Bangladesh Crit Care J March 2016; 4 (1): 58-60

Case Report

A Unique Case of Central Venous Sinus Thrombosis in Type 2 Diabetes with Severe Hypertriglyceridemia

Debmalya Sanyal[1], Kingshuk Bhattacharjee[2], Soumyabrata Roy Chaudhuri[3]

Abstract:

We present a case of superior sagittal sinus thrombosis caused by uncontrolled type 2-diabetes and extremely high triglyceride. We believe this is the first report that demonstrates an association of CVST with uncontrolled type-2-diabetes and hypertriglyceridemia in an adult but in the absence of concomitant ketoacidosis, hyperosmolar state or any known pro-thrombotic conditions. Our case highlights the need for clinicians to consider CVST among patients with uncontrolled diabetes and hypertriglyceridemia. New diagnostic measures may become necessary which may also have an implication on the risk of recurrence and duration of anti-coagulant therapy in this group of patients.

Key words : *Cerebral venous sinus thrombosis (CVST), Hypertriglyceridemia, Type 2 diabetes, cerebral haemorrhage.*

Introduction :

Cerebral venous sinus thrombosis (CVST) is a rare and potentially fatal condition warranting astute assessment and expert critical care. CVST can be attributed to scores of risk factors and may present in different ways. We present a case of superior sagittal sinus thrombosis caused by uncontrolled type 2-diabetes and extremely high triglyceride. We believe this is the first report that demonstrates an association of CVST with uncontrolled type-2-diabetes and hypertriglyceridemia in an adult but in the absence of concomitant ketoacidosis, hyperosmolar state or any known pro-thrombotic conditions.

Case Report:

A forty-year-old Indian male known to have type 2 diabetes mellitus (DM) since 10 years presented with sudden onset occipital headache accompanied with weakness and focal convulsion of right upper limb. There was no focal neurodeficit, except for Grade 4 power in right upper limb. Computed tomography (CT) scan revealed intra cerebral hemorrhage in the left parietal lobe (Figure 1).

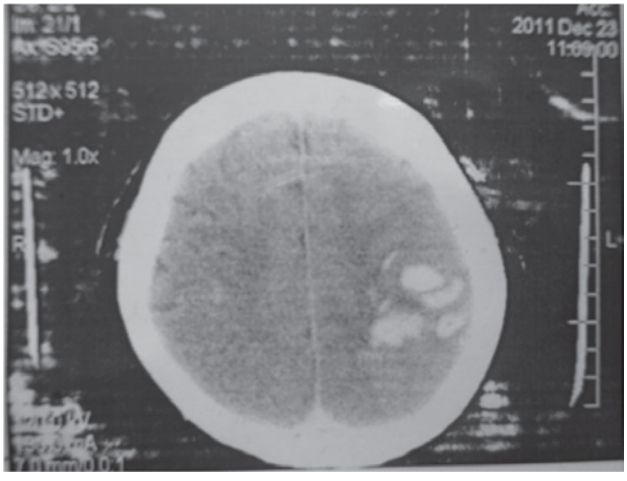

Fig 1 : Computed tomography (CT) scan revealing intra cerebral haemorrhage in the left parietal lobe

MRI (magnetic resonance imaging) of brain & Magnetic Resonance venogram showed "subacute hemorrhagic infarct in left parietal lobe with almost complete occlusion of superior sagittal sinus sparing only posterior aspect". (Figure 2).

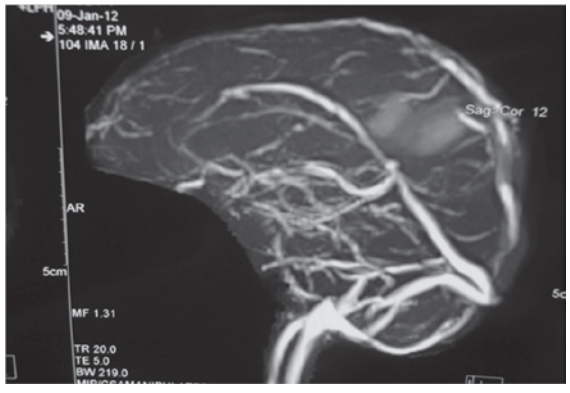

Fig 2 : Magnetic Resonance Venogram showed almost complete occlusion of superior sagittal sinus sparing only posterior aspect.

1. Dr Debmalya Sanyal, Department of Endocrinology, KPC Medical College, Kolkata, West Bengal, India – 700032
2. Dr Kingshuk Bhattacharjee, Independent Clinician & Medical Writer, 547, Dumdum Park, Kolkata, West Bengal, India - 700055
3. Dr Soumyabrata Roy Chaudhuri, Senior Registrar, Department of Endocrinology, KPC Medical College, Kolkata, West Bengal, India - 700032

Corresponding Author :

Dr Debmalya Sanyal
36 Block H New Alipore Kolkata
West Bengal, India, 700053
drdebmalyasanyal@gmail.co
dr_debmalya@hotmail.com

The patient was diagnosed with hypertriglyceridemia three months back and started on fenofibrate. Patient was non-adherent to fenofibrate but compliant to oral anti-diabetic drugs. Despite regular oral anti-diabetic medications (glimepiride 2mg and metformin 1gm), his glycaemic control was not optimal with fasting plasma glucose (FPG) and post prandial plasma glucose (PPPG) well above the recommended targets (Table 1). Moreover his lipid profile revealed extremely high serum triglyceride level but total cholesterol (TC), high density lipoprotein (HDL), low density lipoprotein cholesterol level, Lipoprotein (a) were all within normal limits. (Table 1). Arterial pH, serum osmolality, neutrophil count, bicarbonate and serum lipase were also within the normal ranges (Table 1).

Table 1 : Baseline Laboratory Examinations

Test	Result	Normal
Triglyceride, mg/dL	3580	<200
Low Density Lipoprotein Cholesterol, mg/dL	122	<130
High Density Lipoprotein Cholesterol, mg/dL	41	>40
Lipoprotein(a)	11	<30
Total Leucocyte Count, per mm^3	9000	4000-11000
Fasting Plasma Glucose, mg/dL	202	< 100
Post Prandial plasma Glucose, mg/dL	328	< 140
Glycosylated hemoglobin, in %	8.40	<5.7
Arterial pH	7.4	7.35-7.45
Serum Osmolality, mosm/kg (mmol/kg)	288	275-295
Serum Sodium, mEq/L	134	136 - 149
Bicarbonate, mEq/L	23	21-28
Serum Lipase, U/L	30	< 60
Polymorphonucleur neutrophil, %	72%	45-75%
Serum potassium, mEq/L	4	3.8 - 5.2
Serum calcium, mEq/L	7.8	
AST, U/L	90	<34
ALT, U/L	32	<34
Serum albumin, g/dL	4.1	3.5 - 5.3
Total bilirubin, mg/dL	0.4	0.1-1.0
Gamma glutamyl transpeptidase, IU/L	30	< 49
Urea, mg/dL	24	21-43
Creatinine, mg/dL	0.7	< 1.5
Serum amylase, U/L	55	< 100

His past medical history was remarkable for acute pancreatitis attributed to hypertriglyceridemia 9 months back. No family history of hypertriglyceridemia. No history of alcohol/beer intake. Screening for coagulopathies and auto-immune disorders showed normal antithrombin, normal protein C, normal protein S, normal activated protein C resistance, normal homocysteine, normal human factor V and absence of lupus anticoagulant, anticardiolipin antibodies and antinuclear antibodies.

A diagnosis of left parietal hemorrhagic infarct due to superior sagittal sinus thrombosis in a case of uncontrolled diabetes & hypertriglyceridemia was made. The patient was treated with premixed Insulin 22U twice daily, carbamazepine 600mg, warfarin, fenofibrate 200mg, rosuvastatin 10mg. The patient recovered well and laboratory examination three weeks later revealed a FPG-135, PPG-182, international normalized ratio (INR-2.1) but slightly elevated TG. (Table 2)

Table 2 : Pertinent Laboratory Values 3 weeks after CVST[a]

Test	Result	Normal Range
Low Density Lipoprotein Cholesterol, mg/dL	78	< 130
High Density Lipoprotein Cholesterol, mg/dL	34	> 40
Triglyceride, mg/dL	308	< 200

[a]CVST- Central venous sinus thrombosis

Discussion :

Our case report suggests that the major risk factors for central venous sinus thrombosis are associated with uncontrolled DM and extremely high triglyceridemia. The causal relationship of diabetes and venous thromboembolism still remains controversial. Diabetes itself is a condition associated with several defects of platelet function, coagulation and fibrinolytic systems resulting in a hypercoagulable state, the increased rate of thrombotic complications is often observed in the presence of additional risk factors.[1] It is clearly understood now that the rate of thrombotic complications is increased in diabetic patients with frequent episodes of hyperketonaemia and hyperosmolarity.[1,2] Published literature on CVST and diabetes are confined to case reports predominantly among patients with type 1 diabetes and is in the context of diabetic ketoacidosis (DKA), dehydration or hyper-osmolarity.[2] In association with type 2 diabetes, two cases of CVST are reported: one presenting with DKA while the other patient had transverse sinus aplasia.[3,4] To our knowledge, this is the first report of a CVST in association with uncontrolled type 2 diabetes and hypertriglyceridemia but in the absence of concomitant ketoacidosis, hyperosmolar state or any known pro-thrombotic conditions.

In our case hypertriglyceridemia is one of the pre-disposing factors for developing CVST. Ageno and associates did a meta-analysis of eleven studies measuring the triglyceride levels to investigate its effect on VTE comprising of eight case-control and 3 cohort studies[5]. In most studies, the patients with VTE had higher triglyceride levels than that of the control population, with a mean difference of 21.0 mg/dL

(95% CI, 11.0 to 31.0) in the case-control studies and 8.6 mg/dL (95% CI, 1.2 to 16.0) in the cohort studies**.** Furthermore, our patient had a normal leucocyte count on admission, thereby strengthening a tenable relationship between elevated hypertriglyceridemia and superior sagittal sinus thrombosis (SSST).

The optimum treatment for CVST and its duration still remains uncertain, especially in the background of unidentifiable aetiology. On the basis of limited available evidence at our disposal, anticoagulation appears to be safe and probably reduces the risk of death or dependency. There are also reports of treatment with systemic or local thrombolysis in cases with a deteriorating clinical course despite anticoagulation therapy. However, no randomised controlled trial data is available to support the efficacy or safety of thrombolysis in CVST. The duration of anti-coagulant therapy in CSVT depends on the aetiology as well. CSVT in presence of transient risk factors are generally given an anti-coagulant therapy for three to six months whereas those with unknown causes or with hereditary thrombophilia are anti-coagulated for longer duration due to high chances of recurrence.

Our case might be considered among the 65–80% of CVST cases with one or more identifiable causes. Here, the main causes being uncontrolled hyperglycaemia and extremely high triglyceridemia which may have played a role in increasing our patient's thrombotic risk, and highlight a potential direct association between CVST, hypertriglyceridemia and hyperglycaemic state even in the absence of dehydration or ketoacidosis. If such a relationship between diabetes, hypertriglyceridemia and CVST were to be found, new diagnostic measures might be warranted and this may also have an implication on the risk of recurrence and duration of anti- coagulation in this group of patients.

Conclusion :

Our case highlights the need for clinicians to consider CVST among patients with uncontrolled diabetes and hypertriglyceridemia. New diagnostic measures may become necessary which may also have an implication on the risk of recurrence and duration of anti-coagulant therapy in this group of patients.

References :

1. Keenan CR, Murin S, White RH. High risk for venous thromboembolism in diabetics with hyperosmolar state: comparison with other acute medical illnesses. *J Thromb Haemost*. 2007; 5: 1185–1190
2. Kalis NN, van der Merwe PL, Schoeman JF et al. Cerebral oedema with coning in diabetic ketoacidosis. Report of 2 survivors. *S Afr Med J.* 1991; 79: 727–731
3. Usdan LS, Choong KWL, McDonnell ME. Type 2 diabetes mellitus manifesting with a cerebral vein thrombosis and ketoacidosis. *Endocr Pract*. 2007; 13: 687–690
4. Nagai T. Diabetes mellitus with left transverse sinus thrombosis and right transverse sinus aplasia. *Diabetes Res Clin Pract.* 2001; 51: 39–43
5. Ageno W, Becattini C, Brighton T, Selby R, Kamphuisen PW. Cardiovascular risk factors and venous thromboembolism: a meta-analysis. *Circulation*. 2008; 117: 93-102.