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RESEARCH ARTICLE

A CASE REPORT OF PSEUDO WELLENSSYNDROME IN PATIENT WITHPULMONARYEMBOLISM

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Abstract

Wellens' syndrome is described asbiphasic or deeplyinverted T waves in the precordialleads thatis indicative of criticalstenosis of the leftanteriordescendingartery. It isseenin a subset of patients withunstableanginaduring the pain-free interval. . Similar characteristic ECG changes associated with causes other than LAD stenosis have been described as pseudo-Wellens' syndrome. In this case report, we present a young 31-year-old man who presented with characteristic Wellens' ECG changes in the setting of pulmonary embolism with right ventricular strain., biphasic T waves in the setting of pulmonary embolism are rare. Pulmonary embolism was seen in our patient three weeks after starting risperidone. There is a reported association between antipsychotic drugs and increased risk of thromboembolism. Risperidone could have potentially contributed to the pulmonary embolism in our patient given the temporal association and absence of risk factors.

Introduction:-

Wellens' pattern is an electrocardiogram (ECG)finding of biphasic or deeplyinverted T waves in the precordialleads thatis indicative of criticalstenosis of the leftanteriordescendingartery (LAD) (1-2). It isseenin a subset of patients withunstableanginaduring the pain-free interval.

A clinicalentity in which a Wellens' electrocardiographic pattern is apparent withangiographically normal coronaryarteries has been defined as pseudo-Wellens' syndrome.Several conditions are described as pseudo-Wellens'syndrome, such as cocaine and marijuana use and myocardial bridging (1-3-4).

Wepresent a rare case of pseudo-Wellens' syndrome secondary to pulmonaryembolism (PE). Inversion of T-waves inprecordial leads V1–V3is a well-recognizedsubtle ECG change in right ventricularstrain.However, inversed T waves in the setting of PE, althoughpreviousreported(5) are rare. Further, our patient haddeveloped PE threeweeksafterstartingusingantipsychoticdrugs. An association betweenatypicalantipsychoticdrugs and venousthromboembolism (VTE)is been reported. In this case report, wewilldiscussthesetwosalient observations.

Case Report:

A 31-year-oldMoroccan manwithmedicalhistory of psychoticdisorder, presented to ouremergency departmentcomplaining of sudden intermittent cardiacchest pain in leftprecordium, whichstartedearlymorning 13hoursbeforepresentation. It wasassociatedwithpalpitations.Pain wasdescribed as cramping in type with no radiation and referral,and relieved by takingnitroglycerinsublingually. The patient was a smokerwith a 20-pack-year,

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hedeniedhistory of alcohol, illicitsubstances and had no familyhistory of anycardiacdisease. He deniedhistory of fever, sick contacts, vomiting, and light-headedness. The patient has recentlystartedantipsychotictreatment(risperidone) 3 weeksbefore for schizoaffectivedisorder. On physicalexamination, hewas vitallystable. Hisblood pressure was 124/708 mmHg, heart rate was91 beats/min in sinus rhythm, respiratory rate was 16 breaths/min and saturated 98 % on room air, cardiac auscultation revealed normal first and second heartsoundswith no murmurs. Othersystemicexaminationswere normal.

ECG realized duringchestpain showed sinus rhythm, normal progression for R waves in precordial leads, isoelectric ST segment in the absence of pathological Q waves anddeepsymmetric T-waves inversions in leads V1–V6, consistent with Wellens' syndrome (figure 1). A repeat ECG obtainedduring pain-free intervalrevealedresolution of previouslyobserveddeep T waves in leads V1-V6.Initiallaboratory investigation showedslightincrease of cardiac enzyme: Troponin T measuring41ng/L (normal<34 ng/l). Lipid panel wasunremarkablewith total cholesterolmeasuring 165 mg/dL and low-density lipoprotein (LDL) measured 90 mg/dL. Randomblood glucose was 107 mg/dL and serumcreatinine were 0.7 mg/dL, hemoglobinwas 14.4g/dl. An echocardiography wasperformed and waswithin normal limits, the leftand right ventricle(RV)chamber size and cavitywere normal with no regionalwall motion abnormality. Evaluation of valves and pericardiumwere normal.He subsequentlyunderwent distal trans radialcoronaryangiographyand therewas no coronaryarteriesdisease.

Differential diagnoses for precordial T-wave inversions such as cerebrovascularevents, hypertrophiccardiomyopathy (HCM), and Myocardial bridging were ruled out,coronaryspasmwassuspectedin front of nitrate responsive anginawithrestanginaespeciallybetween night and earlymorningwithoutdocumentedspontaneousepisodeand transient ischemic ECG changes according to COVADIS diagnostic Criteria, the smoking history and normal coronarography. Oral nitrates and diltiazem wereaddedwithoutimprovement of symptoms. Thereafter, the patient experienced new-onsetdyspnea on minimal exertion, hisoxygen saturation dropped to 91%. There was no chest pain, tachypnea or tachycardia at this time. Supplementaloxygen at 2l/min via nasal cannulaimproved saturation to 97%.In view of these new findings, abedsideechocardiogram (2D-TTE)was made and demonstrateddilatedRVcavitywithballooning of the interventricular septum into the leftventricle in systole, withakinetic RV apex and hyperkinetic RV free wallsuggestedincreasedresistance in pulmonary circulation described as McConnell'ssign. This raised the possibility of PE.Thus, a CT angiography triple rule-out study of the chestwasperformed and revealedproximal bilateralpulmonaryembolismwithsigns of acute pulmonaryheart(figure 2).

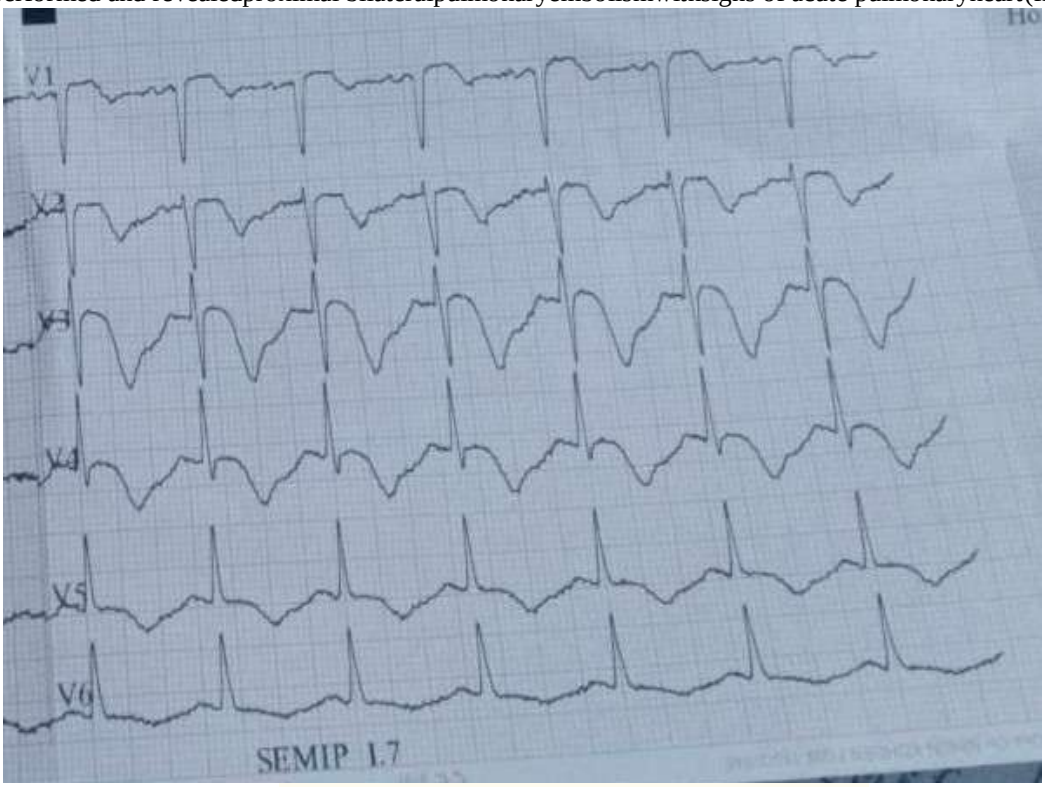

Figure 1:-ECG showingdeep T wave inversions in leads V1–V6 .

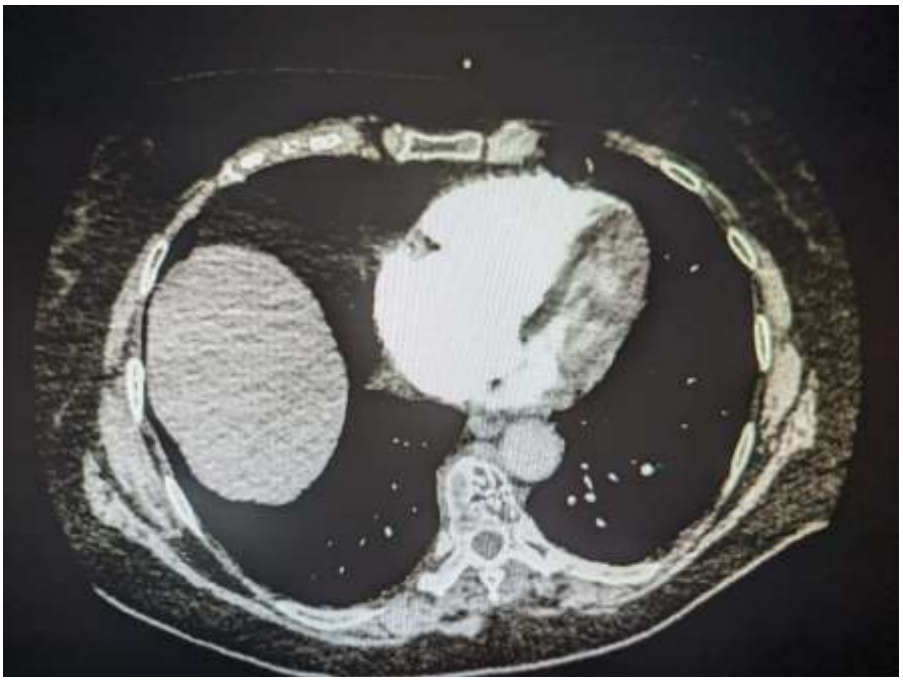

Figure 2a:- CT angiography of the chestshowingenlarged right ventricularcavitywithstraightening of the interventricularseptum.

Figure 2b:- Axial CTA of the chestshowing**proximalbilateralpulmonaryembolism**.

Treatment and follow up:

Thus, after confirmation of PE, our patient wasinitiallytreatedwith a protocol-drivenheparin infusion. He waslatertreatedwithrivaroxaban for a duration of 3 monthsconsidering a provokedevent. Considering the temporal association of PE withrisperidone use, itwasdiscontinued. Hisschizo-affectivedisorderwastreatedwithhaloperidol and citalopram.

On scheduled follow-up monthlyappointments, hewasevaluated by hisprimary care physicianin 3 months. He has had a gradualimprovement of his effort tolerance and deniedsymptomsincludingdyspnea on exertion, orthopnea, paroxysmal nocturnal dyspnea or light-headedness. Echocardiography at 3 monthsafterdischargerevealed right ventricular pressure of 25/8 mm Hg.

Discussion:-

Welles' syndrome was first described by De Zwaan et al as a characteristic T-wave changes in precordial leads in a subset of patients withunstableangina. It wasassociatedwith >90% stenosis of proximal LAD artery and 75% of patients developedanteriorwallmyocardialinfarctionwithin a few days to weeks of presentation (6-7).

As of today, the criteria for Wellen's syndrome are as follows: history of anginalchest pain, minimal or no elevation of cardiac enzymes, no significant ST segment elevation (< 1 mm), no pathologicalprecordial Q waves, no loss of precordial R-wave progression, and deeplyinverted or biphasic T wavesmainly in leads V2 and V3 and sometimes in leads V1, V4, V5, and V6 as noted in our patient (9).

Wellens' syndrome iswell known in cardiology and emergency medicine as a subtleyetominous manifestation of critical proximal LAD occlusion. Biphasic T waves have also been reported in association withcoronaryvasospasm (9).Our case demonstratesthat the presentations of this syndrome may have somedeviationsfrom the traditionaldefinition.

Pseudo-Wellen's syndrome is a termused to describe a constellation of clinicalpresentations and ECG pattern similar to Wellens' syndrome but without the finding of criticalstenosis of LAD coronaryartery. Differentstudiesreported cases of pseudo-Wellens'syndrome..

Wellen's changes in our patient werelikely an ECG change related to right ventricularstrainsecondary to PE. Wenoticedthatsimilar reports have been in publication before. Sedhai YR et al (10) described a patient with pseudo-Wellens' syndrome caused by EP . The patient reported by themhas some differencewith our case.

This case report further highlights the importance of recognizing thisdevioussign as itmaybe a potential manifestation of other alternative life-threatening condition like PE with right ventricularstrain. Other rare causes like coronaryarteryvasospasmassociated cannotbecompletelyexcluded in ourcase.

It is important to note thatour patient haddeveloped PE threeweeksaftertakingrisperidone. The association betweenantipsychoticmedication and increasedrisk for VTE has been reportedsince the introduction of first-generationantipsychotic agents (11-12).

Severalstudiessincethen have shown an increasedrisk of VTE in patients treatedwithconventionalantipsychoticdrugs. A study by Allenet et al(11) reportedthat the risk of PE washigher in atypicalantipsychoticusers (OR 1.17 (95% CI 1.13 to 1.21), p<0.001).

Similarly, a study by Liperoti et al (13) foundantipsychotic agents to increase the risk of VTE.

Conclusion:-

Pseudo-Wellens' syndrome is a rare entitywhichmimics Wellens' syndrome but without the presence of LAD coronaryarterystenosis. It is important for cardiologists to keep in mindthat Wellens'syndrome can bemimicked

Acute PE can presentwith ECG changes seen in Wellen'ssyndrome. Here wedescribea rare case of combination of Pseudo-wellens' and paradoxicalMcConnell's in patient with PE.

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