Hyperdense Vessel Sign: Beyond Thrombosis -A Case Report

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Abstract: Raised haematocrit can cause abnormal hyperdensity of intracranial vessels and dural venous sinuses on NCCT producing an appearance of contrast enhanced scan. Increased attenuation of venous sinuses may also be seen in cortical venous thrombosis on NCCT. Here we report a case of a child with double outlet right ventricle who showed the hyperdense intracranial arteries and venous sinuses on non contrast scan mimicking the dural sinus thrombosis and also showed the presence of cerebral abscess. Presence of hyperdense arteries along with normal enhancement of the venous sinuses on contrast enhanced scans suggested high haematocrit as the cause of vascular hyperdensity.

Keywords: Cortical venous thrombosis; cyanotic heart disease; haematocrit; hyperdense vessels;Non contrast CT(NCCT).

I. Introduction

Conditions which can cause rise in haematocrit like cyanotic heart disease can give rise to hyperdense vessels on NCCT which give the appearance of contrast enhanced scan and mimic the appearance of cortical venous thrombosis. Here we report a case of a child who was known case of double outlet right ventricle and showed the similar findings on imaging.

II. Case Report

7 year old child presented with headache localized to frontal and parietal region for 7 days, vomiting, low grade fever and decreased oral acceptance for 3 days. The child was a known case of double outlet right ventricle with pulmonary stenosis diagnosed since early infancy. Cardiovascular examination revealed central cyanosis, clubbing and presence of a pan-systolic murmur at left lower sternal border. Echocardiography revealed common AV valve with right ventricular dominance with large ASD and VSD. On neurological examination, child was irritable, with presence of hyperreflexia and neck rigidity. There was no neurological deficit. Hemogram revealed Hb-18g/dl (normal range:11.5-15.5g/dl), haematocrit of 54% (normal range:35-45%) and polymorphic leucocytosis. Chest X ray PA view revealed upturned ventricular apex, concave pulmonary bay and right sided aortic arch (Fig 1a).Cerebrospinal fluid examination revealed mild lymphocytic pleocytosis with raised protein levels.

On the basis of clinical history and neurological examination, possibility of cerebral abscess or meningitis were considered which are the most likely neurological complications seen in cyanotic heart disease. For evaluation of neurological symptoms. CT scan of the brain was done. NCCT Brain revealed hyperdense intracranial arteries, superficial and deep venous system. (Fig 1 b). There was also presence of vasogenic edema in right parietal lobe and NCCT attenuation of superior sagittal sinus was 56HU (fig 1 c). On post contrast scan, the dural venous sinuses showed normal contrast enhancement with no evidence of hypodense filling defect to suggest thrombosis. In addition a hypodense rim enhancing lesion (approx. 18x13mm) with perifocal edema suggestive of cerebral abscess was seen in right posterior parietal lobe. There was partial effacement of the right lateral ventricle (Fig 1d). The cause of hyperdense arteries and venous sinuses were attributed to high haematocrit. The child was treated with IV antibiotics and showed marked clinical improvement. The child was discharged and referred to cardiovascular surgeon for further management.

Hyperdense vessel sign on non contrast CT brain is well described as the early sign of intra-vascular thrombus however hyperdense vessels on non contrast CT can also be seen in conditions with raised haematocrit. Increased attenuation of the venous sinuses due to thrombosis or polycythemia (erythrocytosis) can be differentiated from each other by clinical history and haematocrit value . Definite diagnosis of cerebral venous thrombosis can be established by performing MR or catheter venography. CT attenuation of the blood is determined by its hemoglobin concentration. Primarily, X-ray absorption of blood is by the globin content of the hemoglobin with a minor contribution from its iron content [1]. So, an increased hemoglobin concentration causes hyperdensity of vessels on NCCT. Hyperdense vessels due to polycythemia have been reported very rarely [2]. A linear correlation between hyperdensity of intracranial blood vessels and haematocrit concentration has been demonstrated by New et al [3]. The CT attenuation can be helpful in differentiating hyperdensity due to polycythemia and cortical venous thrombosis. Attenuation more than 70HU on NCCT favours cortical venous thrombosis and less than 70 HU favours polycythemia [4]. Also, polycythemia will cause hyperdensity of all the intracranial vessels while cortical venous thrombosis will cause selective hyperdensity of the involved sinus. However since both these conditions can coexist as high haematocrit may cause hypercoagulability predisposing to venous thrombosis [5] so it is important to confirm the diagnosis by imaging.

In patients with cyanotic heart diseases, in addition to the hyperdense vessels, embolic infarctions can also be seen which occur due to turbulent flow with resultant endocardial vegetations leading to thromboembolic events [6]. Meningitis and brain abscess can also be present as seen in our case. Both hypoxia and hyperviscosity can lead to abscess formation [7].

To conclude, it should be remembered that hyperdense vessels on non-contrast CT, which are generally related to intra arterial or cortical venous thrombosis can as well be seen in conditions with a raised haematocrit . However, referring to the clinical symptoms and haematological values can be of help in differentiating between them.

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The Article has not been sent for publishing elsewhere.

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Legends

Fig1(a-d)Chest X ray PA view shows upturned cardiac apex, concave pulmonary bay(large white arrow) and right sided aortic arch(small white arrow).(b)NCCT Brain axial scans depicting the hyperdense arteries. (c)Non contrast Sagittal reformatted image shows hyperdense superior sagittal sinus and arteries. (d)Contrast enhanced CT Brain axial scans show the abscess in right parietal lobe with surrounding vasogenic edema and normal enhancement of the sinuses.

