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Case Report

Cerebral Venous Sinus Thrombosis precipitated by oral Tranexamic Acid use for Menorrhagia

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Abstract:

An 18-year-old female was referred to the IALCH Neurology department with a one-week history of severe holocephalic headaches. The headaches were associated with diplopia to distant vision, bilateral tinnitus, transient visual obscurations and two episodes of vomiting. No seizures were reported. On medical history she reported irregular, heavy menses for the last three years. She was managed by her general practitioner and started on oral tranexamic acid (1000mg 12 hourly during menstruation) two days prior to the onset of the headaches. There was no history of oral contraceptive use, retinoids, recent illness or trauma. There was no history of recent COVID infection or vaccine. She had never been pregnant with no previous history to suggest miscarriage. There was no family history of note. On social history, she was a student with no history of cigarette smoking, alcohol or illicit substance use.

Key words: human umbilical cord blood serum; regenerative medicine; cytokines; cell therapy; clinical application

Introduction

An 18-year-old female was referred to the IALCH Neurology department with a one-week history of severe holocephalic headaches. The headaches were associated with diplopia to distant vision, bilateral tinnitus, transient visual obscurations and two episodes of vomiting. No seizures were reported. On medical history she reported irregular, heavy menses for the last three years. She was managed by her general practitioner and started on oral tranexamic acid (1000mg 12 hourly during menstruation) two days prior to the onset of the headaches. There was no history of oral contraceptive use, retinoids, recent illness or trauma. There was no history of recent COVID infection or vaccine. She had never been pregnant with no previous history to suggest miscarriage. There was no family history of note. On social history, she was a student with no history of cigarette smoking, alcohol or illicit substance use.

On examination she was noted to be thin with a body mass index of 17.2 kg/m^2. On general exam, conjunctival pallor was seen. She was well hydrated with normal vital signs. Her cardiovascular, respiratory and abdominal systems were normal. On neurological exam, funduscopy revealed bilateral swollen discs. Her visual acuity was normal (6/6 bilaterally). Visual fields revealed a mildly enlarged blind spot on the left (see also Figure 3), but no constricted fields. Pupils were normal. Primary gaze appeared normal on first inspection but alternate-cover test revealed a subtle right esotropia. The rest of the neurological examination was normal.

Her baseline blood tests revealed a microcytic hypochromic anemia (haemoglobin 8.1g/dL, mean corpuscular volume 72.3fL, red cell distribution width 19.9%) with normal platelets (441 x10^{-9}/L), international normalized ratio (INR - 1.03), renal function (creatinine 45umol/L) and electrolytes (sodium 138mmol/L, potassium 3.6mmol/L). A thrombophilia screen was negative - including tests for anti-phospholipid syndrome syndrome, protein C and S, antithrombin III, factor V Leiden mutation and factor VIII. Autoimmune screen was negative. HIV screen was negative.

CT brain scan showed an extensive dural venous sinus thrombosis involving the posterior superior sagittal sinus, right transverse sinus, right sigmoid sinus and right proximal internal jugular vein (see Figure 1). No venous infarcts were noted. Retinal nerve fibre layer (RNFL) thickness was found to be markedly elevated bilaterally (right eye 217 microns; left eye 255 microns) on optical coherence tomography (OCT) (see Figure 2).

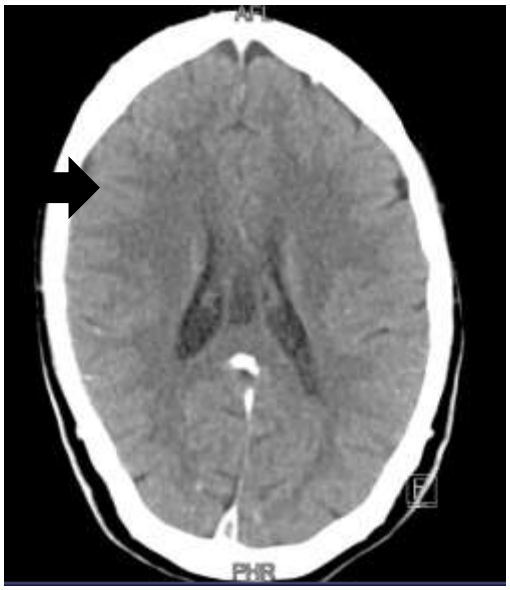

Figure 1: Contrasted CT brain from index patient reveals an empty delta sign in keeping with superior sagittal sinus thrombosis (black arrow)

A diagnosis of venous sinus thrombosis was made, with the risk factor thought to be tranexamic acid use. The patient was started on low molecular weight heparin and warfarin, with instructions to avoid the future of use tranexamic acid. She was also referred to gynaecology for further work-up.

On one month follow up at neurology clinic, the patient reported no further headaches or visual problems. Funduscopy revealed mild blurring of the medial optic disc but with significant improvement, confirmed on repeat OCT which revealed a RNFL thickness of 126 microns and 134 microns on the right and left eye respectively see Figure 4). She is to complete six months of warfarin in total, with monthly INR measurements (target INR 2-3).

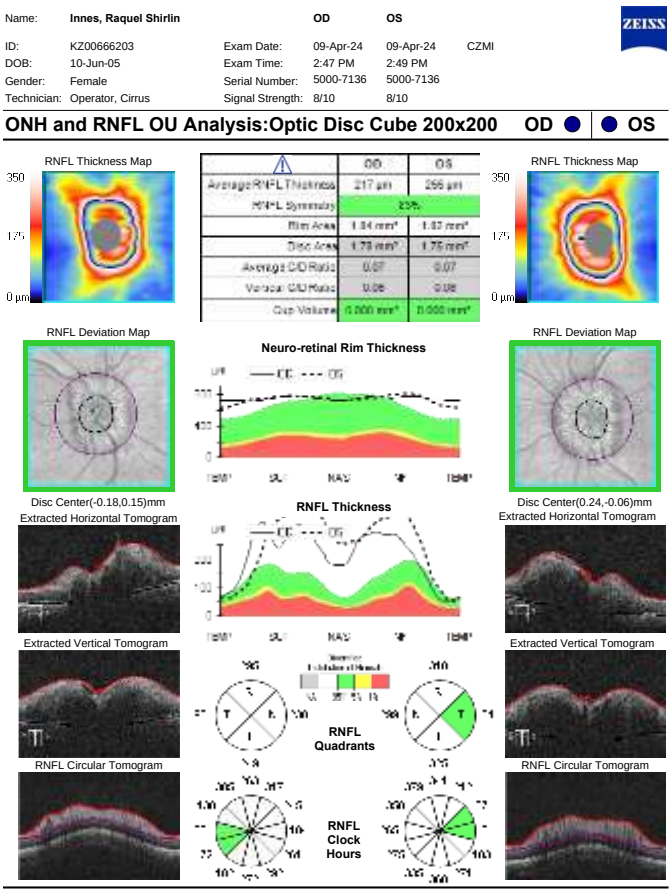

Figure 2: Optical coherence tomography (OCT) from index patient on presentation reveals bilateral optic disc swelling

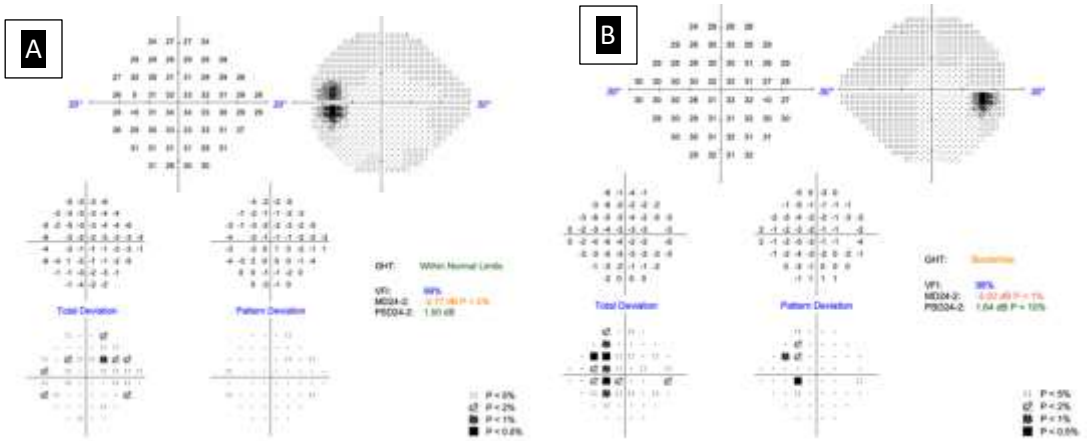

Figure 3: Formal visual field testing from index patient (A: left eye; B: right eye).

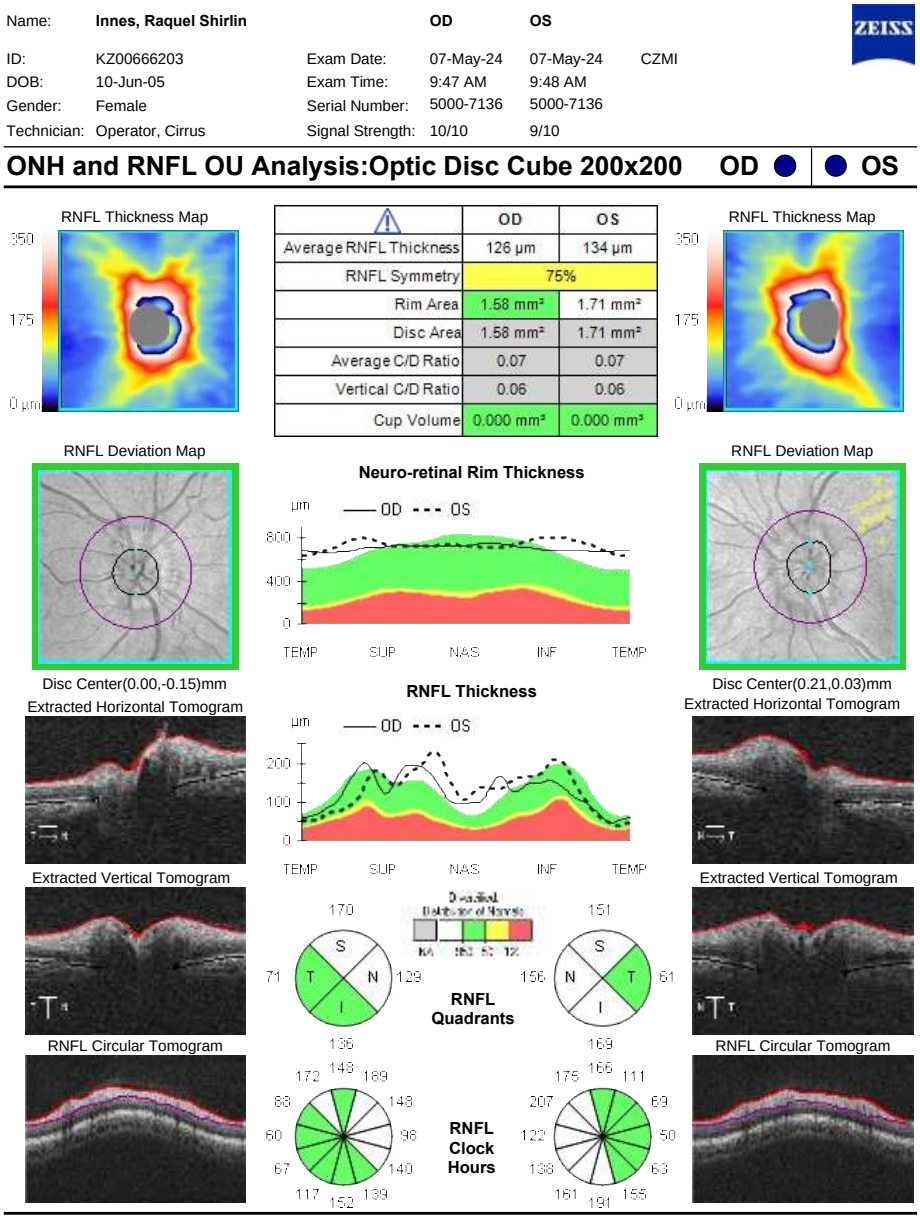

Figure 4: Optical coherence tomography (OCT) from index patient at one month follow-up

Discussion

Cerebral venous sinus thrombosis (CVST) refers to the presence of a blood clot in the dural venous sinuses or cerebral veins (1). It represents a small percentage of overall stroke incidence (0.5-3%) (1). Common predisposing factors include oestrogen-containing oral contraceptives, pregnancy/ puerperium, obesity, dehydration and thrombophilias such as anti-phospholipid syndrome (1, 2). Considering these common predisposing factors, it is unsurprising that two-thirds of CVSTs occur in females (1, 2).

Certain medications have been implicated as a transient risk factor for CVST – namely corticosteroids, L-asparaginase, thalidomide and tamoxifen (1). Vaccine-induced thrombotic thrombocytopaenia (VITT) may also be a provoking factor (1). The anti-fibrinolytic agent tranexamic acid, despite it being a potent haemostatic agent, is not a recognized predisposing factor for CVST.

Oral tranexamic acid is indicated in both the acute and long-term treatment of heavy menstrual bleeding (3). It is particularly useful in patients with menorrhagia who wish to fall pregnant and in whom hormonal contraceptive options are contraindicated (3). While there is a considerable body of literature detailing a possible association between tranexamic acid and venous thromboembolism (deep vein thrombosis, pulmonary embolism) and arterial thrombosis (myocardial infarction, arterial stroke) (1-5), current associations between tranexamic acid and CVST are limited to a few case reports only.

A large Danish cohort study by Meaidi *et al.* (2021) of more than 2 million women found an adjusted incidence rate ratio of 4.0 (1.8-8.8) for venous thromboembolism, when comparing oral tranexamic acid use with on-use (3). This study did not however include CVSTs in the venous thromboembolism group, as only deep vein thrombosis and pulmonary embolism were included in this group (3). Of note - the number needed to harm per five days of oral tranexamic acid treatment was 78,549 women - leading the authors to conclude that 'venous thromboembolism is a very rare adverse event of oral tranexamic acid when used for a short term and in generally healthy women' (3).

A systematic review by Taeuber *et al.* (2021) that looked at 216 surgical studies across the world and found that 'tranexamic acid is not associated with increased risk of any thromboembolic event' (5). Non-surgical patients were also not shown to be at increased risk in a systemic review by Long and April (2020) (6). More specifically in the context of menorrhagia, a case-control study conducted in Sweden by Sundström *et al.* (2008) found that although tranexamic acid was associated with an increased risk of venous thromboembolism (adjusted odds ratio of 3.20 [95% CI 0.65-15.78]), the result was not statistically significant owing to a relatively small sample size (4). As in the Danish cohort noted earlier, none of these studies included CVSTs in their analyses.

A case report from China of a 31-year-old male who developed CVST three days post-tonsillectomy and initiation of tranexamic acid was published in *Auris Nasus Larynx* in 2022 (7). While the authors concluded that the 'CVST was related to tranexamic acid therapy' (7), the recent surgery and post-operative infection may have also been contributing factors in this case. There was also a published case in 1990 of another anti-fibrinolytic, ε-aminocaproic acid, linked to CVST in a 42 year old female in Israel (8). In this case report, the patient was on chronic ε-aminocaproic acid therapy (3 days a month for 7 months) for menorrhagia and developed superior sagittal and left transverse sinus thrombosis, in the absence of other predisposing factors (8).

To the authors' knowledge there are no published data on the topic of tranexamic use and CVST/ venous thromboembolism in South Africa or Africa. This includes case reports. In our index patient, we believe that the CVST was related to tranexamic use. This is assertion is supported by the short duration between initiation of tranexamic acid therapy (2 days) and the development of symptoms related to CVST, as well as the absence of any other known predisposing factors for CVST. There are plausible pathophysiological mechanisms by which tranexamic acid may contribute to venous thromboembolism beyond deep vein thrombosis and pulmonary embolism, and we encourage further studies specifically analysing CVST in the context of tranexamic acid use.

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