*Case Report*

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# Anton's syndrome as a presentation of Trousseau syndrome involving the bilateral optic radiation

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**Abstract**

Anton's syndrome is a rare neuropsychiatric syndrome that is characterized by cortical blindness and anosognosia with visual confabulation, but without global cognitive impairment. We herein report a rare case of Anton's syndrome as a presentation of Trousseau syndrome involving the bilateral optic radiation. The patient had been diagnosed with gallbladder cancer 2 months previously, and he was admitted to the hospital with confusion and quadriplegia. He was found to be blind, but denied any visual impairment and demonstrated visual confabulation despite evidence of his blindness. These signs were consistent with a diagnosis of Anton's syndrome. Brain computed tomography (CT) and magnetic resonance imaging revealed infarcts in the bilateral temporo-parieto-occipital junction with hemorrhagic transformation, mainly involving the bilateral optic radiation. The presence of gallbladder cancer with peripheral metastasis on abdominal CT, as well as markedly increased tumor markers and D-dimer levels, supported the presence of cancer-related hypercoagulability and the diagnosis of Trousseau syndrome.

**Keywords**

Anton's syndrome, cortical blindness, stroke, tumor, optic radiation, Trousseau syndrome

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## Introduction

Anton's syndrome is a rare form of visual anosognosia that is characterized by the denial of a loss of vision; it is associated with confabulation in the context of

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obvious visual loss and cortical blindness.[1] Patients with Anton's syndrome are either partially or completely blind. However, they strongly believe that they can see what they cannot, and they behave and talk as if they were sighted. Although some causes of cortical blindness may potentially result in Anton's syndrome,[1–4] cerebrovascular disease—particularly bilateral occipital infarction—is the most common cause.[1] However, to the best of our knowledge, there have been no previous reports of Trousseau syndrome as the cause of Anton's syndrome. Herein, we report a case of Anton's syndrome as a presentation of Trousseau syndrome involving the bilateral optic radiation.

## Case report

A 79-year-old man was admitted to hospital because of confusion and quadriplegia that had persisted for 5 hours. Two months prior to admission, he had been diagnosed with diabetes mellitus and gallbladder cancer with multiple metastases, but had not undergone surgery. A neurological examination revealed blurred consciousness, vague speech, right central facial paralysis, and quadriplegia (the Medical Research Council (MRC) scale for muscle strength: left, grade II; right, grade IV). Brain computed tomography (CT) showed cerebral hemorrhage in the left temporal–occipital junction (Figure 1a). The patient was initially given treatment for dehydration as well as symptomatic therapy. The following day, he became clear-minded and alert. He was found to be blind, but denied any visual impairment despite evidence of his blindness. He was unable to see fingers in front of him. When asked to name the "torch, key, or mobile phone" in the doctor's hand, he named some unrelated objects. He lacked the blink-to-threat reflex and visual pursuit ability, with his head always turning toward voices. His pupils were equal and pupillary reflexes were intact. He was suspected to have Anton's syndrome (cortical blindness plus visual anosognosia).[1] Brain CT reexamination revealed infarcts in the bilateral

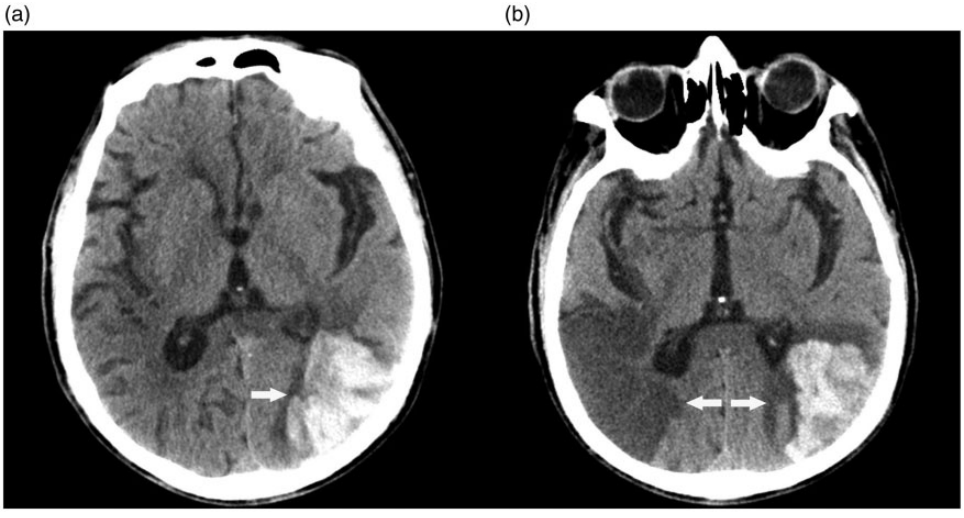


**Figure 1.** Brain computed tomography (CT) images. (a) Brain CT on admission showed hemorrhage (arrow) in the left temporal–occipital junction. (b) Brain CT reexamination the following day revealed infarcts (arrows) in the bilateral temporo-parieto-occipital junction with hemorrhagic transformation.

temporo-parieto-occipital junction with hemorrhagic transformation (Figure 1b). Abdominal CT showed gallbladder cancer with peripheral metastasis. The patient's markedly increased tumor markers (CA-50: 227.24 U/mL, CEA: 18.46 ng/mL, CA-199: 674.30 U/mL, CA-125: 551.29 U/mL, CA-211: 28.24 ng/mL, CA-724 > 300.00 IU/mL, CA-242 > 200.00 U/mL, and NSE: 17.38 ng/mL) and D-dimer levels (4.370 and 6.940 μg/mL respectively) in two consecutive sessions supported the presence of cancer-related hypercoagulability. Further brain magnetic resonance imaging (MRI) 2 weeks after admission revealed subacute infarcts in the right hemicerebrum and cerebellum, as well as in the left temporo-parieto-occipital junction, with hemorrhagic transformation on diffusion-weighted imaging (Figure 2a, partially not shown). No vascular abnormalities were observed using magnetic resonance angiography (Figure 2b). The patient's state was consistent with a diagnosis of Trousseau syndrome[5] presenting with Anton's syndrome. He continued to receive dehydration therapy, blood sugar control, and symptomatic treatment. At discharge, his right visual field had partially recovered and he was aware of his blindness. His muscle strength of the extremities (MRC scale: left, grade III; right, grade $IV^{+}$) had also markedly improved. The patient continued to receive rehabilitation treatment after discharge.

## Discussion

Our patient developed Anton's syndrome following damage to the bilateral optic radiation, which was caused by Trousseau syndrome. It has not been previously reported that Trousseau syndrome can present as Anton's syndrome. Anton's syndrome is a rare neuropsychiatric syndrome that is characterized by cortical blindness and anosognosia with visual confabulation, without global cognitive impairment.[1–4]

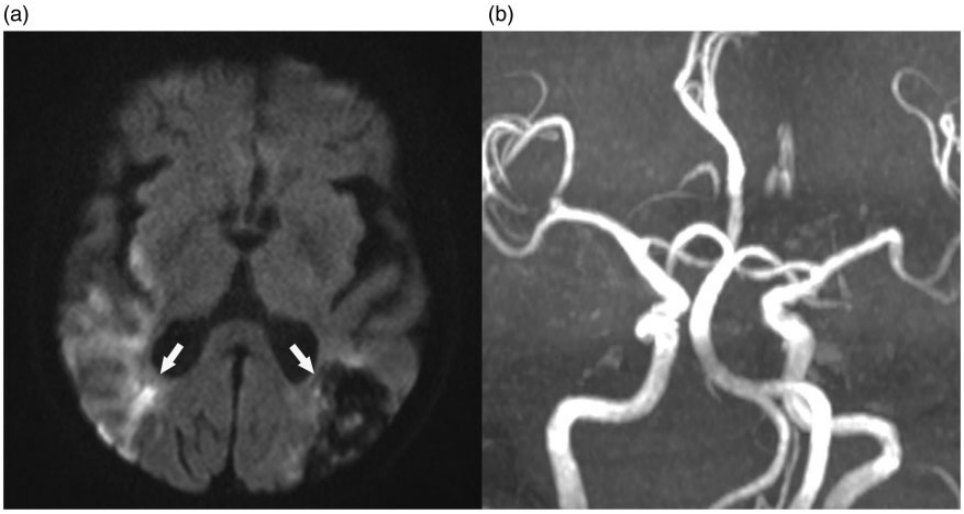


**Figure 2.** Brain magnetic resonance imaging (MRI) scans. (a) Brain MRI 2 weeks after admission revealed hyperintense lesions (arrow) in the right hemicerebrum (mainly in the right temporo-parieto-occipital junction) and cerebellum (partially not shown), and hypointense core lesions with high signal edge (arrow) in the left temporo-parieto-occipital junction on diffusion-weighted imaging. (b) No vascular abnormalities were observed with magnetic resonance angiography.

Anton's syndrome (also called Anton–Babinski symptom) was named by Gabriel Anton and Joseph Babinski, and is characterized by confabulation as a result of the denial of vision loss.[6] That is, patients with Anton's syndrome behave as if they can see despite obvious visual loss. For patients with cortical blindness, especially those who deny the presence of blindness, Anton's syndrome should be suspected.

Anton's syndrome is generally caused by damage to the occipital lobe, extending from the primary visual cortex into the visual association cortex. Cerebrovascular disease, particularly bilateral occipital infarcts or hemorrhage, is by far the most common cause.[1,7] Other rare causes of Anton's syndrome include brain tumors,[8] trauma,[9] neuroleptospirosis,[2] and decompression illness.[3] The patient described in the present report had Anton's syndrome caused by Trousseau syndrome, which to the best of our knowledge has not been documented previously.

The neuropsychological mechanisms of classical Anton's syndrome remain controversial. As suggested by Anton himself, damaged visual cortices may be effectively disconnected from functioning areas.[6] In the absence of input, functioning speech areas often confabulate a response. As well as bilateral visual cortical damage, Anton's syndrome can also be caused by lesions in the precortical visual pathway (such as in the bilateral lateral geniculate bodies, posterior limbs of the internal capsules, and optic radiation) as well as in the corpus callosum.[8,10] It has been reported that a patient with Anton's syndrome, caused by a giant frontal fossa meningioma involving the corpus callosum, became aware of visual deficits after the removal of the bifrontal meningioma.[4] This finding further indicates that precortical lesions of the visual pathway can also be responsible for Anton's syndrome. The focus that was responsible for our case was located in the bilateral optic radiation, rather than the bilateral occipital lobes; Anton's syndrome involving this site is extremely rare. We have previously reported a case of bilateral posterior watershed infarction caused by atrial fibrillation and third-degree atrioventricular block, presenting with Anton's syndrome. The responsible focus in this previous patient was also the bilateral optic radiation.[10]

In summary, the causes of Anton's syndrome are variable. Trousseau syndrome can be a rare cause of Anton's syndrome. Our case further indicates that extensive lesions of the bilateral optic radiation may lead to Anton's syndrome.

## Declaration of conflicting interest

The authors declare that there is no conflict of interest.

## Ethical statement and informed consent

The requirement for ethics approval to publish this case report was waived by the Institutional Review Board of the Affiliated Hefei Hospital of Anhui Medical University and the Second Affiliated Hospital of Anhui Medical University. Written informed consent to publish the case was provided by the patient's son.

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