Psychotic symptoms associated with superior vena cava syndrome consecutive to methamphetamine abuse

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INTRODUCTION
Superior Vena Cava Syndrome (SVCS) is a manifestation arising from compression or obstruction of Superior Vena Cava (SVC), characterized by edema of face, neck, and upper extremities as well as collateral venous distension of neck and anterior chest wall [1].

Mostly caused by mediastinal malignancies, SVCS can be secondary to SVC thrombosis with a prevalence of 1-5% [2].

Amphetamine-type stimulants can cause systemic effects [3] including vascular conditions, psychotic and manic episodes [4].

Here, we describe a previously unrecognized association between psychosis and SVCS in a patient with history of Methamphetamine abuse.

CASE REPORT
A 24 year old man presented acute agitation, aggressive behavior. He showed self-neglect, delusional speech, auditory and cenesthetic hallucinations, inappropriate uncontrolled laughter, perseveration and misrecognition.

Physical examination: face and neck edema and voluminous collateral blood-flow network of the anterior chest wall veins, consistent with SVCS.

Chest CT-scan (Figure 1) revealed SVC thrombosis with distended collateral vasculature and stenosis of SVC. Intramuscular Heparin (Tinzaparine: 14000 UI/d) was delivered to prevent further complications.

Coagulation tests were normal, thrombophilia screening negative.

No evidence for cryoglobulinemia, antiphospholipid syndrome neither ANCA-vasculitis.

Myeloproliferative neoplasm, celiac, Biermer and Behçet diseases were ruled out.

Paraneoplastic etiology was rejected: Thoracic CT-scan and full body PET-scan found no evidence of neoplasia.

Brain CT and electroencephalography were normal. Brain SPECT showed mild frontal cortex and anterior cingulum hypo-perfusion.

Methamphetamine abuse history.

Schizophrenia was diagnosed according to Mini-DSM [5]. PANSS total score was 133.

The patient received first Risperidone 6 mg/d without success. Olanzapine 20 mg/d was stopped because of weight gain and Aripiprazole 15 mg/d discontinued after Akathisia.

Eventually, symptoms resolved with Clozapine 350 mg/d facilitating history-taking: acute rheumatic fever at 11. First drug abuse at 18: occasional cannabis and cocaine abuse. At 22, high doses of sniffed crystal Methamphetamine (ice), over 10 days.

He remembers clearly the sudden swelling of upper limbs, face, neck and trunk the day after and persistent ever since.

Despite several radiological interventions, thrombus was unremovable. Tinzaparine was relayed by oral anticoagulant: Rivaroxaban.

Figure 1: Chest contrast-enhanced (CT) scan with frontal plane bi-dimensional reconstruction. The arrow is pointing to the thrombus with stenosis of Superior Vena Cava.
DISCUSSION

To our knowledge this is the first case of schizophrenia with deep venous thrombosis, prior to psychotropic medication and without coagulation disorders.

But Amphetamines can induce both psychotic symptoms [5] and venous thrombosis (cerebral [6] or renal [7]).

Here, Methamphetamine was taken in crystal form, supposed to be particularly harmful because of its high potency compared to other forms [8].

The sudden swelling in the patient’s reports highlights the chronological relationship between ice-Methamphetamine and thrombosis. In point of fact, SVCS occurs more abruptly in thrombotic cases than compressive ones, often resulting in medical emergencies [9].

Amongst many complications, Amphetamines have a direct vascular toxicity their neurotoxic effects appear to be due to induction of inflammatory genes in small vessel endothelial cells [10].

In 2005 Hanson and Gottesman [11] proposed that some psychosis resulted of damage to the micro-vascular system in the brain, initiated by genetically influenced abnormal inflammatory processes, in response to environmental factors. Inflamed micro-vessels lose their coupling with astrocytes, leading to deregulated cerebral blood flow, and damaged Blood-Brain Barrier (BBB).

In 2015, Turowski and Kenny [12] suggested that the direct action of Methamphetamine on vascular endothelium induces acute opening of BBB while striatal effects and resultant neuro-inflammatory signaling could lead to its chronic dysfunction.

This case raises the question of a tendency to abnormal inflammatory processes in schizophrenia. The risk of serious vascular complications should be emphasized and prevention developed. Systematic radiological exams (contrast enhanced CT scans f.i) could be prescribed in psychotic patients with Methamphetamine abuse history, to detect thrombosis that normally goes unnoticed.

CONFLICT OF INTEREST

None declared.

REFERENCES


