Unusual association of diseases/symptoms

Unmasking aortic dissection in patients of transient global amnesia: case report and diagnostic algorithm for the emergency department

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It is not unusual for doctors working in the emergency department (ED) to diagnose a benign syndrome of transient global amnesia (TGA). It is rare that the TGA patient is in danger of life because of a 'forgotten' acute aortic dissection (AAD). The present article deals with one of such rare cases of association between TGA and AAD, which presented laboratory findings never reported before. A practical diagnostic algorithm has also been drafted to help ED physicians facing such a puzzling situation.

BACKGROUND

Transient global amnesia (TGA) is a syndrome of uncertain aetiology characterised by a sudden anterograde memory loss and repetitive questioning.¹ The prognosis is generally considered benign. Thus, psychological support for TGA patients and their relatives has been suggested as the cornerstone of a therapeutic approach within the emergency department (ED).²⁻³

Nevertheless, in literature only a few cases of patients diagnosed with TGA are reported, who suffered from a more severe and life-threatening condition: acute aortic dissection (AAD).⁴⁻⁸

The new case of association between AAD and TGA presented in this article highlights that the time has probably come for ED doctors to become aware and prepared to face such a tricky condition, since a prompt and correct diagnosis is mandatory to ensure patient survival.

Therefore, a diagnostic algorithm for a new approach to TGA patients admitted to the ED is also provided.

CASE PRESENTATION

In the evening, a 64-year-old Caucasian man was admitted to ED suffering from evident memory loss and repetitively lamenting chest pain. His wife reported that her husband went out to work early in the morning and came back home in the afternoon without any memory of what had happened during the day.

His medical history was unremarkable.

On examination, the patient's level of consciousness was normal. He could easily answer to questions about his identity (name, date and place of birth, present job) and the identity of his family members; he was also aware of the time of the day and of the date, and he was aware to be in the ED of his referral hospital. Moreover, he could clearly recall the events of the previous day and the moment when he woke up but was unable to remember the rest of

his day after leaving his house early in the morning. There were no signs of trauma or seizure-like activity. The neurological exam was completely normal except for anterograde amnesia. Indeed, the patient could not remember three easy objects mentioned by the physician for more than 1 min. Furthermore, he complained about chest pain, repeating, almost obsessively, 'I've got chest pain'. Blood pressure was 150/80 mm Hg in both arms. Palpation of peripheral pulses did not show any deficit. ECG, chest x-ray and blood gas analysis were normal. Cardiac myonecrosis markers were negative. Neutrophilic leukocytosis (18,000 cells/mm³ neutrophils 88.9%), elevated concentration of serum creatinine (1.6 mg/dl) and a marked increase of D-dimer levels (4611 ng/ml with a cut-off value of 228) were shown.

A decisive diagnostic clue was given by the patient when he changed his statement in: 'I've got chest... and back pain'. These few more words together with the marked increase of D-dimer levels were a hint for AAD.

INVESTIGATIONS

A spiral CT angiography confirmed the suspected aortic disease. A Stanford type A dissection involving the arch and the thoracic aorta up to the abdominal tract was found (figures 1 and 2). The dissection involved the first section of the subclavian artery on the left and extended to the common carotid artery on the right, sparing the right subclavian artery. Brain CT scan performed without contrast injection was negative.

TREATMENT

The patient underwent urgent surgery and he did not show any intraoperative or postoperative complications. After the intervention, the anterograde amnesia was resolved; whereas a retrograde amnesia about the onset of pain and the following hours spent in the ED emerged.

Figure 1 CT angiography of the thoracic aorta showing the intimal flap in the aortic root extending up to the descending aorta (arrows).

Figure 2 CT angiography of the dissected aortic arch showing an intimal flap separating the two lumens (arrow).

Figure 3 Diagnostic algorithm for TGA association with AAD.

OUTCOME AND FOLLOW-UP

Six months later at a new clinical evaluation he showed no sequelae. Retrograde amnesia about the whole day of the episode was still present.

DISCUSSION

To our knowledge, literature reports only three other similar cases of an apparently benign TGA syndrome (without any additional neurological sign) that was instead concealing Stanford type A AAD.^{4 6 7} Two further cases of AAD associated with TGA could be found in conjunction with other neurological symptoms – such as anisocoria or motor deficit – alongside with a case of association between Stanford type B AAD and TGA.

The relationship between AAD and TGA is still uncertain and it is too early to have a clear clinical picture of such an unusual association. On the other hand, it is mandatory for ED physicians to uncover a life-threatening condition such as AAD ‘hidden’ by TGA.

In our opinion, the first thing to investigate when examining a TGA patient in ED is whether anyone witnesses the onset of amnesia attack (see figure 3 – diagnostic algorithm). We recommend asking relatives, colleagues or any other possible witness about the onset of the episode. In the past, sudden paleness, syncope, acute chest or back

pain immediately preceding the amnesic disorder have been described as clues of AAD.^{4-6 8}

In case it is not possible to rely on witnesses – as it happened in the present case – the unique source of information is the patient himself. The evident difficulties in dealing with amnesic patients should not deceive or discourage. TGA patients probably cannot recall the onset of symptoms (or even pain), but they are able to describe a continuous chest pain or a variation in the perception of pain when it occurs.

After taking a detailed medical history, the second step is to carefully check the presence of the following clinical and laboratory signs that were described in our and/or in previously reported cases:

1. hypotension/hypertension or difference in blood pressure between the two arms (refs ⁴⁻⁶, our case)
2. syncope, paleness^{4 6 8}
3. chest/back/lumbar pain (refs ^{4 5}, our case)
4. fever⁸
5. leukocytosis (refs ^{7 8}, our case)
6. increase of D-dimer levels (our case)
7. unexplained increase of serum creatinine levels (refs ^{7 8}, our case).

If none of the above findings is registered, it is correct to suspect a pure TGA syndrome. In any case, the

Learning points

- ▶ An apparently benign pure TGA syndrome may hide AAD.
- ▶ The first step in dealing with TGA patient is to collect information about the onset of amnesic attack.
- ▶ Amnesic patients are still a reliable source of information and doctors should keep on carefully listening to their repetitive statements even during the amnesic attack.
- ▶ Increased D-dimer levels should lead to suspect AAD as well as the following reported important signs that are:
 - (A) chest/back/lumbar pain
 - (B) hypotension/hypertension or difference in blood pressure between arms
 - (C) fever
 - (D) leukocytosis
 - (E) unexplained increase of serum creatinine levels.

patients must also fulfil all the other important diagnostic criteria, such as: (1) preservation of consciousness and identity; (2) absence of accompanying focal neurological symptoms or epileptic features; (3) no history of recent head injury or active epilepsy; (4) a complete remission of symptoms within 24 h and, of course, the presence of

anterograde amnesia appeared unexpectedly in front of witnesses.⁹

On the contrary, if any of the above abnormalities is present, the possibility of AAD has to be considered and performance of an urgent angio-CT scan of the aorta must be considered.

Competing interests None.

Patient consent Obtained.

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