

# Trousseau's Syndrome Caused by Bladder Cancer

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**Abstract** An 82-year-old woman was admitted due to disturbance of consciousness. Six months previously, she had been diagnosed with nested variant of urothelial carcinoma. Magnetic resonance imaging showed multiple bilateral small acute cerebral infarctions, corresponding to Trousseau's syndrome. Best supportive care was chosen and she finally died. This is the sixth case of Trousseau's syndrome induced by bladder carcinoma and the first case of Trousseau's syndrome in a patient with nested variant of urothelial carcinoma. This unique case adds one more cause to the list of documented etiologies of Trousseau's syndrome.

**Keywords:** bladder cancer, multiple cerebral infarctions, Trousseau's syndrome, nested variant of urothelial carcinoma

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## 1. Introduction

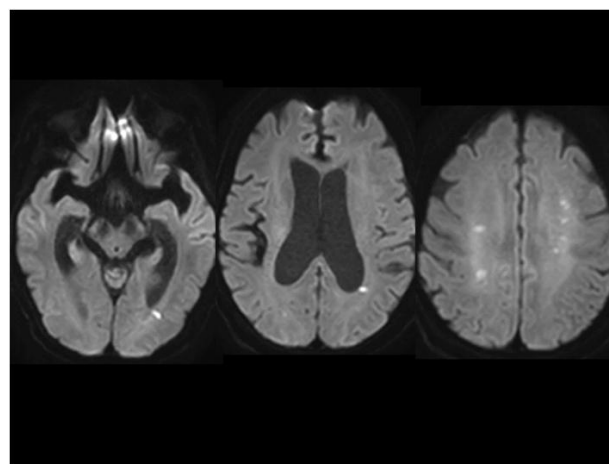
Trousseau's syndrome is a form of cancer-associated thrombosis, and the second leading cause of death in cancer patients, after death from cancer itself. [1] The risk of a venous thromboembolism in patients with cancer is 4- to 7-fold higher than that in individuals without cancer [1]. Trousseau's syndrome is the consequence of multiple pathophysiological mechanisms that apparently contribute to the hypercoagulability associated with cancer [2]. The causes of the hypercoagulability range from exaggerated fluid-phased thrombosis that is dependent on prothrombotic agents such as tissue factor to a platelet, endothelium-based selectin-dependent microangiopathy associated with mucin-producing carcinomas along with thrombin and fibrin production, and/or genetic pathways within tumor cells that might trigger these thrombotic phenomena [2]. Malignancies most commonly associated with Trousseau syndrome include those of the pancreas, lung, prostate, stomach, and colon, with pancreatic cancer accounting for 50% of all cases [3]. However, there have been only five reports on Trousseau syndrome induced by bladder cancer. We report the sixth case of a female patient in whom Trousseau's syndrome was caused by bladder cancer.

## 2. Case Report

An 82-year-old woman was admitted for disturbance of consciousness (Glasgow Coma Scale E1V1M4). She had a history of osteoporosis, pubic fracture, compression fracture of the first lumbar vertebra, and bladder carcinoma. Six months previously, she had been admitted

another hospital for rehabilitation after experiencing compression fracture of the first lumbar vertebra. Whole body CT showed right hydronephrosis and thickening of the bladder wall. Cystoscopy and biopsy revealed a nested variant of urothelial carcinoma.

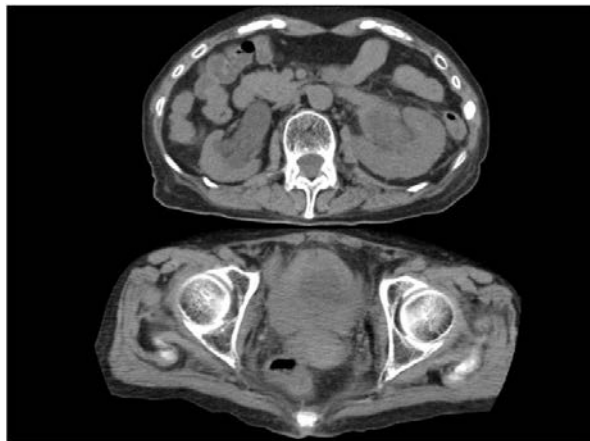
On examination, the patient's blood pressure was 151/84 mmHg and her heart rate was 81 beats per minute, with a regular pulse, normal heart sound, and no carotid bruits. A physical examination revealed left-dominant motor paresis (Manual Muscle Test was 2 of 5 at every extremity). Computed tomography (CT) of the head was performed but did not show stroke. Cerebral magnetic resonance imaging (MRI) showed multiple bilateral small acute cerebral infarctions (Figure 1).



**Figure 1.** Magnetic resonance imaging (MRI) of the brain (Diffusion-weighted images showed fresh multiple infarctions)

At the time of admission, the laboratory examination results revealed elevated fibrin degradation products (FDP), 21.28  $\mu\text{g/mL}$  (normal,  $<5$ ) and D-dimer,

17.24 µg/mL, (normal, <1). A complete blood count revealed a white blood cell count of 12,900, and hemoglobin 9.9. Other coagulation study results were within the normal ranges: prothrombin time international normalized ratio (PT-INR), 0.92 (normal, 0.8-1.2) and activated partial thromboplastin time (APTT), 27.3s (normal, 21-40).



**Figure 2.** Abdominal computed tomography (CT) (CT revealed bilateral hydronephrosis and a huge mass in the pelvis)

Serum biochemistry revealed the following: albumin 6.4 g/dL (normal, 4.1-5.1), blood urea nitrogen (BUN) 110.2 mg/dL (normal, 8.0-20.0), creatinine 8.76 mg/dL (normal, 0.46-0.79), C-related protein 2.35 mg/dL (normal, <0.15), sodium 127 mmol/L (normal, 138-145), potassium 6.5 mmol/L (normal, 3.6-4.8), chlorine 89 mmol/dL (normal, 101-108), and C-related protein 2.35 (normal, <0.14). The serum levels of carcinoembryonic antigen (CEA) and carbohydrate antigen 19-9 (CA19-9) were 2.6 ng/mL (normal, < 5.0) and 58.5 U/mL (normal, <37.0), respectively. Electrocardiography showed no signs of atrial fibrillation. Echocardiography and ultrasonic evaluation of the carotid artery showed no abnormal

findings. Whole body CT was performed and showed bilateral hydronephrosis and a tumor in the pelvis (Figure 2). Her family selected palliative care and she died 30 days after admission to our hospital.

### 3. Discussion

This is the sixth reported case of Trousseau's syndrome induced by bladder carcinoma and the first reported case of Trousseau's syndrome with a nested variant of urothelial carcinoma. We summarized the previous cases of Trousseau's syndrome induced by bladder carcinoma in Table 1 [4,5,6,7,8], including the present case. The average age was 63 years (range, 38-82). The youngest patient had a history of neurogenic bladder, which is a well-known risk factor for bladder carcinoma. The male to female ratio was 1 to 2. The histological types were as follows transitional cell carcinoma in (2 cases), squamous cell carcinoma (2 cases), lymphoma-like high-grade urothelial carcinoma (1 case) and nested variant of urothelial carcinoma (1 case). All cases died within one year, with the exception of one case with an unknown outcome. The reason why Trousseau's syndrome is associated with a poor prognosis in bladder cancer may be because it becomes impossible to administer chemotherapy due to post-renal failure induced by bladder cancer itself, in addition to the advanced stage of the cancer at the time of the diagnosis. The histological type in most cases of Trousseau's syndrome is adenocarcinoma, especially mucin-producing carcinoma. However, adenocarcinoma of the urinary bladder arising from the urothelial lining is an uncommon malignant neoplasm, accounting for 0.5–2.0% of all malignant vesical tumors [9]. This is why bladder cancer is rare in Trousseau's syndrome. In the present case, inflammation, dehydration, and/or hypercoagulability associated with the cancer might have been involved in the development of Trousseau's syndrome.

**Table 1.** Previous case reports on bladder cancer accompanied by multiple cerebral infarctions

Author	Age	Sex	Symptom	Site	Histological type	Treatment	Outcome
Orrell	67	Male	Achromatopsia	Bilateral occipital lobe	Transitional cell carcinoma	Heparin	Dead (3 months)
Kato	38	Female	Visual field defects, lower limb weakness	Bilateral cerebral white matter	Transitional cell carcinoma	Heparin	Dead (3 months)
Kayukawa	71	Female	Right hemiplegia	Left cerebral white matter, basal ganglion	Squamous cell carcinoma	Radiotherapy	Dead (8 months)
						warfarin	
Ando	46	Male	Disturbance of confidence	Bilateral cerebral white matter	Lymphoma-like high-grade urothelial carcinoma	Not described	Dead (3 weeks)
Kimura	75	Female	Language impairment	Right cerebral white matter, left cerebellum	Squamous cell carcinoma	Heparin	Dead (56 days)
			left hemiplegia				
Present case	82	Female	Disturbance of confidence	Bilateral cerebral white matter	Nested variant of urothelial carcinoma	none	Dead (30 days)

### 4. Conclusion

This is the sixth reported case of Trousseau's syndrome induced by bladder carcinoma and the first reported case of Trousseau's syndrome with a nested variant of urothelial carcinoma. This unique case adds one more cause to the list of documented etiologies of Trousseau's syndrome.

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