(International Print/Online Journal)

SJIF IMPACT FACTOR: 5.565
PUBMED-National Library of Medicine ID-101739732

ISSN (Print): 2209-2870
ISSN (Online): 2209-2862

International Journal of Medical Science and Current Research (IJMSCR)
Available online at: www.ijmscr.com
Volume 5, Issue 5 , Page No: 700-702
September-October 2022

IJMSCR

Acute ischemic Stroke: Unusual Neurological Manifestation Of Ebstein's Anomaly In An Elderly Male

Dr. Himanshu Kaushal[1] , Dr. Sulena[2]
[1]Senior Resident, [2]Associate Professor,
[1]Department of Medicine, [2]Division of Neurology,
Guru Gobind Singh Medical College and Hospital, Faridkot, Punjab-151203

***Corresponding Author:**
Dr. Himanshu Kaushal
Senior Resident, Department of Medicine,
Guru Gobind Singh Medical College and Hospital, Faridkot, Punjab-151203

Type of Publication: Case Report
Conflicts of Interest: Nil

Abstract

Background: Patients with congenital heart disease (CHD) are known to exhibit a 10-100 fold increased risk of CVA with associated morbidity and mortality. Ebstein's anomaly (EA) is one of the congenital heart diseases involving right side and includes characteristic features being ventricular displacement of tricuspid valve in the right ventricle, dilatation of tricuspid valve annulus with dysmorphic and enlarged tricuspid valve leaflets.

Case: 65 years elderly male with diabetes presented with right basal ganglia acute infarct with haemorrhagic transformation incidently found to have Ebstein's anomaly.

Conclusion: Congenital heart diseases are unusual risk factors for stroke however timely diagnosis and timely treatment can prevent this complication.

Keywords: NIL

Introduction

Patients with congenital heart disease (CHD) are known to exhibit a 10-100 fold increased risk of CVA with associated morbidity and mortality. Ebstein's anomaly (EA) is one of the congenital heart diseases involving right side and includes characteristic features being ventricular displacement of tricuspid valve in the right ventricle, dilatation of tricuspid valve annulus with dysmorphic and enlarged tricuspid valve leaflets. There also occurs atrialization of the right ventricle and enlargement of right atria. The disease can present alone or as a part of complex syndromes in association with other heart diseases like atrial septal defect, arrhythmias etc. With structurally abnormal chamber morphology, the young patients are at higher risk of developing arrhythmias and consequent cerebrovascular sequalae especially stroke.[1] The most common associated anomalies include atrial septal defect (ASD) and patent foramen ovale (PFO), which occur in up to 80–90% of EA patients, and pre-excitation, which occurs in approximately 15%.[2]

The etiopathogenesis of stroke in EA is not well understood. However, multifactorial pathogenesis has been implicated for stroke in EA. These factors being atrial arrhythmia, paradoxical embolism, prior surgery of tricuspid valve, comorbidities and polycythemia, hypercoaguable state etc.

Case Report:

65 years diabetic male presented with complaint of left sided weakness for 4 days involving upper limb more than the lower limb. Patient had difficulty in holding objects and difficulty in walking. Patient had tremor on the left hand at rest. There was history of generalized slowing and behavioural change following the weakness. On examination patient had MMSE of 16, GCS of 15. Bradyphrenia on speech

examination. Cranial nerve examination shown upper motor neuron type facial palsy of left side of face. Motor examination revealed power of MRC grade 3/5 at shoulder, elbow and wrist in left upper limb and 5/5 MRC grade in right upper limb. Patient had MRC grade 4/5 power in left lower limb at hip, knee and ankle joint, whereas, a power of 5/5 MRC grade was noticed in the right lower limb. Patient had exaggerated DTRs on the left side with extensor plantar that were consistent with UMN type of weakness. Sensory examination was normal. No cerebellar signs were present during examination

Investigations:

MRI BRAIN: T2/FLAIR hyperintensity with no obvious restricted diffusion seen in the right basal ganglia, with blooming on SWI with possibility of haemorrhagic residua. Few prominent vessels are also seen around this area on SWI which show enhancement on delayed scan, possible underlying sequalae to thrombosed vascular malformation (DVA), cannot be completely ruled out. Likely possibility of subacute infarct. T2/FLAIR hyperintensities showing no restricted diffusion are noted in periventricular and deep white matter regions – chronic ischemic changes.

NCCT head: Mild cerebral atrophy with bilateral deep white matter ischemia.

2D ECHO: Ectopics noted during study. Apically displaced tricuspid valve (Ebstein anomaly), mild to moderate tricuspid regurgitation. PASP- 35mm Hg, LVEF 50%.

CBC: 16 Haemogloboin, TLC 4600, PLT 146000

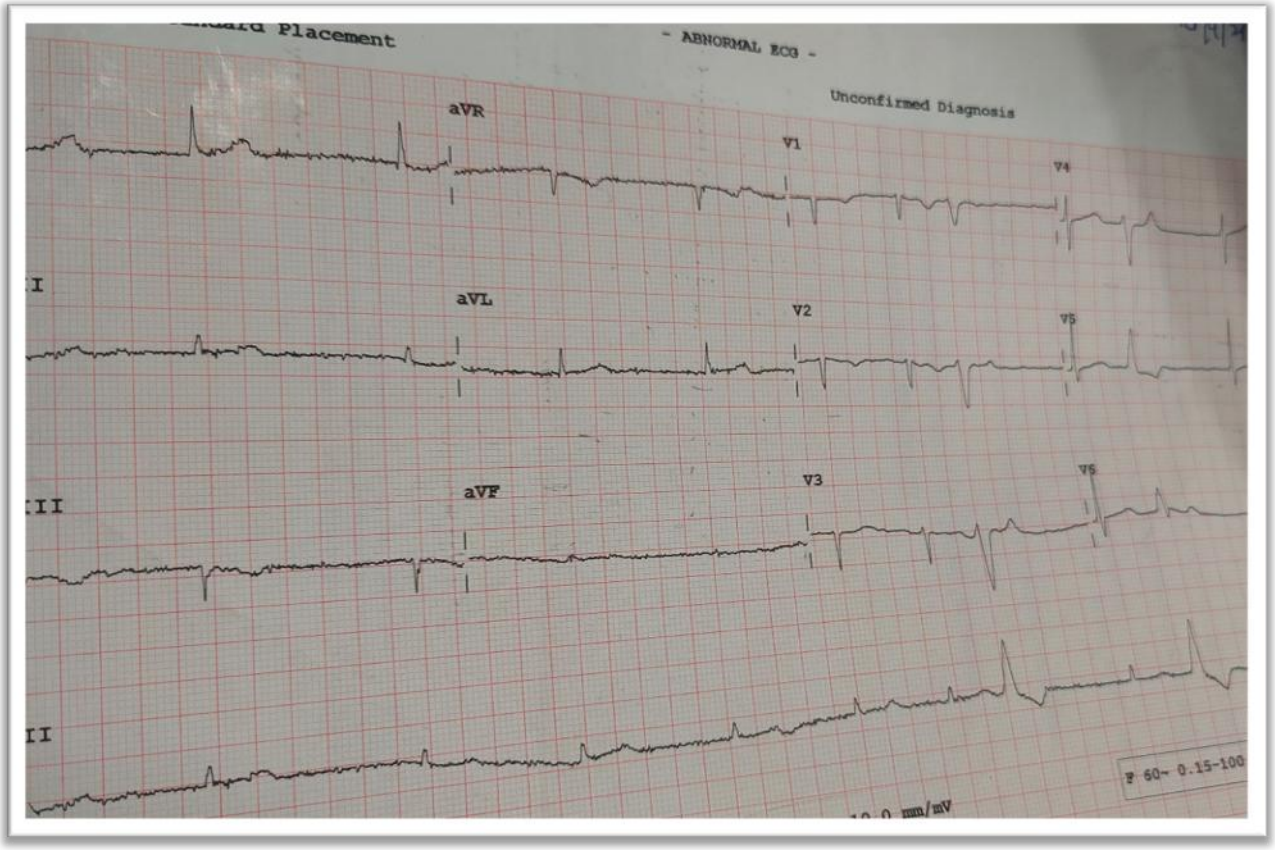
RFT: B Urea 19.8, S. Creat 0.7

LFT: S. Bilirubin 0.7, SGOT 19, SGPT 28, ALP 76

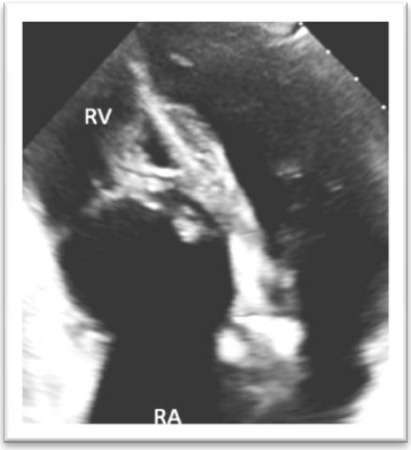
S. Electrolytes: Na 136, K 4, Cl 96, Ca 8.2, Mg 2.05

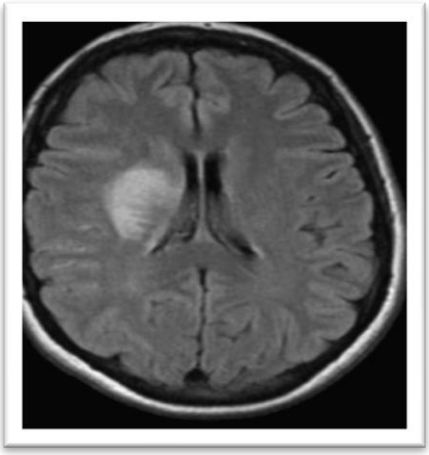
S. Chol 180, TG 65, HDL 41, LDL 126, VLDL 13

Trop-I <0.10, CKMB 26



Volume 5, Issue 5; September-October 2022; Page No 700-702





Discussion:

The incidence and risk factors of CVA specific to adult CHD have recently been alluded to in large registries across North America.[3,4] For example, the cumulative risk of ischemic stroke in a cohort of 29 638 adult CHD patients from Quebec was 6.1% in women and 7.7% in men, respectively.[4]

Study done by Tan et al. in 2019 on 968 patients with congenital heart disease revealed that the patients with ebstein anomaly are at substantial risk of cerebrovascular accident.[1] A similar case report was published in 2020 by Bouwmeester and coworkers discussed that more than 50% of patients with EA have associated shunt between right and left atria in the form of atrial septal defect or patent foramen ovale. Similar to our case, they also mentioned a case of female with left sided weakness on investigation found to have EA. However, the age of stroke was much more lower than our case.[5]

Conclusion:

Congenital heart diseases are unusual risk factors for stroke however timely diagnosis and timely treatment can prevent this complication.

References:

1. Tan NY, Attenhofer-Jost CH, Polkinghorne MD, et al. Cerebrovascular accidents in Ebstein's anomaly. Congenital Heart Disease. 2019;00:1–9.
2. Danielson GK, Driscoll DJ, Mair DD, Warnes CA, Oliver WC Jr. Operative treatment of Ebstein's anomaly. J Thorac Cardiovasc Surg. 1992;104:1195– 1202.
3. Marelli A, Miller SP, Marino BS, Jefferson AL, Newburger JW. Brain in congenital heart disease across the lifespan: the cumulative burden of injury. *Circulation*. 2016;133(20):1951-1962.
4. Lanz J, Brophy JM, Therrien J, Kaouache M, Guo L, Marelli AJ. Stroke in adults with congenital heart disease: incidence, cumulative risk, and predictors. *Circulation*. 2015;132(25):2385-2394.
5. S. Bouwmeester et al. Stroke due to paradoxical embolismin Ebstein's anomaly. European Heart Journal - Case Reports. 2020:1-2 doi:10.1093/ehjcr/ytaa116

Volume 5, Issue 5; September-October 2022; Page No 700-702