CLINICAL PROFILE AND IN-HOSPITAL OUTCOME OF PATIENTS WITH RIGHT VENTRICULAR MYOCARDIAL INFARCTION IN INFERIOR WALL MYOCARDIAL INFARCTION

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ABSTRACT

BACKGROUND
The present study deals with the clinical profile of right ventricular infarction as diagnosed by right precordial electrocardiography in patients with acute inferior wall myocardial infarction. Recognition of the syndrome of RVI is important, as it identifies a significant clinical entity which is associated with and is an independent predictor of major complications and in hospital mortality after acute IWMI. The main purpose of this study was to become aware of RVI and its complications like hypotension, arrhythmias and conduction disorders, which not only need appropriate therapy but also requires avoidance of inappropriate therapy, which may reduce right ventricular filling pressure and cardiac output further worsening the condition.

Aims and Objective - The study emphasises on the incidence of RVMI in patients of acute inferior wall MI using right precordial ECG, the clinical profile of patients with RVMI in acute IWMI and to study the incidence of complications in patients of acute IWMI.

MATERIALS AND METHODS
This is a prospective study carried out on patients admitted with inferior wall MI in our tertiary care hospital from March 2015 to May 2016. At the time of admission, a seventeen lead ECG consisting of twelve conventional leads and additional right precordial leads V3R, V4R, V5R, V6R was taken and subsequent recordings were made for conventional and right precordial leads.

RESULTS
Out of 90 patients of acute IWMI admitted to ICCU Department of Medicine, 25 patients were diagnosed with RVMI (28%). Patients were continuously monitored during hospital stay and their serial ECGs clinical course and response to treatment were analysed. There was five times increased incidence of mortality in patients of IWMI with RVMI as compared to no RVMI. Complications like cardiogenic shock, various arrhythmia viz. AV block, VT were higher in RVMI group.

CONCLUSION
RVMI occurs in a significant number of patients with inferior wall MI. It can be easily diagnosed with considerable accuracy by a simple bedside routine 12-lead ECG along with right-sided ECG leads. Early diagnosis of RVMI can prevent fatal complications like high degree heart blocks, ventricular arrhythmias, cardiogenic shock and high in-hospital mortality. Patients who survive the acute phase have no long-term consequences and complete recovery over a period of weeks to months is the rule in majority. Therefore, right-sided ECG should be taken routinely in all patients of IWMI.

KEYWORDS
Inferior Wall Myocardial Infarction, Right Ventricular Infarction, Electrocardiography, Jugular Venous Pressure.

BACKGROUND
Myocardial infarction was previously thought to be a disease of mainly the left ventricle. Right ventricular infarction was just a pathological entity. In 1974, Cohn[1] for the first time described potentially serious and unique haemodynamic consequences of right ventricular infarction. The advent of more sophisticated diagnostic techniques and more precise

haemodynamic measurement has demonstrated that right ventricular infarction is a well-defined clinical entity and value of recognising patients with predominant right ventricular dysfunction is related not only to instituting appropriate therapy for severe pump failure, but also to avoid inappropriate therapy. It has also been shown that right ventricular infarction occurs most commonly in association with inferior myocardial infarction or inferoposterior myocardial infarction.[2] Although, isolated right ventricular infarction had been described in autopsy reports as less than 3% of all acute myocardial infarction,[2] the incidence of right ventricular infarction associated with IWMI has been shown to be as high as 30% - 50%.[3,4,5]

It results primarily from occlusion of the right coronary artery and infrequently from involvement of the left anterior descending artery and occasionally in inferoposterior left ventricular infarction also.[2,4]

Right ventricular myocardial infarction can lead to diminished right-sided stroke volume with concomitant right ventricular dilatation and septal changes. The potential haemodynamic derangement associated with right ventricular infarction renders the patient's unusually sensitive to diminished ventricular preload. These two circumstances can result in a severe decrease in right and secondarily left ventricular output resulting in a clinical triad of hypotension and jugular venous pressure distension in the presence of clear lung fields.[6,7,8]

It is known that ST segment elevation of 0.1 mV or greater in one or more of right precordial leads V4R to V6R is highly sensitive (90%) and specific (91%) in identifying acute right ventricular infarction.[9,10]

A subtle clue to the presence of haemodynamically significant right ventricular infarction is a marked sensitivity to preload reducing agents such as nitrates, morphine or diuretics. Prompt fluid therapy may abort the vicious cycle set in motion by right ventricular infarction, which if treated in conventional way or neglected tends to lead to true cardiogenic shock.[11,12]

Recognition of the syndrome of RVI is important, as it identifies a significant clinical entity, which is associated with and is an independent predictor of major complications and in-hospital mortality after acute inferior MI.[12,13]

It is associated with increased risk of death, shock, ventricular tachycardia or fibrillation and Atrioventricular Block (AVB) and a higher mortality rate for the first month post MI in patients with RVMI, but without haemodynamic impairment. These complications in RVMI may be due to the increased parasympathetic tone, Sinoatrial (SA) node dysfunction, Atrioventricular (AV) node dysfunction.[4,13,14]

Thus, the present study deals with clinical profile of right ventricular infarction as diagnosed by right precordial electrocardiography in patients with acute inferior myocardial infarction. Main purpose of selecting this study was to become aware of right ventricular infarction and its various complications like arrhythmias and conduction disorders, which not only requires appropriate therapy but also avoidance of inappropriate therapy that might reduce right ventricular filling pressure and cardiac output and in turn prove to be disastrous.

Aims and Objective
The present study titled incidence, clinical profile and in-hospital outcome of patients of RVMI in IWMI was carried out in patients admitted in Department of Medicine, S. S. Medical College and Associated S. G. M. H, Rewa (M. P.) from March 2015 to May 2016 with the following aims and objectives -

1. To study the incidence of right ventricular infarction in patients of acute inferior myocardial infarction using right precordial electrocardiography.
2. To study the clinical profile of patients of RVMI in acute IWMI.
3. To study the incidence of complications in patients of acute IWMI with and without RVMI.
4. To compare the mortality in patients of IWMI with and without RVMI.
5. To study the response of specific therapy in patients with right ventricular infarction.

MATERIALS AND METHODS
This was a prospective clinical study conducted on 90 patients between March 2015 - May 2016 admitted in ICCU of our Tertiary Care Centre with IWMI.

At the time of admission, a seventeen lead ECG consisting of twelve conventional leads and additional right precordial leads V3R, V4R, V5R, V6R were taken and subsequent recordings made for conventional and right precordial leads. For recordings, a single channel ECG machine was used. All ECG's were recorded in 25 mm/second 10 mV setting. The points on chest wall used for recording chest leads were marked with a skin pencil, so that same points could be used serially in a given patient. Serial ECG's were taken on admission, at the end of 6 h, end of 12 h and daily till ST segment became isoelectric in right precordial leads. ECG's were also repeated whenever patient complained of chest pain. Patients were continuously monitored. The diagnosis of acute inferior wall myocardial infarction was made as typical history of chest pain, ST segment elevation in leads II, III and avF and by development of pathological Q waves in the above mentioned leads. Tall 'R' waves in V1 and V2 and increased serum cardiac enzymes Troponin-T [qualitative]. The diagnosis of right ventricular myocardial infarction will be made as by the criteria of ST segment elevation of 0.1 mV or more in one or more of the right precordial leads (V3R, V4R, V5R and V6R) in those patients who satisfied the criteria for an inferior wall myocardial infarction.

Patients were classified into Two Groups
Group A: Inferior wall infarction with right ventricular infarction.
Group B: Inferior wall infarction without right ventricular infarction.

Their clinical course were studied and compared. The clinical course and ECG analysis was compared in both the groups during their entire hospital stay. Complications viz. ventricular tachycardia, AV blocks, hypotension, cardiogenic shock and deaths in both the groups were analysed and compared.

Inclusion Criteria
All patients with definite evidence of acute inferior wall myocardial infarction as proved by 12-lead ECG along with right precordial leads and chest pain of duration less than 24 hours were considered in our study.

Exclusion Criteria
1. History of chest pain of more than 24 h duration.
2. Patients whose initial ECG's showed an anteroseptal or anterior wall myocardial infarction will be also excluded, because these infarctions may produce an anteriorly oriented ST vector which may also cause ST segment elevation in the right precordial leads. For the same reason patients with pericarditis, left bundle branch block were excluded.
3. Patients with chronic lung disease, cor pulmonale were also excluded because they may be associated with a right ventricular dysfunction.
4. Patients with previous history of a myocardial infarction were also excluded to avoid a false positive result for right precordial electrocardiography.

Following Clinical Parameters were considered for Comparison viz.,

1. Hypotension was defined as systolic arterial blood pressure < 90 mmHg or which has declined by at least 30 mmHg below previous level.
2. Right heart failure was considered when the hepatojugular reflux or abdominal compression test will be positive. JVP more than 4 cm in 45° position with pedal oedema and systemic venous congestion.
3. Ventricular tachycardia.
4. AV block 2º and higher.
5. Death.
6. Cardiogenic shock.

Left heart failure was considered when bilateral pulmonary rales was present on inspiration; dullness on percussion over lung bases and on auscultation S3 and S4 were present. Peripheral pulse shows pulsus alternans. Chest X-ray shows Kerley B lines. Present study deals with clinical profile of right ventricular infarction as diagnosed by right precordial electrocardiography in patients of acute inferior wall myocardial infarction. The results obtained were analysed for their statistical significance using Fisher's exact test and unpaired 't' test.

RESULTS

90 patients of acute inferior wall MI were admitted in ICCU, Department of Medicine during the study period from March 2015 to May 2016 and their serial 12-lead ECGs along with right-sided leads RV3, RV4 were taken at the time of admission, then every six hourly and thereafter whenever the patient complained of chest pain. Patients were continuously monitored during hospital stay and their serial ECGs, clinical course and response to the treatment was analysed.

Out of 90 patients admitted with IWMI, 25 patients had RVMI (28%) and 65 patients were without RVMI (72%) in IWMI (Table 1). Incidence of RVMI was more in females (36%) as compared to males (22%); 24% hypertensive patients had RV involvement, while in diabetics the incidence was 30%. Tobacco and positive family history contributed significantly in IWMI with incidence being 40% and 34.4% respectively. Maximum number of patients presented with chest pain (100%) followed by perspiration (33%), dyspnoea (22%), palpitations (17.77%) and syncope (10%). Raised JVP was an important sign in RVMI (60%) as compared to patients without RVMI (30%) (Table 2). There was five times increase in mortality in patients of IWMI with RVMI (16%) as compared to (3%) in no RVMI group (Table 3). There was 5% mortality in the thrombolysed group compared to 17% in non-thrombolysed group in IWMI, suggesting that mortality was higher in non-thrombolysed group in IWMI. In RVMI also mortality was higher in non-thrombolysed (50%) patients compared to thrombolysed patients (13%). There was a drastic increase in mortality in patients of RVMI who present late after the onset of symptoms from nil presenting within 7 hours to 37.5% within 8 - 12 hours and 50% after 12 hours. Incidence of cardiogenic shock was much higher among patients with RVMI (16%) as compared to (3%) in patients without RVMI. There was doubling of incidence of AV blocks in RVMI patients (24%) as compared to non-RVMI (12.3%). There was doubling in the incidence of ventricular tachycardia in RVMI patients (12%) as compared to without

RVMI (6%). There was more than three times increase in incidence of hypotension in RVMI patients (40%) as compared to non-RVMI patients (12.3%) (Table 4).

	No. of IWMI Patients	%
No. of Patients with RVMI	25	28%
No. of Patients without RVMI	65	72%
Total Patients	90	100%
Table 1. Incidence of RVMI in Inferior Wall MI		

In the above table among 90 patients of AC IWMI, 25 patients were found to have right ventricular infarction which accounts for 28% of incidence.

	Total Patients	Raised JVP	%
RVMI	25	15	60%
Without RVMI	65	20	30%
Total Patients	**90**	**35**	**39%**
Table 2. Jugular Venous Pressure			

P = 0.01

In the above table, out of 25 patients with right ventricular involvement, 15 patients (60%) had their JVP raised, whereas it was increased only in 20 patients out of 65 patients (30%) of IWMI without right ventricular involvement.

	IWMI Patients	Deaths	%
RVMI	25	4	16%
Without RVMI	65	2	3%
Total Patients	**90**	**6**	**6.66%**
Table 3. Mortality in IWMI Patients with and without RVMI			

P = 0.04

In the above table, out of total mortality of 6 patients, 4 deaths occurred in RVMI group, which accounts for 16% mortality compared to 2 deaths in IWMI without RVMI group out of 65 patients, which is equal to 3% mortality in that group.

	IWMI Patients	Hypotension	%
RVMI	25	10	40%
Without RVMI	65	8	12.3%
Total Patients	**90**	**18**	**20%**
Table 4. Incidence of Hypotension in IWMI Patients with and without RVMI			

P = 0.006

In the above table, out of 25 patients with RVMI 10 patients had hypotension during their hospital stay, which is equal to 40% incidence, while only 8 patients in IWMI

without RV involvement had hypotension accounting for 12.3% incidence.

DISCUSSION

In the present study entitled Incidence, clinical profile and in-hospital outcome of patients of RVMI in IWMI, 90 patients with acute inferior wall MI who were admitted in Medicine ICCU Ward with chest pain duration of < 24 hours were taken, because most ECG changes of acute myocardial infarction subsides within 24 hours. Patients who have history of COPD and with previous MI were not taken in this study, as these may act as confounding factors in the diagnosis of right-sided heart condition.

In the present study, most patients were in the age range of 41 - 80 years with slightly more patients falling in the age group of 41 - 60 years in both RVMI+ and RVMI- groups. This was not significant P > 0.05, so this may be a chance association. This may be due to non-consecutive selection of patients. It shows that right ventricular myocardial infarction does not show any preponderance to a particular age group.

The mean age of our study population was 59.7 years, which correlated very well with the study of Mehta et al.[13]

In the present study population, there were 57 male patients (63%) and 33 female patients (37%). There were 13 patients with RVMI among 57 male patients of IWMI, which accounts for 22% incidence of RVMI in male patients. Of 33 female patients 12 were diagnosed to have right ventricular infarction, which accounts for 36% incidence of RVMI in female population. Incidence of RVMI was found to be more in female population. Since (p > 0.05) this correlation was not significant, although increased incidence of RVMI was also reported by Mehta et al,[13] but this was also statistically insignificant.

So, distribution of RVMI did not show any sexual predilection.

Hypertension

21 out of 90 patients of IWMI gave the history of hypertension, which accounts for 23% prevalence of hypertension. In the present study group among RVMI patients, 5 patients were hypertensive accounting for 20% prevalence in the RVMI group. Among 65 patients without RV involvement 16 patients were hypertensive leading to a prevalence of 24% in that group, but P value of this correlation was 0.7 indicating that this correlation might be by chance and no actual relationship between hypertension and increased incidence of RVMI exists.

However, Iqbal et al[15] found hypertension prevalent among 44% population of IWMI. However, this may be affected by different geographical, cultural and educational strata of the study population.

This shows that although hypertension is a well-known risk factor for coronary artery disease, it did not affect RV involvement.

Diabetes Mellitus

Type 2 diabetes mellitus was found in 27 out of total 90 patients of IWMI accounting for 30% incidence in the study population, whereas prevalence in RVMI group was 6 patients out of 25 patients (24% prevalence) and prevalence in patients without RVMI was 21 out of 65 patients (32% prevalence). But P > 0.05 in this association therefore

appears to be no significant correlation between RV involvement and diabetes mellitus.

Similar results were found by Mehta et al,[13] who found 18% prevalence of T2DM in IWMI patients with 16% in RVMI group, 15.9% among non-RVMI group.

Risk Factors other than T2DM and Hypertension were as follows

40% patients were tobacco chewers and 29% were smokers, 9% patients were both smokers and tobacco chewers, 12% had history of alcohol abuse and 34% had family history of CAD. All these are well-known risk factors for CAD and other vascular disease.

All these risk factors were distributed in both RVMI+ and RVMI- groups and were not found to be associated specifically with either of groups p > 0.05.

Presenting Complaints

In the present study almost all patients presented with chest pain (100%), 22% patients also complained of breathlessness, while 17.77% patients also had palpitations, 33% patients complained of perspiration and 10% patients had syncope or giddiness.

Clinical Examination

In the present study, JVP was found to be elevated in 35 patients of IWMI, out of which 15 had RVMI which accounts for 60% of patients with RVMI, while it was raised in only 30% patients without right ventricular involvement. Since P value of this observation < 0.05, it is considered significant. So, it can be concluded that raised JVP is significantly associated with RVMI. However, in some other studies the percentage of raised JVP was found to be much higher.[16]

Since it is a clinical parameter, subjective variation in the observation is likely. Nonetheless, elevated JVP remains strongly associated with RVMI.

Also, some studies have shown that since left ventricular function is also depressed secondary to RVMI, this along with conduction disorders associated with RVMI offsets the clinical manifestations of RVMI.

Incidence of RVMI in Inferior Wall MI

In the present study, RVMI was diagnosed in 25 patients out of 90 patients of IWMI.

Incidence of RVMI is variable depending upon the criteria used to diagnose it, so in many other studies its incidence varies from 10% to 50%.[4,11,13,15] In the present study we used ECG criteria as the sole method to diagnose RVMI, while most other studies have used echocardiography, right heart catheterisation and technetium scan to diagnose RVMI[3,13,16] and because ST elevations in lead V4R mostly persists up to 12 hours after the MI, some patients are likely to be missed.

Mortality in IWMI Patients

In the present study, we followed the clinical course of 90 hospitalised IWMI patients. Out of 90 patients, we had 6 deaths leading to 7% mortality. Out of 6 deaths, 4 were diagnosed to have RVMI leading to mortality of 16% in RVMI group, whilst 2 deaths occurred in non-RVMI group leading to 3% mortality in that group.

There were 4 deaths among 25 RVMI patients (16% mortality) and 2 deaths out of 65 patients without RVMI (3%

mortality). Mortality was significantly higher in RVMI group P < 0.05.

Also in the study by Mehta et al[13] mortality was higher in RVMI subgroup, 7.1% of IWMI compared to 5.4% in non-RVMI group.

Therefore, presence of right ventricular infarction in IWMI qualifies as high risk group. But mortality rate varies among different studies due to different management protocols and facilities available in different centres.

We divided IWMI patients according to whether they were thrombolysed or not. Out of 90 patients, 78 patients were thrombolysed including RV+ and RV- patients and 12 patients were treated without thrombolysis.

There were 4 deaths among thrombolysed patients leading to 5% mortality compared to 17% mortality in non-thrombolysed patients (P = 0.15).

Similarly, patients with exclusively RVMI were divided in the thrombolysed and non-thrombolysed group, mortality was higher in non-thrombolysed sub-group (50%) compared to 13% mortality in thrombolysed group.

In both IWMI group and RVMI group, mortality improved after thrombolysis more so in RVMI group. Many studies show improved outcome after successful thrombolysis.[17,18,19,20,21,22,23]

TIMI second phase also shows reduction in the incidence of mortality in RVI patients with successful thrombolysis.[19]

Even though mortality improved with thrombolysis, in the present study it was statistically insignificant P > 0.05, but outcome improved.

Berger et al[21] found thrombolytics reduces incidence of RVI.

O'Rourke[22] recommends that patients with evidence of RVI by ECG criteria or by other non-invasive methods, who have clinical evidence of depressed right ventricular function are candidates for coronary angioplasty (if available), thrombolytic therapy (if not contraindicated).

Chest Pain Duration
In the present study, we classified our study cohort into 3 groups according to duration of chest pain; 48 patients out of 90 presented within 7 hours of onset of chest pain, 30 patients presented with chest pain duration of 8 - 10 hours.

In patients presenting within 7 hours of onset of chest pain no death occurred, while 4 deaths occurred in the 8 - 12 hours group accounting for 13.33% mortality. Out of 12 patients presenting with 12 - 24 hours chest pain, 2 deaths occurred accounting for 16.66% mortality.

Similarly, in RVMI subgroup also, maximum no. of patients presented within initial 7 hours and had better prognosis compared to the late presenters with 3 deaths occurring in 8 - 12 hours subgroup accounting for 37.5% mortality, whilst of 2 patients presenting after 12 hours 1 death occurred.

In both IWMI and RVMI alone, prognosis was better in early presenters compared to late presenters and this difference was more prominent in the RVMI group and this was found to be significant P < 0.05 in the RVMI group.

In IWMI without RVMI too mortality was higher among late presenters with no mortality among early presenters and 4.5% mortality in patients with chest pain duration of 8 - 12 hours and 10% mortality among patient presenting with more than 12 hours chest pain.

In the present study, early presenters had lower mortality which may be because of early treatment with thrombolysis and reperfusion of ischaemic myocardium and timely management of haemodynamic compromise. It may be influenced in case of RVMI by early recognition of RV involvement by electrocardiogram in the early hours of IWMI as ECG changes indicating right ventricular infarction mostly settle early within 12 hours.

Complications
Cardiogenic Shock
Out of 25 patients with RVMI 4 patients had cardiogenic shock accounting for 16% incidence in that group, while only 2 patients out of 65 patients had cardiogenic shock (3%). This was significant (P = 0.04).

Mehta et al[13] also found 6.9% incidence of shock in RVI patients compared to 5.5% incidence without RVMI.

AV blocks: In the present study, we evaluated the incidence of 2º and higher blocks in patients with and without RVMI. Out of 25 patients with RVMI 6 had AV blocks, which is 24% incidence in that group, whilst 8 patients of IWMI without RVMI had AV blocks accounting 12.3% incidence. So incidence of AV blocks was found higher in RVMI group.

P = 0.01, therefore there is significant association between AV blocks and incidence of RVMI.

Mehta et al[13] also showed similar results in their study with 21% incidence in RVMI group compared to 9% incidence in non-RVMI group.

Bueno et al[14] in 1998 found AV blocks in 25% of patients of RVMI and 6.7% patient of IWMI without RVMI.

Prevention and management of AV blocks and bradyarrhythmia are very important, because they cause AV dyssynchrony leading to loss of atrial contribution to ventricular filling further jeopardising already decreased cardiac output.

Ventricular Tachycardia
Out of 25 patients with RVMI 3 had Ventricular Tachycardia which is 12% incidence in that group, whilst 4 patients of IWMI without RVMI had Ventricular Tachycardia accounting for 6% incidence. So, incidence of VT was found to be higher in RVMI group. Although p > 0.05, incidence was found to be twice that of patients without RVMI

Mehta et al[13] also found increased incidence of VT/VF (Three times) in patients with RVMI. They attributed increased mortality in RVMI group to increased arrhythmias.

Berger et al[21] reported 8.6% incidence in RVMI group compared to 2.5% in non-RVMI group.

Hypotension - It is one of clinical hallmark of RVMI. Out of 25 patients with RVMI 10 patients had hypotension during their hospital stay, which is equal to 40% incidence, whilst only 8 patients in IWMI without RV involvement had hypotension accounting for 12.3% incidence P = 0.006, which was very significant. Therefore, hypotension was much more common in patients with RVMI.

Mehta et al[13] reported hypotension in 29% patients with RVMI compared to 19.3% in patients of IWMI without RV involvement.

Hypotension - In RVMI occurs due to impaired filling of left ventricle secondary to impaired right ventricular cardiac output and decreased LV filling pressures, therefore volume

loading in case of RVMI is an important therapeutic approach to prevent cardiogenic shock.

CONCLUSION

Right ventricular myocardial infarction occurs in a significant number of patients with inferior wall myocardial infarction. Extent of right ventricular involvement varies from patient to patient and ranges from myocardial stunning to full blown infarction.

It represents a high risk subgroup of IWMI with distinct clinical presentation comprising of raised JVP and hypotension with clear lung fields, which requires a high degree of clinical suspicion.

However, it can be easily diagnosed with considerable accuracy by a simple bedside routine 12-lead ECG along with right-sided ECG leads. Since ECG changes are transient in right-sided ECG, it is difficult to diagnose RVMI after 12 hours. So, ECG should be supplemented by urgent routine echocardiography to improve diagnostic yield.

Frequent accompaniments of RVMI include atrial infarction, sinus bradycardia, ventricular tachycardia, atrial fibrillation, atrioventricular block and cardiogenic shock. Patients with RVI who are haemodynamically unstable should be managed with volume loading to maintain adequate right ventricular preload. Early recognition and prompt reperfusion with thrombolytic therapy or coronary angioplasty, rate and rhythm control and inotropic support may also be warranted since patients with RV infarction have high incidence of complications and in-hospital mortality.

Patients who survive the acute phase have no long-term consequences and complete recovery over a period of weeks to months is the rule in majority of patients, suggesting right ventricular "stunning" rather than irreversible necrosis.

Therefore, right-sided ECG should be taken routinely in all patients of inferior wall MI.

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