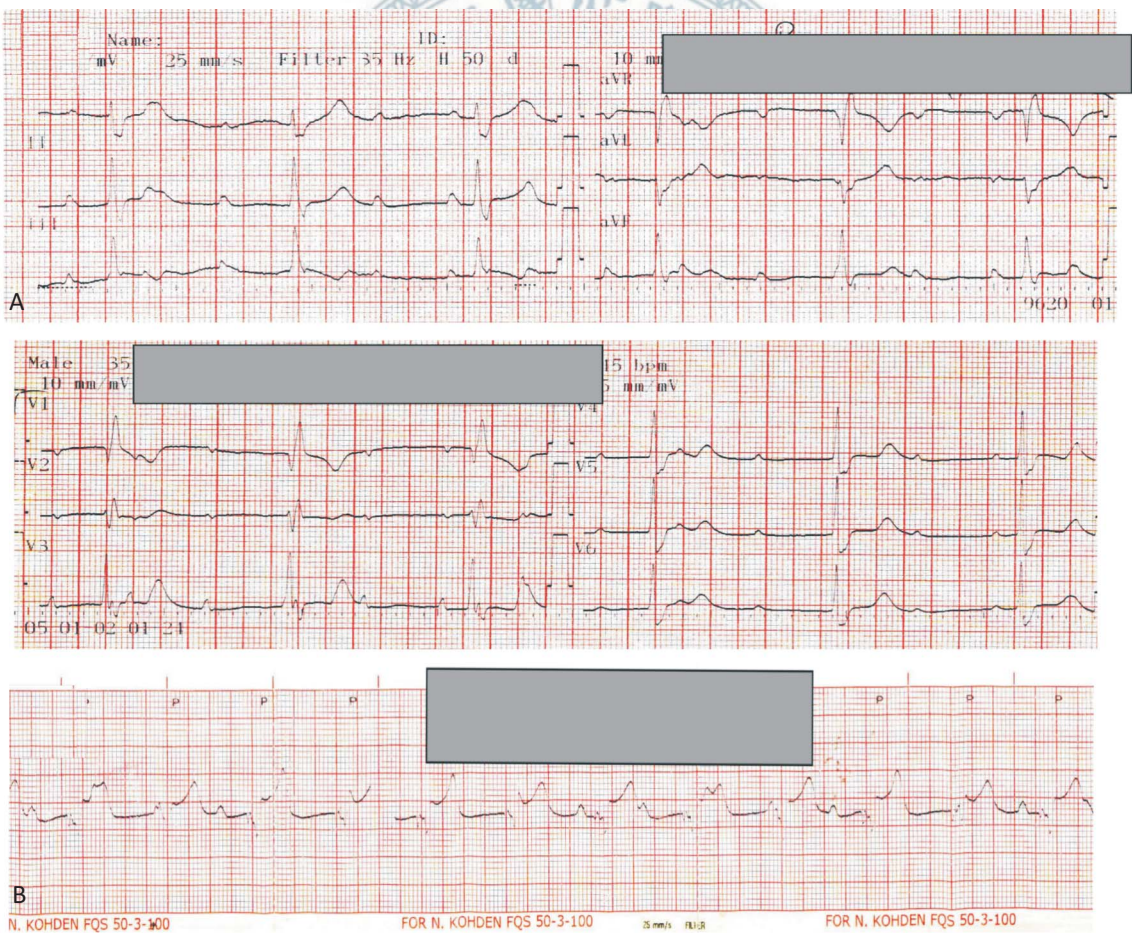

Figure 1. *(A) Electrocardiography (ECG) of our patient on admission in ER. Note complete AV block with right bundle branch block (RBBB) and heart frequency of 45 beats per minute. (B) Acute ST elevation myocardial infarction of inferior wall (leads D2, 3) in ECG on our patient. Grey blocks overlap the patient personal data.*

Histologically, the tumor was composed of small and uniform tubular structures reminiscent of renal tubuli, without signs of cellular atypia and pleomorphism. Such tumor histomorphology was consistent with the diagnosis of metanephric adenoma (Figure 2C, D).

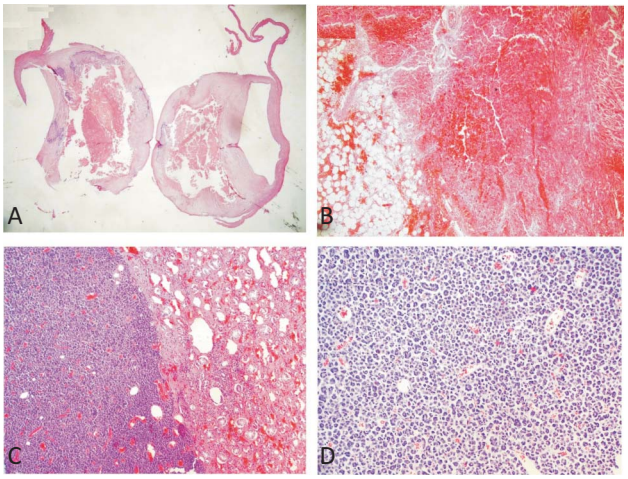

Figure 2. *(A) Serial sections through right coronary artery (RCA) show partially calcified fibroatheromatous lesions containing fibrous cap separating necrotic core from reduced arterial lumen. (B) Samples of left ventricle wall in vicinity of rupture site with blood dissecting through necrotic myocardium and epicardial fat. (C) MA is composed of small uniform round acini and tubular structures with scant stroma; the acini are lined by bland, uniform, hyperchromatic cells with minimal cytoplasm; there is no nuclear atypia, mitoses nor necrosis. (D) Tumor is well-circumscribed, but not encapsulated and directly about the surrounding renal parenchyma.*

DISCUSSION

MA is derived from the metanephric blastema.[1] Histologically, MA is composed of tightly packed uniform, small epithelial cells with small regular nuclei, a basophilic cytoplasm, a high nuclei-to-cytoplasm ratio and mitotic figures and no pleomorphism.[1-3] Cell culture supernatant shows that MA produces a high concentration of erythropoietin, granulocyte-macrophage colony stimulating factor (GM-CSF), granulocyte colony stimulating factor (G-CSF), interleukin-6 (IL-6) and interleukin-8 (IL-8).[8] Among the possible clinical findings, the incidence of polycythemia with MA is remarkable.[6] Davis et al. showed that polycythemia was found in 6/50 cases (12%), making MA the renal tumor with the highest incidence of polycythemia.[3,6] In comparison, renal cell carcinoma had a 1-6% incidence of polycythemia.[6]

Erythropoietin is a glycoprotein produced by the renal cortex in response to hypoxia, which is a major regulator of erythropoiesis and induces erythrocyte differentiation.[8,9] Increased erythropoietin levels have frequently been described in patients with renal tumors,[9] yet the mechanism of production remains unclear. Erythropoietin may be produced by tumor cells or by normal cells in response to the relative hypoxia induced by tumor.[9]

Thrombosis is a common complication of polycythemia, which frequently causes death.[10] Polycythemia with an increasing number of blood cells causes hyperviscosity and, in 20-40% of cases, lethal thrombosis or hemorrhage. Hyperviscosity in our patient and coronary artery disease caused the acute myocardial infarction with the subsequent rupture of posterior left ventricle wall and hemathopericardium.

We assume that this renal tumor (MA) and polycythemia promoted the development of the organ-associated coagulopathy (low number of platelets and low level of fibrinogen, prolonged prothrombin time and activated partial thromboplastin time). We also assumed that the polycythemia observed in our patient was secondary to erythropoietin-secreted MA. Low platelet count (59×10^9/L) and high erythropoietin level (34 mU/ml) speak in favor of this assumption since primary polycythemia is commonly accompanied by thrombocytosis and very low levels of erythropoietin.[11]

So, in this case, the undiagnosed tumor had been a contributing factor for myocardial infarction that facilitated pericardial hemorrhage, cardiac tamponade and, eventually, the lethal outcome.

Knowing that patients with metanephric adenoma have a good prognosis after total nephrectomy or tumor resection with kidney preservation,[6] and bearing in mind this case, we can conclude that early diagnosis and treatment of these tumors is essential.

REFERENCES

1. Sharma PK, Tiwari P, Singh JP, Bera MK. Metanephric adenoma: a case report and review of the literature. *Saudi J Kidney Dis Transpl* 2013;24(5):1027-30.

2. Amin MB, Amin MB, Tamboli P, et al. Prognostic impact of histologic subtyping of adult renal epithelial neoplasms: an experience of 405 cases. *Am J Surg Pathol* 2002;26:281-91.
3. Kuroda N, Tol M, Hiroi M, Enzan H. Review of metanephric adenoma of the kidney with focus on clinical and pathobiological aspects. *Histol Histopathol* 2003;18:253-7.
4. Birgisson H, Einarsson GV, Steinasrsdottir M, Jónasson JG. Metanephric adenoma. *Scand J Urol Nephrol* 1999;33:340-4.
5. Bouzourene H, Blaser A, Francke ML, et al. Metanephric adenoma of the kidney: a rare benign tumour of the kidney. *Histopathology* 1997;31:485-6.
6. Zhang LJ, Yang GF, Shen W, Lu GM. CT and ultrasound findings of metanephric adenoma: a report of two cases and literature review. *The British Journal of Radiology* 2011;84:e51-4.
7. Brisigotti M, Cozzutto C, Fabbretti G, et al. Metanephric adenoma. *Histol Histopathol* 1992;7:689-92.
8. Yoshioka K, Miyakawa A, Ohno Y, et al. Production of erythropoietin and multiple cytokines by metanephric adenoma results in erythrocytosis. *Pathol Int* 2007;57:529-36.
9. Erslev AJ, Caro J. Physiologic and molecular biology of erythropoietin. *Med Oncol Tumor Pharmacother* 1986;3:159-64.
10. Hermanns B, Handt S, Kindler J, Füzesi L. Coronary vasculopathy in polycythemia vera. *Pathol Oncol Res* 1998;4(1):37-9.
11. Michiels JJ, Bernema Z, Van Bockstaele D, et al. Current diagnostic criteria for the chronic myeloproliferative disorders (MPD) essential thrombocythemia (ET), polycythemia vera (PV) and chronic idiopathic myelofibrosis (CIMF). *Pathol Biol* (Paris) 2007; 55(2):92-104.