CEREBRAL VENOUS SINUS THROMBOSIS INDUCED BY CHRONIC ABUSE BY MDMA ("ECSTASY") CASE REPORT

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Background: Cerebral venous sinus thrombosis (CVST) accounts for approximately 1% of all stroke events. Venous hemorrhagic infarction (VHI) presents severe form of clinical symptom and a bad prognostic factor of CVST (1,2). Methylenedioxymethamphetamine (MDMA), commonly known as ecstasy, is a hallucinogenic compound structurally related to amphetamine. Severe neurological toxicity of ecstasy can cause seizures, subarachnoidal hemorrhage, cerebral infarction, intracranial bleeding and rarely CVST (3,4).

Case report: Female, 19 year-old presented to the Emergency Room after serial tonic-clonic seizures with right hemiparesis and divergent strabismus in somnolent state. During last 2 years, she chronically consumed alcohol with benzodiazepines. Subsequently, the data on the chronic abuse of ecstasy on the day of admission, but in unknown doses, was obtained. She did not use oral contraceptives.

The patient had the following vitals: blood pressure 80/60 mmHg, heart rate 130 beats/min, respiratory rate 22 breath/min, afebrile. On admission MultiGnost Test Panel (Urine) analysis was proved to be positive for MDMA. Blood toxicology analysis identified diazepam and nordazepam in pathological concentrations (the day after).

First brain computed tomography scan and angiography (CT/CTA) showed massive intracerebral hemorrhage in the left frontal and parietal lobes (9,5 x 4,5 x 5cm) with a compressive edema and suspected thrombosis of the superior sagittal sinus, but CT angiography was normal (Figure 1-3).

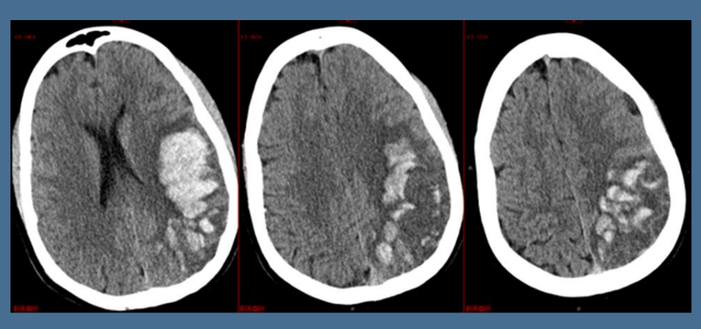

Figure 1: Initial CT showing massive confluent hemorrhagic infarct in left cerebral hemisphere.

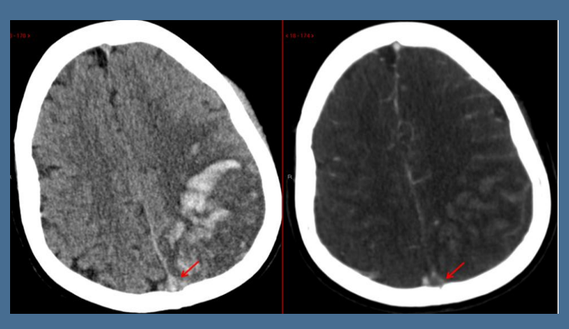

Figure 2. Hyperdense superior sagittal sinus on native CT (left) and filling defect on contrast enhanced CT (right) indicating sinus thrombosis.

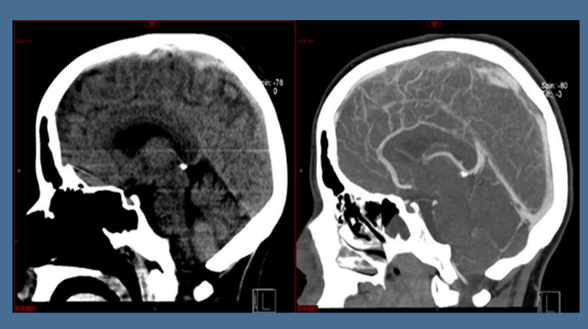

Figure 3. Hyperdense superior sagittal sinus on native CT (left) and filling defect on contrast enhanced CT (right).

Full blood count, renal function panel, autoimmune screen (erythrocyte sedimentation rate, complement 3, complement 4, antinuclear antibody, and anti–double-stranded DNA), prothrombotic screen (protein C, protein S, antithrombin III, anticardiolipin IgG, and IgM, lupus anticoagulant), liver function tests, genetic tests, lipid profile, chest X-ray, electrocardiogram were normal. HBsAg, anti-HCV, and anti-HIV 1 / 2 were negative.

On the second day, Glasgow Coma Scale (GCS) was 3 with increase of inflammatory factors (procalcitonin, C-reactive protein). Coagulation profile showed hyperfibrinogenemia with elevated D-dimer.

After two days, magnetic resonance imaging, angiography and venography (MRI/MRA/MRV) revealed superior sagittal sinus and corresponding cortical veins thrombosis with massive VHI and subfalcine and uncal herniation (Figure 4).

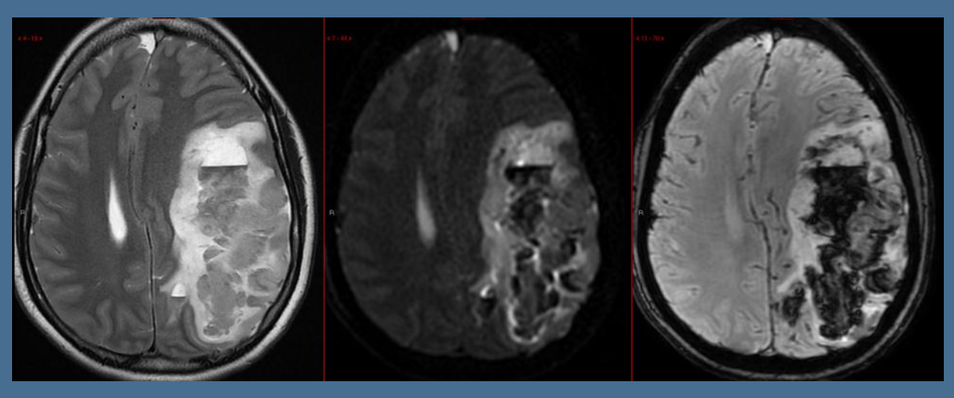

Figure 4. Axial T2W (right), DWI (center) and SWI (left) showing extensive confluent hemorrhagic infarct of the left cerebral hemisphere.

Repeated CT scan showed extensive infarction and edema with significant midline shift (subfalcine, uncal and tonsillar herniation) (Figure 5).

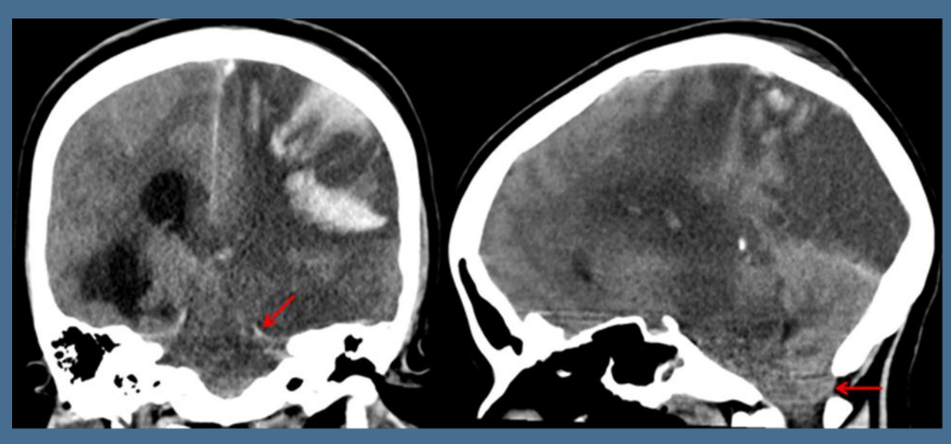

Figure 5. CT coronal (left) and sagittal image (right) showing uncal and tonsillar herniation.

Large hemorrhage was considered as contraindication for heparin administration; therefore, no anticoagulants or thrombolytic agents were administered, and patient died on day 4.

Conclusion: There are still many unanswered questions regarding the pathophysiology and pharmacology of the toxic effects of MDMA. Venous thrombosis could be a result of thrombogenic effect and dehydration induced by MDMA (2,4,5). Parenchymal edema with venous infarction and hemorrhage as complications, develops in 10-50% of patients. Hemorrhagic infarcts are bad prognostic factors of CVST. Elevated cerebral venous pressure due to cerebral venous occlusion can result in a spectrum of phenomena including dilated venous and capillary vessels, development of interstitial edema, increased cerebrospinal fluid production, decreased cerebrospinal fluid absorption and rupture of venous structures (hematoma) (6-8).

This case shows that although systemic anticoagulation is first-line treatment for CVST, in patient intoxicated with MDMA due to large hemorrhage, fast and deleterious course of the disease, different approach might be needed.

Literature:

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