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A Study of Prevalence of Patients with Junctional Rhythm with Special Reference to Etiological Correlation

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ABSTRACT:-
The aim of the study to prevalence of patients presenting with ECG features s/o junctional rhythm, their presenting symptomatology, disease course and management and to find out the different etiological as well as risk factors responsible for the said ECG changes, evolution of the changes with rectifying these factors and prognosis.

Among all the patients with ECG findings of junctional rhythm 50 patients were randomly selected to find out the different etiologies who were admitted in department of cardiology & followed up in DEPARTMENT OF CARDIOLOGY, ICVS, IPGME&R, S.S.K.M. Hospital, Kolkata from 2016 – 2017.
In our study we found overt hypothyroidism as a cause in only one patient. Subclinical hypothyroid was seen in one patient having sick sinus syndrome. There are numerous toxins that can cause, in overdose, electrocardiogram (ECG) changes, even in patients without history of cardiac pathology. Very few case reports are published. We had on patient having junctional bradycardia with h/o yellow oleander ingestion and in the absence of any other factors or cardiac ailments.

Junctional rhythm is a less frequent rhythm disturbance in comparison to other brady and tachyarrythmias. Most of the time the underlying cause are reversible and does not require implantation of permanent pacemaker.

Keywords: JUNCTIONAL RHYTHM, ETIOLOGICAL CORRELATION, RISK FACTORS, ECG CHANGES, PROGNOSIS.

I. INTRODUCTION

Junctional rhythm occurs when the sinoatrial node (SA node) fails and area around the atrioventricular (AV) junction takes over as the heart's pacemaker. Cardiac rhythms arising from the atrioventricular (AV) junction occur as an automatic tachycardia or as an escape mechanism during periods of significant bradycardia with rates slower than the intrinsic junctional pacemaker. A junctional rhythm includes the following characteristics-
The ventricular rate (QRS complex) is 40-60beats/min.

The P wave occurs before, during or after the QRS complex. P wave occurring before the QRS complex may negative or inverted especially in lead II,III, aVF & the PR interval will be short.QRS complexes are regular and narrow because the ventricle is depolarized using the normal conduction pathway.

Because of their depressant effects on the sinus rate and conduction time at the more proximal (AV nodal) segments, Beta-blockers and nondihydropyridine calcium channels antagonists (verapamil and diltiazem) are considered a common cause of the same. Both junctional rhythm & non paroxysmal junctional tachycardia has been described as ECG changes of digoxin toxicity. In a study conducted by David Zeltser et al[1] Atrioventricular block that was "truly caused by drugs" was found in only 15% of patients. Sinus bradycardia with junctional escape beats was the most common ECG finding in drug related bradycardia as described by Jang Hoon Lee, et al.[2]Bindon MJ reported a case of Glucagon treatment for bradycardia and a junctional rhythm caused by excessive beta-blockade.[3]Similar to the above mentioned study drug was found to be responsible in16% patients & majority of them returned to sinus rhythm after with drawl of the offending drug(60%).[2]One patient receiving beta blocker had hypothyroidism also however sinus rhythm was restored after withdrawing beta blocker only.

Sinus node dysfunction may present as severe sinus bradycardia, sinus pause or arrest, periods of junctional rhythm, and/or alternating tachycardia-bradycardia periods. Sinus node dysfunction increases in

frequency with age of the patient. The aetiology remains unproven but the most probable seems to be the loss of the inherent rhythmicity of the sinoatrial node associated with a primary degenerative disease. In the study by Eraut and Shaw[4] Junctional rhythm was observed at one time or another in I5 patients & the frequency of junctional rhythm does not appear to have been emphasized.

Hypothyroidism is usually associated with bradycardia, and AV block occurring in patients with such a disorder is thought to be reversible and curable with levothyroxine therapy. There are few such case reports in literature.[5,6,7] No data are available regarding prevalence of junctional rhythm in hypothyroid state. Not only overt hypothyroidism but also subclinical hypothyroidism is reported to be a possible cause. In our study we found overt hypothyroidism as a cause in only one patient. Subclinical hypothyroid was seen in one patient having sick sinus syndrome. Similar to one case report in literature we had one patient with junctional bradycardia from overt hypothyroidism that responded to levothyroxine replacement.

The present study was conducted with the following aims & objectives:

(1) Prevalance of patients presenting with ECG features s/o junctional rhythm, their presenting symptomatology, disease course and management.

(2) To find out the different etiological as well as risk factors responsible for the said ECG changes, evolution of the changes with rectifying these factors and prognosis.

II. MATERIALS AND METHODS

Study Design:

Among all the patients with ECG findings of junctional rhythm 50 patients were randomly selected to find out the different etiologies who were admitted in department of cardiology & followed up in DEPARTMENT OF CARDIOLOGY, ICVS, IPGME&R, S.S.K.M. Hospital, Kolkata from 2016 – 2017.

Inclusion Criteria:

All patients aged more than 14 years admitted in SSKM hospital with ECG diagnosis of junctional rhythm.

Exclusion criteria:

Following patients are excluded from study
- Critically ill moribund patients
- Age less than 14 years.
- Serious ventricular arrthymias (VT, VF)
- Cerebrovascular accident

Study tools & techniques:

Various epidemiological, clinical, hematological, biochemical, radiological, angiographic parameters were recorded in these patients, as described below.

- All patients admitted in SSKM hospital with ECG findings of junctional rhythm were examined in detail.
- Detail history was taken which included h/o syncope, h/o angina, h/o diabetes, h/o intake of offending drugs e.g. beta blocker, calcium channel blocker, digoxin, other antiarrythmic agents, h/o diabetes, hypertension or hypothyroidism, past h/o myocardial infarction or coronary angiography
- A base line ECG was obtained immediately, troponin kit test and quantitative estimation of CPK-MB were done for all 50 randomized patients.
- Routine investigation like complete blood count, renal function test and lipids, fasting blood sugar, thyroid function test & electrolytes were done. Repeat blood counts, renal function & electrolytes were done in selected patients with initial abnormal results to monitor the effect of treatment as well as to assess prognosis.
- Echocardiography (vivid6 GE series) was performed in all 50 patients with junctional rhythm to assess any RWMA, LV function (ejection fraction), any valvular heart disease.
- Coronary angiography was performed in patients with an indication for the same and if not already done in past.
- 24 hr Holter monitoring (three channel) was performed in selected patients as indicated.
- Management of each case was also recorded.

Statistical Analysis:

For statistical analysis data were entered into a Microsoft excel spreadsheet and then analyzed by SPSS 24.0 and Graph Pad Prism version 5. Data had been summarized as mean and standard deviation for numerical variables and count and percentages for categorical variables.

III. RESULT AND ANALYSIS

We found that the mean age (mean± s.d.) of patients was 54.0400±11.5811years with range 22.00-74.00 years and the median age was 55.00 years.
It was found that 23(46%) patients were female and 27(54%) patients were male.
We found that 34(68%) patients had no H/O intake of offending drug, 10(20%) patients had beta blocker, 1(2%) beta blocker + CCB, 1(2%) patients had DIGOXIN, 2(4%) patients had DIGOXIN+BETA blocker, 1(2%) patients had VERAPAMIL and 1(2%) patients had yellow oleander
We found that 34(68%) patients had no H/O intake of offending drug and 16(32%) patients had H/O intake of offending drug.
31(62%) patients had no H/O SYNCOPE and 19(38%) patients had H/O SYNCOPE.
It was found that 29(58%) patients had noH/O ANGINA and 21(42%) patients had H/O ANGINA.
We found that 31(62%) patients had no H/O DIABETES and 19(38%) patients had H/O DIABETES.
It was found that the mean BMI (mean± s.d.) of patients was 24.3556 ± 4.6375.
It was found that the mean PULSE (mean± s.d.) of patients was 46.5600±14.6024
We found that the mean SBP (mean± s.d.) of patients was 129.6000±21.1506.
It was found that the mean DBP (mean± s.d.) of patients was 83.0800±7.2191.
We found that the mean HEMOGLOBIN (gm%) (mean± s.d.) of patients was 10.7680±1.0287.
It was found that the mean Urea (mean± s.d.) of patients was 34.7800±7.9006.
It was found that the mean Createnine (mean± s.d.) of patients was 1.4352±.5403.
We found that 39(78%) patients had no RENAL DYSFUNCTION and 11(22%) patients had RENAL DYSFUNCTION.
It was found that the mean TLC(4000-10000) (mean± s.d.) of patients was 9169.9200±2833.7632.
We found that the mean Na (mean± s.d.) of patients was 132.7400±4.7026.
It was found that the mean K (mean± s.d.) of patients was 4.8076±.8909.
We found that 35(70%) patients had CK-MB not raised and 15(30%) patients had CK-MB raised.
We found that 38(76%) patients had no HYPERKALEMIA and 12(24%) patients had HYPERKALEMIA.
It was found that 3(6%) patients had TROP-T faint positive, 32(64%) patients had TROP-T negetive, 15(30%) patients had TROP-T positive.
We found that the mean FBS (mean± s.d.) of patients was 119.3800±30.1059.
It was found that the mean TSH (mean± s.d.) of patients was 4.7926±4.0908.
We found that 47(94%) patients had no hypothyroidism and 3(6%) patients had hypothyroidism.
We found that the mean EF (mean± s.d.) of patients was 53.9600±8.2955.
It was found that 27(54%) patients had no RWMA, 5(10%) patients had Absent (GLOBAL)RWMA, 18(36%) patients had RWMA.
We found that 10(20%) patients had SSS(24 HR HOLTER).

IV. DISCUSSION

In our present study we sought to explore the prevalence of patients with ECG diagnosis of junctional rhythm during their hospital visit and also find out the relative distribution different etiologies of the same by evaluating 50 such patients who were randomly selected. Apart from some isolated case reports so far very few data are available in literature till date estimating the prevalence, distribution and different etiologies of junctional rhythm.

In the study by Hingorani et al[8] which analysed 24 hr holter recording of 1273 total healthy volunteers prevalence of junctional **rhythm was 0.2%** and that was exclusively observed in individual less than 45yrs age. However in that study Individuals with a body mass index <18 or >30 kg/m2 were excluded, as were subjects with clinically significant abnormality at the screening medical assessment which included history, physical examination, clinical laboratory tests, and an ECG. Only subjects with no history of drug or alcohol abuse who were nonsmokers or had not used tobacco or nicotine products in the preceding 6-month period were included. Female subjects were included only if they were not pregnant. Subjects aged > 65yrs were excluded. A Study by Ilson BE[9] showed that certain arrhythmias are frequently noted in normal healthy adults, including sinus bradycardia, sinus arrhythmia, the Wenckebach type of second-degree AV block (Mobitz I), atrioventricular junctional rhythm, PAC's, and PVC's. A survey was made by Tàmmaro AE et al[10] on a population of 6059 subjects aged more than 60 years with the aim to assess 1. The prevalence of heart arrhythmias and 2. The

relationships between arrhythmias and some other ECG alterations. Arrhythmias resulted present in 29.0% of the whole population with a significantly higher prevalence among males (30.7% vs. 28.1%, P less than 0.05) and among subjects over 75 years of age (33.2% vs. 23.9%, P less than 0.001). Supraventricular extrasystoles, atrial fibrillation and ventricular extrasystoles were the most frequent arrhythmias, followed by sinus bradycardia (SB, 2.04%), sinus arrhythmia (SA, 1.35%), atrial flutter (AFL, 1.09%) and **junctional rhythms (JR, 0.20%).**All the above arrhythmias, with the exception of AFL and JR resulted significantly more frequent among subjects over 75. A significantly higher prevalence of ECG signs of left ventricular hypertrophy, ischemia, previous myocardial infarction (MI) and of the so-called "minor" T-wave changes (MTC) was found among the subjects with arrhythmia as compared with those free from rhythm disturbances. To determine the prevalence of palpitations, cardiac arrhythmias and associated cardiovascular risk factors in an ambulatory elderly population,[11] 1454 ambulatory elderly people (219 men and 1235 women, age range 60-94 years) were assessed in a territory-wide health survey including anthropometric measurements, biochemical blood tests, questionnaire interview and resting surface ECG examination. Prevalence of palpitations and ECG abnormalities were determined and correlated with coronary risk factors and biochemical blood tests. Palpitations were present in 121 subjects (8.3%) and cardiac arrhythmias were found in 183 subjects (12.6%). Conduction abnormalities and sinus bradycardia were the commonest findings (9.8%).Compared with those without arrhythmia on ECG, people with arrhythmias were predominantly males and were older, had a higher prevalence of smoking and coronary heart disease.

In the present study which included all the admitted patents in our cardiology department as well as patients visiting the arrhythmia clinic over a period of one year junctional rhythm was found to be present in 53 patients out of a total 11,680 patients which corresponds to an estimated prevalence of 0.45%.

Out of these 53 patients 50 patients were randomly selected and further evaluated to find out the underlying condition seems to be responsible for the same. Evaluation was done as per the protocol stated before.

In our study among all these 50 patients with junctional rhythm male: female ratio was 1.17:1.So far the literature states that it occurs almost equally in male & female. Symptoms pertaining to the junctional rhythm itself such as syncope were present in only10% patients which show similarity with the literature statement that patients with junctional rhythm are often asymptomatic.

In the present study CAD (newly diagnosed or previously known) was present in38% patients which keeps similarity with the study conducted by Gwang Sil Kim et al[12] in which it was present in 40% cases. Similar to the said study h/o diabetes was present in 38% patients in our study group. Arrythmia occur as a frequent complication after myocardial infarction.[13]Bradyarrythmia includes sinus bradycardia, junctional bradycardia, or idioventricular bradycardia. Most of these arrhythmias are associated with inferior AMI.[14-20,21,22] In our study also acute myocardial infarction was the principal cause behind junctional rhythm which accounted for 28% cases and majority had RCA occlusion.

Electrolyte disturbance particularly hyperkalemia is an important cause of junctional rhythm. Among the different ECG changes of hyperkalemia junctional rhythm is often found to be one. An increased likelihood of short-term adverse event was found for hyperkalemic patients whose ECG demonstrated QRS prolongation, bradycardia (HR<50), and/or junctional rhythm.[23]Severe hyperkalemia was found in 21.3% patients with bradycardia in a study conducted by Chon SB et al.[24]Similar to that finding we found hyperkalemia as an underlying etiology in24% patients. Most of these patients had renal dysfunction and 5 of them (total 12) were also receiving beta blocker which is similar to the study published by Isabel J et al[25].In all these patients sinus rhythm was restored with correction of serum potassium level including those who were receiving beta blocker and the drug could be resumed in selected patients with hard indication although at a lower dosage.

Because of their depressant effects on the sinus rate and conduction time at the more proximal (AV nodal) segments, Beta-blockers and nondihydropyridine calcium channels antagonists (verapamil and diltiazem) are considered a common cause of the same. Both junctional rhythm & non paroxysmal junctional tachycardia has been described as ecg changes of digoxin toxicity. In a study conducted by David Zeltser et al[1] Atrioventricular block that was “truly caused by drugs” was found in only 15% of patients. Sinus bradycardia with junctional escape beats was the most common ECG finding in drug related bradycardia as described by Jang Hoon Lee, et al.[2]Bindon MJ reported a case of Glucagon treatment for bradycardia and a junctional rhythm caused by excessive beta-blockade.[3]Similar to the above mentioned study drug was found to be responsible in16% patients & majority of them returned to sinus rhythm after withdrawl of the offending

drug(60%).[2]One patient receiving beta blocker had hypothyroidism also however sinus rhythm was restored after withdrawing beta blocker only. There were 5 patients in our study who were receiving beta blocker in the setting of hyperkalemic renal failure in whom sinus rhythm was restored with correction of serum electrolytes and in most of them beta blocker could be resumed in follow up if indicated.

We found sick sinus syndrome as diagnosed by 24hr holter monitoring in a total 10 patients (20%).Keeping in similarity with the other literatures majority of these patients had h/o syncope and drug history was positive in two of them and one had subclinical hypothyroidism.

In our study we found overt hypothyroidism as a cause in only one patient. Subclinical hypothyroid was seen in one patient having sick sinus syndrome.

Accelerated junctional rhythm has been described in post-RF ablation of AVNRT.[26]In the present study we had one such patient. A malignant variation of accelerated junctional rhythm is junctional ectopic tachycardia (JET). JET occurs almost exclusively in neonates or young children undergoing major congenital cardiac surgery.[27]In our present study we had one such patient.

There are numerous toxins that can cause, in overdose, electrocardiogram (ECG) changes, even in patients without history of cardiac pathology. Very few case reports are published. We had on patient having junctional bradycardia with h/o yellow oleander ingestion and in the absence of any other factors or cardiac ailments.

V. CONCLUSION

- Junctional rhythm is a less frequent rhythm disturbance in comparison to other brady and tachyarrythmias.
- Equally distributed in male & females and can be found in all age groups.
- More often it has a benign course.
- Major causes of this includes hyperkalemia, AMI, drugs and sick sinus syndrome.
- Most of the time the underlying causes are reversible and does not require implantation of permanent pacemaker.

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Table: Distribution of mean in all parameters

	Number	Mean	SD	Minimum	Maximum	Median
Age (yrs)	50	54.0400	11.5811	22.0000	74.0000	55.0000
BMI	50	24.3556	4.6375	16.5000	34.6000	23.9900
PULSE	50	46.5600	14.6024	36.0000	138.0000	44.0000
SBP	50	129.6000	21.1506	90.0000	166.0000	126.0000
DBP	50	83.0800	7.2191	70.0000	104.0000	80.0000
HEMOGLOBIN	50	10.7680	1.0287	7.8000	12.6000	11.0000
Urea	50	34.7800	7.9006	20.0000	56.0000	34.0000
Createnine	50	1.4352	.5403	0.7000	3.6000	1.3000
TLC	50	9169.9200	2833.7632	5600.0000	19870.0000	8750.0000
Na	50	132.7400	4.7026	118.0000	144.0000	132.0000
K	50	4.8076	.8909	3.7000	6.8000	4.6000
FBS	50	119.3800	30.1059	76.0000	172.0000	107.0000
TSH	50	4.7926	4.0908	2.4600	31.6000	3.9500
EF	50	53.9600	8.2955	38.0000	66.0000	56.0000

Table: Distribution of all parameters

		Frequency	Percent
Sex	**FEMALE**	23	46.0%
	MALE	27	54.0%
H/O intake of offending drug(yes/no)	**NO**	34	68.0%
	YES(BETA BLOCKER)	10	20.0%
	YES(BETA BLOCKER+CCB)	1	2.0%
	YES(DIGOXIN)	1	2.0%
	YES(DIGOXIN+BETA BLOCKER)	2	4.0%
	YES(VERAPAMIL)	1	2.0%
	YES(YELLOW OLEANDER)	1	2.0%
H/O intake of offending Drug(yes/no)	**Absent**	34	68.0%
	Present	16	32.0%
H/O syncope(a/p)	**Absent**	31	62.0%
	Present	19	38.0%
H/O angina	**NO**	29	58.0%
	YES	21	42.0%
H/O diabetes	**NO**	31	62.0%
	YES	19	38.0%
Renal dysfunction(A/P)	**Absent**	39	78.0%
	Present	11	22.0%
CK-MB	**NOT RAISED**	35	70.0%
	RAISED	15	30.0%
Hyperkalemia (A/P)	**Absent**	38	76.0%
	Present	12	24.0%
Trop-t	**FAINT POSITIVE**	3	6.0%
	NEGATIVE	32	64.0%
	POSITIVE	15	30.0%
Hypothyroidism	**Absent**	47	94.0%
	Present	3	6.0%
RWMA(P/A)	**Absent**	27	54.0%
	Absent (GLOBAL)	5	10.0%
	Present	18	36.0%
CAG	**DIFFUSE TVD**	1	2.0%
	DIFFUSE TVD(PREV CAG) ICMP	1	2.0%
	DVCAD(LAD/LCX)	1	2.0%
	DVCAD(LCX/RCA)	1	2.0%
	MINOR CAD	1	2.0%
	NO	9	18.0%
	NOT DONE	22	44.0%
	SVCAD(LCX)	3	6.0%
	SVCAD(RCA)	9	18.0%
	TVCAD	1	2.0%
	TVD(PREV CAG)/ICMP	1	2.0%
24 hours holter	**SSS(24 HR HOLTER)**	10	20.0%
	Others	40	80.0%

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