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Imaging the diverse presentations of cerebral venous thrombosis occurring at high altitude area in Armed Forces personnel: Case series

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ABSTRACT

Cerebral venous thrombosis (CVT) is a relatively uncommon condition in general population. However, occurrence in Armed Forces personnel serving at high altitude area (HAA) is relatively more common. Due to varied clinical presentation and other mimickers at HAA, diagnosis and treatment gets delayed. Imaging forms the corner stone in diagnosis. A high index of suspicion and awareness about findings on cross-sectional imaging especially non contrast enhanced computed tomography (NCCT) which is widely available at zonal service hospitals supplemented by contrast enhanced computed tomography (CECT), CT venography (CTV) and magnetic resonance imaging (MRI)/MR venography (MRV) enables early diagnosis and treatment.

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Introduction

Cerebral venous thrombosis (CVT) is a relatively uncommon condition in general population. However, occurrence in Armed Forces personnel serving at high altitude area (HAA) is relatively more common. Due to varied clinical presentation and other mimickers at HAA imaging forms the corner stone in diagnosis. We present few such cases who presented with different neurological symptoms and signs with imaging leading towards the diagnosis of CVT.

Case1

Forty one year old male was evacuated from HAA (>3500 m) as a suspected case of high altitude cerebral oedema. He had three days history of severe progressive headache with altered sensorium. At time of admission patient had Glasgow coma score (GCS) of 8/15. His blood pressure (BP) was 108/70 mm of Hg, pulse (P) 64/minute regular and temperature (T) was 98.8 degree Fahrenheit (F). Patient had neck rigidity. His left pupil showed sluggish reaction. Biochemical investigations revealed

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Fig. 1 – Thrombosis SSS with ICH and SAH. (a) NCCT axial image: distended hyperdense SSS (white arrow) with ICH left parietal lobe (yellow star). (b) NCCT axial image: thrombosis SSS (white arrow) with SAH (open arrow) and mass effect in form of chinked left lateral ventricle and mid line shift towards right (open curved arrow). (c) CT venogram axial image: non-opacified SSS 'empty delta sign' (white arrow). (d) CT venogram coronal MIP image: non-opacified SSS (white arrow) and left sigmoid sinus (open arrow).

haemoglobin (Hb) 17.1 mg%, total leucocyte count (TLC) 16,800/cubic mm, differential leucocyte count (DLC) Neutrophils (N)78 Lymphocyte (L)18 Eosinophil (E)04, platelet count 2.89 lakh/cubic mm, prothrombin time (PT) 13 s, INR 1.2. Patient underwent non contrast enhanced computed tomography (NCCT) scan of head which revealed intracerebral haemorrhage (ICH) left parietal lobe with subarachnoid haemorrhage (SAH) and associated mass effect along with hyperdense superior sagittal sinus (SSS) (Fig. 1a and b). CT venogram revealed non-opacification of SSS, left transverse and sigmoid sinuses (Empty delta sign) (Fig. 1c and d). An impression of CVT with haemorrhagic venous infarct and SAH was given. Patient

was managed conservatively, unfortunately he succumbed to his illness on day 4 of admission.

Case 2

Thirty eight year old male in HAA (>3000 m) was admitted with single episode of witnessed seizure. At admission BP was 134/86 mm of Hg, P 78/minute regular and T: 97.8 F. Tongue bite was present. His systemic clinical examination including neurological examination were normal. Biochemical investigations revealed Hb 15.8 mg%, TLC 10,200/cubic mm, DLC

Fig. 2 – Thrombosis right transverse sinus with venous infarct right temporo-occipital lobes. (a) NCCT axial image: distended hyperdense right transverse sinus (white arrow) with venous infarct right temporo-occipital lobes (yellow star). (b) CECT axial image: non-opacified right transverse sinus (white arrow), normally opacified left transverse sinus (curved open arrow) and venous infarct right temporo-occipital lobes (yellow star). (c) CECT axial image: non-opacified right sigmoid sinus 'empty delta sign' (open arrow).

N66L32E02, platelet count 2.1 lakh/cubic mm, PT 12 s, INR 1.1. Patient underwent CECT head for provisional clinical diagnosis of seizure (investigation). Non-contrast images revealed hyperdense and distended right transverse and sigmoid sinuses with hypodensity in the right temporo-occipital lobes (mean HU value of hypodensity: 31) (Fig. 2a). On post contrast images non-opacification of these sinuses (Fig. 2b and c) was observed. An impression of CVT with venous infarct right temporo-occipital lobes was given. Patient was managed conservatively with anticoagulation therapy.

Case 3

Twenty four year old male at HAA (3800 m) for 3 months was admitted with recurrent episodes of non-projectile vomiting of 10 days duration. He also had loss of memory for recent events with generalised body ache. At time of admission BP was 124/86 mm of Hg, P 86/min, T 99 F. Patient had non-specific agonising burning pain all over the body(bilateral) on touch and pressure with no focal motor or sensory deficit. A provisional diagnosis of psychosomatic complaints was made. Biochemical investigations including CSF analysis were normal. NCCT revealed mildly distended and hyperdense straight sinus, vein of Galen and internal cerebral veins (Fig. 3a). Magnetic resonance imaging (MRI) brain revealed loss of flow void in straight sinus, vein of Galen and internal cerebral veins. These venous sinuses were appearing hyperintense on T1 weighted (T1W), T2 weighted (T2W)/FLAIR (fluid-attenuated inversion recovery) images with blooming on susceptibility weighted imaging (SWI) images. Altered signal intensities were also observed in ventromedial thalami and

splenium of corpus callosum appearing hypointense on T1W images and hyperintense on T2W/FLAIR images with blooming in bilateral thalami on SWI images (Fig. 3b–f). An impression of CVT involving deep venous system with venous infarct bilateral thalami and splenium of corpus callosum was given. Patient was managed conservatively with anticoagulation therapy.

Case 4

Twenty five year old male in HAA was (>3000 m) admitted with persistent, global, gradually worsening headache of 15 days duration. At admission BP was 124/88 mm of Hg, P 84/minute, T: 98.4 F. Systemic examination including neurological examination was normal. Investigations were Hb 16.8 mg %, TLC 9800/cubic mm, DLC N67L30E03, platelet count 2.43 lakh/cubic mm, PT 12 s, INR 1.1. NCCT revealed distended and hyperdense SSS (Fig. 4a).

MRI brain revealed loss of flow void in SSS appearing hyperintense on T1W and T2W images with blooming on SWI images (Fig. 4b–e). Magnetic Resonance Venography (MRV) revealed non-visualised SSS with prominent facial collaterals (Fig. 4f). An impression of CVT of SSS was given and patient was managed with anticoagulation therapy.

Discussion

CVT refers to thrombotic occlusion of cerebral venous system which includes deep venous system, superficial venous system and dural venous sinuses.¹ CVT is a relatively

Fig. 3 – Thrombosis of deep cerebral vein with venous infarcts bilateral thalami and splenium of corpus callosum. (a) NCCT axial image: distended hyperdense straight sinus and vein of Galen (white arrow). MRI brain (b) T1 weighted axial image hyperintense straight sinus and vein of Galen (white arrow) with hypointense thalamic infarct thalami (open arrow). (c) T1 weighted sagittal image: hyperintense straight sinus, vein of Galen and internal cerebral veins (white arrow). (d) T2 weighted axial image and (e) FLAIR axial image: absent flow void and hyperintensity of straight sinus and internal cerebral veins (white arrow). Bilateral thalamic (open arrow) and corpus callosum (curved arrow) infarct. (f) SWI image: hypointense thrombus straight sinus and vein of Galen (white arrow). Blooming bilateral thalami (left > right) (open arrow) suggestive of haemorrhagic infarct.

uncommon condition with extremely variable clinical presentations, predisposing factors, imaging findings and outcomes. Exact incidence in Indian population are not available but studies have suggested female predominance due to more incidence of puerperal CVT with mean age of 31.3 years while studies in western countries have suggested incidence of 0.22–1.32/100,000/year.² Exact incidence in Indian armed forces are not available, however exposure to HAA and associated physiological changes like polycythemia are important risk factors³ with a case series by Jha et al.⁴ suggesting that approximately 6 percent strokes occurring in serving soldiers at HAA were due to CVT. Other important risk factors described in literature include local factors causing endothelial damage like sinus trauma, regional infection and neoplastic invasion/compression or systemic factors causing hypercoagulable state like coagulopathies, peripartum state,

dehydration, oral contraceptive use.^{2,4} Clinical presentation is non-specific and important features includes headache (75%), Papilledema (49%), seizures (37%), motor or sensory deficit (34%), mental status changes (30%), dysphasia (12%) and cranial nerve palsies (12%).⁶ Imaging lays the foundation for diagnosis with NCCT being the corner stone as first imaging modality. High suspicion is required and often NCCT has to be supplemented with CECT/CT venography and MRI/MRV. On NCCT direct signs (32–50%) are hyperdensity and distension of affected venous sinus (dot/delta sign) or vein (cord sign) due to thrombosis. Indirect signs are venous Infarct (50%) which are usually subcortical and do not conform to an arterial vascular territory (venous infarct may be non-haemorrhagic or haemorrhagic presenting as intracerebral haemorrhage), cerebral oedema (16%), SAH (3–4%) especially convexity haemorrhage near affected sinus/cortical vein and SDH

Fig. 4 – Thrombosis of SSS with collaterals. (a) NCCT axial image: distended hyperdense SSS (white arrow). MRI brain (b) T1 weighted, (c) T2 weighted and showing hyperintense SSS (white arrow). (d) SWI image showing blooming of SSS suggesting thrombosis (white arrow). (e) T2 weighted coronal image showing loss of normal flow void of SSS with hyperintensity. (f) MRV: non visualised SSS (white arrow) with prominent facial collaterals (open arrow).

(1–2%). In 55% cases of CVT there are no indirect signs on NCCT. CECT reveals filling defect with peripheral enhancement (empty delta sign) while CT venography (CTV) in addition to empty delta sign also shows collateral venous channels.^{4,5}

On MRI the CVT is seen as loss of flow void in affected sinuses with direct visualisation of thrombus. Thrombus evolve through the stages of oxyhemoglobin, deoxyhemoglobin, methemoglobin and hemosiderin. In acute stage lasting till 5 days from onset the signals are iso-intense on T1W images and hypointense on T2W images due to deoxyhemoglobin. Subacute stage lasts from 6 to 15 days, the signals are predominantly hyperintense on both T1W as well as T2W images due to methemoglobin. In chronic stage that is after 15 days thrombus show variable signals and is usually isointense on T1W images and iso to hyper intense on T2W images, partial recanalisation of affected sinus may also occur in this stage. In all stages thrombus shows blooming on T2*/SWI images due to susceptibility artefacts. MRI is more sensitive in

identifying indirect signs as compared to CT with better characterisation using various sequences including Diffusion weighted imaging (DWI). MRV using 2D TOF (time of flight) or phase contrast techniques reveals non-visualisation of affected sinus. Contrast-enhanced MRV may be used in doubtful cases as it is less likely to be affected by complex flow.^{1,4,5} CTV has almost similar sensitivity and specificity as compared to MRV in identifying CVT with added advantage of fast scanning, thus it is useful in imaging of uncooperative patients.^{5,6}

Digital subtraction angiography (DSA) is still the standard of reference where non-opacification of affected sinus and presence of collaterals indicates thrombosis. It is an invasive technique and indicated when other imaging modalities had equivocal findings, in presence of SAH to rule out other causes like aneurysms or when catheter thrombectomy is planned.⁶

Important conditions which may mimic CVT on imaging are dehydration, dural sinus hypoplasia/aplasia, giant arachnoid granulations and acute SDH (pseudo empty delta sign).⁵

All the cases in our series had history of exposure to HAA and were admitted with different clinical presentations. Due to these varied presentation especially in HAA initial neurological symptoms and signs may be wrongly attributed to acute mountain sickness or high altitude cerebral edema delaying the proper treatment. Thus a high suspicion and evacuation to nearest centre with imaging facilities is required for early diagnosis and treatment. Nearly all zonal hospitals in armed forces have facility of CT scan, thus the radiologist as well as physician reading the scans should be well versed with findings of CVT on NCCT/CECT/CTV. Findings of convexity SAH, venous infarct or cerebral oedema in patients exposed to HAA should be considered as possible CVT.

Conclusion

CVT is common cause of stroke in young armed forces personnel serving at HAA. Due to varied clinical presentation and other mimickers at HAA diagnosis and evacuation is delayed. Non-specific imaging features further delay the diagnosis. A high index of suspicion and awareness about findings on cross-sectional imaging especially NCCT which is widely available enables early diagnosis and treatment.

Conflicts of interest

The authors have none to declare.

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