

Electrophysiological study of Landry Guillain Barre syndrome at a Tertiary care hospital

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Abstract

A reasonable description of what we now call Guillain-Barre' Syndrome (GBS) was offered in 1892 by Osler. The critical features of the illness were not fully synthesized until after the advent of diagnostic lumbar puncture near the end of this century (Osler, 1892). In 1916, Guillain, G and Barre' JA, then French army neurologists and Strohl, Al from England, simultaneously published reports. All adult patients, diagnosed as Guillain-Barre Syndrome, who strictly complied with the NINCDS criteria, admitted to medical college hospital were included in this study. This includes a retrospective study of four years and prospective study of one year. Ninety seven patients entered the study. Data collection was done according to the proforma attached. In retrospective cases, the information was collected from the patient's records. In the prospective group, a detailed history was taken, all patients were examined and then the data collected. Nerve conduction velocities (NCV) were studied in median, ulnar, common peroneal and posterior tibial nerves. Abnormal nerve conduction velocities were found in 46 (98%) of total. Distal latency was prolonged in 47 (100%) patients in the present study.

Keywords: Guillain-Barre 'syndrome, nerve conduction velocities, electrophysiological study

Introduction

Acute ascending paralytic illnesses have been recognised for centuries. The earliest report was by Landry in 1859 who described the presenting symptoms, course and outcome of this disease in 10 patients. His findings were those of a progressive paralysis beginning in the distal extremities, preceded by paresthesia and transitory muscle cramps. Two of his ten patients succumbed to respiratory failure. In those who recovered, the process was generally one of rapid recession in a descending pattern ^[1].

A reasonable description of what we now call Guillain-Barre' Syndrome (GBS) was offered in 1892 by Osler. The critical features of the illness were not fully synthesized until after the advent of diagnostic lumbar puncture near the end of this century (Osler, 1892). In 1916, Guillain, G and Barre' JA, then French army neurologists and Strohl, Al from England, simultaneously published reports. Their title can be translated as follows: "Concerning a syndrome of radiculoneuritis with increased albumin in the cerebrospinal fluid without

cellular reaction: remarks on its clinical characteristics and description of tendon reflexes" [2]. Later in 1918 came the controversy when Bradford, Bashford and Wilson reported 30 patients with disorder in a report entitled 'Acute infective polyneuritis' (Bradford *et al.*). They claimed to have isolated an organism transmissible into other primates. However, their claim of infectivity was retracted the following year. An allergic basis for this disease was put forward sporadically (Bannwarth; Furtado; Asbury, 1990) [3, 4].

Methodology

All adult patients, diagnosed as Guillain-Barre. 'Syndrome, who strictly complied with the NINCDS criteria, admitted to medical college hospital were included in this study. This includes a retrospective study of four years and prospective study of one year. Ninety seven patients entered the study.

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All the Patients underwent lumbar puncture on the day of admission and the cerebrospinal fluid was sent for the cell count, culture, protein content and sugar analysis. Each one of them had a complete haemogram and a urine examination.

Urine was also examined for porphobilinogen. Serum electrolytes and serum calcium levels were estimated on admission to rule out hypokalemic paralysis and hypoglycemic states. Liver function tests and HBsAg and HIV tests were done on all the patients. A throat swab was taken in patients with sore throat to rule out diphtheria.

Results

Table 1: Nerve Conduction Velocities

NCV m/s	Median motor	Ulnar	Common peroneal	Post tibial
25-30	8	10	20	24
31-35	17	16	15	13
36-40	14	11	9	8
41-45	6	6	2	1
46-50	2	3	1	1
More than 50	0	1	0	0

Table 2: Distal Latency response

Nerve studied	Prolonged	Not prolonged
Median	47	0
Ulnar	47	0
Common peroneal	47	0
Post tibial	47	0

Table 3: Conduction block

Nerve studied	No. of patents studied	No. of patients with conduction block
Median	25	23
Ulnar	25	23
Common peroneal	25	23
Post tibial	25	23

Table 4: Needle EMG

Abnormal	Present	Absent
Fibrillations	9	38
Positive sharp waves	8	39
Fasciculation	1	36

Table 5: Results of Electrophysiological study

Type of neuropathy	No. of patients	Percentage
Demyelinating	38	79.8
Mixed	5	10.5
Axonal	4	8.4

Discussion

Forty seven (48.4%) patients had electrophysiological studies. Majority of them were done during the period of recovery. One or more parameters were abnormal in all the patients. Nerve conduction velocities (NCV) were studied in median, ulnar, common peroneal and posterior tibial nerves. Abnormal nerve conduction velocities were found in 46 (98%) of total patients in this study as compared to 73% in the study by Kur *et al.* and 24% by Cornblath *et al.* [5]

Distal latency was prolonged in 47 (100%) patients in the present study against 74% in the study by Kalir and 40% in Cornblath *et al.* group [6, 7].

F wave latency was prolonged in 60% of patients, absent in 18.9% of patients and normal in 18.9% of patients in median nerve. In ulnar nerve, F waves were prolonged in 52% of patients, absent in 27.3% and normal in 18.9% of patients. In the common nerve, 31% had prolonged, 51.2% had absent and 17.04% had normal F waves. In posterior tibial nerve, 48% had prolonged, 52% had absent F waves.

H reflex: On right side, 63% patients had absent 35% had normal H reflex. On left side, 67.2% had absent and 31 % had normal H reflex. 23 out of 25 patients in whom H reflex was studied, it was abnormal [8].

Abnormal sensory nerve conduction was seen in (R) median sensory (62%), (R) ulnar sensory (63%) and Sural Sensory (67.5%) nerves.

Fibrillations were observed in 9 (19%) patients and absent in 38 (81%) of patients. Positive sharp waves were present in 39 (83.07%) patients and absent in 8 (16.9%) patients.

Conclusion

- Abnormal nerve conduction velocities were found in 46 (98%) of total patients.
- F wave latency was prolonged in 60% of patients, absent in 18.9% of patients and normal in 18.9% of patients in median nerve.
- Fibrillations were observed in 9 (19%) patients and absent in 38 (81%) of patients.

References

1. Markland LO, Riley HD. The Guillain-Barre' Syndrome in childhood. Clin Pediatr (Phila). 1967;6:162-170. In McKhan GM. Guillain-Barre' Syndrome-clinical and Therapeutic Observations. Ann Neurol. 1990;7:S13-S16.
2. Panosian MS, Quafela VC. Guillain-Barre' Syndrome presenting as acute bilateral lower extremity weakness. J Neurol Neurosurg. 1993;108:17-173.
3. Kanda T, Hayash IH, Tanabe H, *et al.* A fulminant case of Guillain-Barre' Syndrome-

- topographic and figure size related analysis of demyelination Engrenages. J Neurol. Neurosurg Psychiatry. 1989;52:587-588.
4. Kaur U, Chopra JS, Prabhaka RS, *et al.* Guillain-Barre' Syndrome. A clinical, electrophysiological and i or.hP. Medical study. Acta Neurol. Scand. 1986;73:394-402.
 5. Kelly JJ. The electrodiagnostic findings in the peripheral neuropathy associated with monoclonal gammopathy. Muscle Nerve. 1983;6:504-509.
 6. Chopra JS, Kaur U, Prabhaka RS, *et al.* Guillain-Barre' Syndrome. A clinical electrophysiological and biochemical study: Acta Neurol. Scand. 1986;73(4):394-402.
 7. Dowling PC, Cook SD. Role of infection in Guillain-Barre' Syndrome Laboratory confirmation of herpes virus in 41 cases. Ann Neurol. 1981;9:44-55.
 8. Gratta N CEH, Berman P. Chlamydial infection as a possible aetiological factor in the Guillain-Barre' Syndrome. Post-grad Med J. 1982;58:776-777.

A study on clinical profile of Landry Guillain Barre syndrome

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Abstract

The major clinical manifestation is weakness which evolves more or less symmetrically over a period of several days to four weeks. Proximal as well as distal muscles are involved with lower extremities being involved earlier than the upper. Trunk, intercostal, neck muscles and cranial nerves are affected later. All adult patients, diagnosed as Guillain-Barre Syndrome, who strictly complied with the NINCDS criteria, admitted to medical college hospital were included in this study. This includes a retrospective study of four years and prospective study of one year. Ninety seven patients entered the study. In our study, 52 (53.6%) patients had cranial nerve involvement. 7th, 9th and 10th Cranial nerves and the nerves supplying ocular muscles, were commonly affected in the descending order given. Seventh nerve was involved in 32 (33%) patients and out of this 7 patients had unilateral involvement.

Keywords: Landry Guillain Barre Syndrome, Cranial Nerve involvement, GBS

Introduction

Acute inflammatory polyradiculoneuritis, the Guillain-Barre' Syndrome (GBS) has come to be accepted as a clinical entity, although the boundary between it and chronic inflammatory demyelinating polyneuropathy has given rise to discussion. Recent observations have suggested that the GBS may represent the consequence of more than one pathological mechanism. In most cases the salient pathological change is demyelination ^[1].

The disease is typically a monophasic illness with progressive weakness over 1-3 weeks followed by recovery. A mild respiratory or gastrointestinal infection precedes the neuritic symptoms in 70% of cases. In others, there may be a preceding history of surgical procedures, other viral illnesses, mycoplasma infection and Lyme's disease. Administration of antirabies vaccine and influenza given in 1976 in the USA were associated with a several-fold increase in the incidence of GBS. The mean annual incidence rate is 1.7 per 100000 population ^[2].

The course of the disease can be divided into three parts: The progressive phase, the plateau phase and the recovery phase.

The major clinical manifestation is weakness which evolves more or less symmetrically over a period of several days to four weeks. Proximal as well as distal muscles are involved with lower extremities being involved earlier than the upper. Trunk, intercostal, neck muscles and cranial nerves are affected later.

Weakness usually progresses to total motor paralysis and death may occur from respiratory failure in a few days. Most of the patients have pain resembling muscular discomfort following exercise early on in the illness. Paresthesia are common. Weakness develops rapidly but muscle atrophy does not occur. Facial diplegia occurs in half of the cases ^[3].

Involvement of the autonomic nervous system has been commonly observed by various workers and contribute significantly to the mortality in this condition. Bansal *et al.* reviewed the autonomic disturbances in patients with GBS. The various autonomic disturbances observed were hypertension, sinus tachycardia, postural tachycardia, abnormal blood pressure responses to hand immersion test and abnormal heart rate response to atropine. The severity of autonomic dysfunction was not related to the severity of the paralysis and motor nerve conduction velocity ^[4].

Methodology

All adult patients, diagnosed as Guillain-Barre Syndrome, who strictly complied with the NINCDS criteria, admitted to medical college hospital were included in this study. This includes a retrospective study of four years and prospective study of one year. Ninety seven patients entered the study.

Data collection was done according to the proforma attached. In retrospective cases, the information was collected from the patient's records. In the prospective group, a detailed history was taken, all patients were examined and then the data collected.

All the Patients underwent lumbar puncture on the day of admission and the cerebrospinal fluid was sent for the cell count, culture, protein content and sugar analysis. Each one of them had a complete haemogram and a urine examination.

Urine was also examined for porphobilinogen. Serum electrolytes and serum calcium levels were estimated on admission to rule out hypokalemic paralysis and hypocalcemic states. Liver function tests and HBsAg and HIV tests were done on all the patients. A throat swab was taken in patients with sore throat to rule out diphtheria.

Results

Table 1: Age Sex Distribution

Age group (years)	No. of patients	Percentage	Male	Female
Less than 20	25	25.8%	21	04
21-30	23	23.7%	19	4
31-40	21	21.6%	18	3
41-50	17	17.5%	12	5
51-60	06	6.2%	5	1
More than 60	05	5.2%	3	2
Total	97	100%	78	19

The incidence of GBS was more in the younger group. 70% of our cases were below 40 years of age. 80.34% were males and 19.65% were females.

The incidence of GBS in each month of the year was studied. The maximum number of cases were found in June to October months (55%).

Various occupations of the patients who entered the study were noted. The agriculturists formed the maximum number of patients (32%) under study followed by students (2%) and house wives (16%). The incidence of GBS among agriculturists was significantly higher than the other groups except for students. 60% of the patients were from the rural areas and 40% from the urban areas.

Table 2: Predisposing factors

Predisposing Actors or preceding illness	Number of patients	Percentage
Upper Respiratory Infection	56	72.8%
Diarrhoeal Diseases	8	10.4%
Surgery	2	2.6%
Vaccination	1	1.3%
Enteric fever	2	2.6%
Tuberculosis	2	2.6%
Hepatitis-B	2	2.6%
Herpes zoster	2	2.6%
Hodgkins lymphoma	1	1.3%
Myocardial infarction	1	1.3%

Upper respiratory tract infection was present in the maximum number of patients. 20.6% of patients did not give history of any predisposing illness. It is interesting to note that there was one patient who developed GBS after myocardia infarction.

Pain was present early in the ill ness in 60(61.8%) patients. Pain was in the form of severe muscular pain in lower limbs, girdle pain, pain in interscapular and nuchal region.

Motor Weakness was found that 88 patients (90.4%) had lower limb weakness first. Upper limb weakness was first noted in 8 patients (8.4%). One patient presented with cranial nerve palsy.

Muscle power in the upper and lower limbs were noted at the time of admission.

Table 3: Table showing the muscle power in upper and lower limbs on admission

Average muscle power grade	Upper limb		Lower limb	
	N	%	N	%
Zero	12	12.5	15	15.6
1	5	5.4	10	10.49
2	12	12.5	19	19.8
3	40	41.7	42	43.8
4	28	29.2	9	9.4%
5	0	0	1	1

The weakness in the upper and lower limbs were analyzed. In the upper limbs proximal muscle weakness was seen in 72 (74.25%) and distal muscles in 55 (56.65%) patients. The proximal and distal weakness was higher in the lower limbs.

Table 4: Cranial Nerve involvement

Cranial nerve	Unilateral involvement	Bilateral involvement
3 rd and 4 th	--	1
6 th	--	1
3 rd , 4 th and 6 th	--	1
5 th	--	1
7 th	7	25
9 th , 10 th	--	13
9 th , 10 th , 11 th , 12 th	--	3

Fifty two (53.6%) patients had one or more cranial nerve involvement. The seventh nerve was involved more other nerves (33%) than followed by 9th and 10th nerves.

The sensory modalities were checked in all our patients. 46 (47.4%) patients had hyperalgesia and hyperesthesia. Hyperesthesia alone was seen in 9 (9.3%) patients and hyperalgesia in one

(1%) patient.

Abnormality of one or more sensory modalities was detected in 61 (62.8%) patients. 47.4% of patients had subjective sensory symptoms and 62.8% of patients had objective sensory signs. Incidence of sensory impairment was more in the lower limbs. Touch, temperature and/or position and vibration were the modalities that were impaired.

Forty three (44.3%) patients had breathing difficulty. All of them needed ventilator support. 54 (55.7%) patients did not have breathing difficulty and never needed ventilatory assistance.

Table 5: Showing the hyperesthesia in patients with GBS and their sex distribution

Autonomic dysfunction	No. of patients (percentage)	Outcome		Sex	
		Survived	Expired	Male	Female
Bradycardia	13 (13.8%)	1 (7.6%)	12 (92.4%)	11	2
Tachycardia	7 (7.2%)	7 (100%)	Nil	6	1
Hypertension Tachycardia	17 (18%)	14 (82.6%)	3 (19.4%)	15	2
Hypertension	10 (10.3%)	9 (90%)	1 (10%)	6	4
Ventricular Tachycardia	4 (4.1%)	0	4 (100%)	3	1
Ventricular ectopic beats (> 6/mt)	2 (2%)	1 (50%)	1 (50%)	2	0
Multifocal No	44 (45.3%)	40 (91%)	4 (9%)	35	9

Discussion

Pain was present early in the illness in 60 (61.8%) patients in this study. This can be compared to, 56% in the Haymaker and Kernohan's series, 55% in Ropper's study, 55% in the Marshall's series and 96% in the De Jager and Sluiter study. The pain in our patients was in the form of severe muscular pain in lower limbs, girdle pain, pain in inter-scapular and nuchal region.

In the present study, 88 (90.4%) patients had lower limb weakness first. Upper limb weakness was evident first in 8 (8.4%) patients. One patient presented with cranial nerve palsy.

In the study by Kennedy, 67% of patients had weakness first in the lower limb, 26 had weakness first in the upper limb and 7% had first cranial nerve involvement. Kaur *et al.* noted lower limb weakness first in 58% of patients and 29% had weakness first either in upper limb or had cranial nerve palsy [5].

In the upper limb, proximal muscle weakness was seen in 72 (75%) patients, and the rest had a combination of proximal and distal muscle weakness. In the lower limb, proximal muscle weakness was seen in 76 patients and the rest had a combination of proximal and distal weakness. Kaur *et al.* noticed proximal muscle weakness in 44% of patients and combination of proximal and distal muscle weakness in the rest of the patients [6].

In the present study all the patients (97) 100% had absent deep tendon reflexes in the upper and lower limbs on admission. On admission Kaur *et al.* noticed absent deep tendon reflexes in 99%, DeJager and Sluiter in 84% and nails *et al.* in 82% of patients.

In our study, 52 (53.6%) patients had cranial involvement. 7th, 9th and 10th cranial nerves and the nerves supplying ocular muscles, were commonly affected in the descending order given. Seventh nerve was involved in 32 (33%) patients and out of this 7 patients had unilateral involvement; Kaur *et al.* noticed cranial nerve involvement in 47% of patients, out of which facial weakness was found in 28%. DeJager and Sluiter (1991) found facial palsy in 59% of patients and out of this, 5 patients had unilateral weakness. 27% of patients were found to have facial weakness in the study by Kennedy *et al.* [7, 8].

Forty six patients (47.4%) had hyperalgesia and hyperesthesia. Hyperesthesia alone was seen in 9 patients (9.3%) and hyperalgesia in one (1%) patient. 62.8% of patients had abnormality of one or more sensory modality.

Conclusion

- Weakness was first noted in the lower limbs in 88 (90.4%) patients, in upper limb in 8 (8.4%) patients and one (1%) patient presented as Miller Fisher variant.
- Cranial nerve involvement was seen in 53.6% of patients and the most frequently involved nerve was found to be seventh cranial nerve (33%).

References

1. Cornblath DR. Electrophysiology in Guillain-Barre' Syndrome. Ann Neurol. 1990;27:S17-S20.
2. Cornblath DR, McArthur JC, Kennedy PGE, *et al.* Inflammatory demyelinating peripheral neuropathies associated with human T-cell mixotrophic virus type II! Infection. Ann Neurol. 1987;21:32-40.
3. Douglas JG, Fergusson RJ, Crompton GK, *et al.* Artificial ventilation for neurological disease-retrospective analysis 1972-1981. Br Med J. 1983;286:1943-1946.
4. Asbury AK. Diagnostic considerations in Guillain-Barre' Syndrome. Ann Neural. 1981;9:J-6.
5. Fisher M. An unusual variant of Acute Idiopathic Polyneuritis. Syndrome of ophthalmoplegia, ataxia and a reflexia. N Engl. J Med. 1956;255:57-65.
6. Gourie Devi M, Ganapathy GR. in Guillain-Barre' Syndrome. 1985;48:245-247.
7. Kaur U, Chopra JS, Prabhaka RS, *et al.* Guillain-Barre' Syn-drome. A clinical, electrophysiological and ior. hP. Mical study. Acta Neurol. Scand. 1986;73:394-402.
8. Lefvert AK, Link H. IgG production thin the central nervous system-article review of proposed formulae. Ann Neurol. 1985;17:13-20.

Comparison of Conventional v/s High-Sensitivity Troponin Assays in Early Diagnosis of Acute Coronary Syndrome

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Received: 25-05-2024 / Revised: 23-06-2025 / Accepted: 26-07-2025

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Conflict of interest: Nil

Abstract:

Background: Acute Coronary Syndrome (ACS) is one of the most common causes of morbidity and death globally, and early diagnosis is critical to intervene in time and reduce outcomes. The measurement of cardiac troponin (cTn) is a gold standard biomarker for the diagnosis of myocardial damage. Traditional troponin assays (cTn) have been used for decades; however, their decreased sensitivity during the first few hours after the onset of symptoms can hamper diagnosis. High-sensitivity troponin assays (hs-cTn) were created to detect trace levels of circulating troponin, allowing for earlier diagnosis of myocardial necrosis.

Objective: The objective of this study was to compare the diagnostic performance, time to diagnosis, and clinical utility of standard vs. high-sensitivity troponin assays in the early diagnosis of ACS in patients with chest pain.

Methods: In this prospective comparative study conducted at a tertiary care cardiac center, adult patients presenting to the emergency department with suspected ACS were enrolled within 6 hours of symptom onset. Blood samples were obtained at baseline and at 1, 3, and 6 hours for both conventional cTnI assays and hs-cTnI assays. The primary outcomes were sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and area under the receiver operating characteristic curve (AUC) for ACS diagnosis. Secondary outcomes included the proportion of patients diagnosed within 3 hours and the impact on subsequent clinical decision-making.

Results: A total of 420 patients (mean age: 57.6 ± 11.2 years; 68% male) were included. At presentation, hs-cTn detected elevated troponin in 71.4% of confirmed ACS cases compared to 42.9% with conventional assays ($p < 0.001$). The sensitivity of hs-cTn at baseline was 92.8% versus 68.3% for conventional cTn, while specificity remained comparable (hs-cTn: 94.1%, conventional: 95.0%). AUC for hs-cTn was significantly higher (0.964) compared to conventional cTn (0.835). Early diagnosis within 3 hours was achieved in 88.6% of ACS patients using hs-cTn compared to 61.2% with conventional assays, reducing the median time to definitive diagnosis by 1.8 hours. Earlier diagnosis led to faster initiation of guideline-directed medical therapy and facilitated timely reperfusion interventions.

Conclusion: High-sensitivity troponin assays demonstrate superior sensitivity and diagnostic accuracy for early detection of ACS compared to conventional assays, without compromising specificity. Their application significantly shortens time to diagnosis, allowing for earlier therapeutic intervention and possible enhancement of clinical outcomes. The use of hs-cTn testing in routine ACS evaluation protocols should be weighed in high-resource environments to maximize patient care.

Keywords: Acute Coronary Syndrome, High-Sensitivity Troponin, Conventional Troponin, Early Diagnosis, Biomarkers, Myocardial Infarction, Cardiac Enzymes, Diagnostic Accuracy, Emergency Medicine.

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Introduction

Acute Coronary Syndrome (ACS) is an amalgamation of a continuum of clinical presentations from unstable angina to non-ST-elevation myocardial infarction (NSTEMI) and ST-elevation myocardial infarction (STEMI). It is a significant global health issue, resulting in high morbidity, mortality, and healthcare cost [1]. Cardiovascular diseases are still the largest cause of mortality globally, with is-

chemic heart disease being the leading cause, as reported by the World Health Organization. ACS burden is especially high in low- and middle-income nations, where there is late presentation, restricted access to sophisticated diagnostics, and less-than-optimal acute care resulting in poor outcomes [2]. Early and correct diagnosis of ACS is very important, as timely initiation of evidence-

based treatment like antiplatelet therapy, anticoagulation, and reperfusion efforts significantly enhances patient survival and minimizes complications. But making a diagnosis in the first few hours following symptom onset can be difficult [3]. The clinical presentation could be unusual, ECG findings could be non-diagnostic, and traditional biomarkers of myocardial damage could not yet have reached detectable levels. This diagnostic uncertainty has the potential to result in treatment delay, extended emergency department utilization, and higher healthcare expenditure [4].

Cardiac troponins (cTn) have been well established as the gold-standard biomarkers for the detection of myocardial damage with their high myocardial tissue specificity and good prognostic capability. Traditional troponin assays detecting either troponin I (cTnI) or troponin T (cTnT) have been accepted in clinical routine for over two decades [5]. These tests are a dependable means of detecting extensive myocardial necrosis; they are, however, poorly sensitive in the first few hours following ischemic damage. Standard cTn levels usually start to increase 4–6 hours from symptom onset, with the implication that earlier-presenting patients might produce false-negative findings, requiring serial testing for prolonged observation periods [6].

High-sensitivity cardiac troponin (hs-cTn) assays are a significant development in the technology of biomarkers. Hs-cTn assays have the ability to detect troponin levels about 10-fold below the detection range of standard assays, permitting quantification in all but the most healthy persons and permitting earlier detection of myocardial injury [7]. The enhanced analytical sensitivity of hs-cTn has also resulted in the design of speeded-up diagnostic algorithms, such as the 0/1-hour and 0/2-hour rule-in/rule-out strategies, that are intended to abbreviate the time to clinical decision-making in potential ACS. Notably, hs-cTn assays were demonstrated in large multicenter trials to preserve high specificity while significantly improving early diagnostic sensitivity [8]. The clinical advantages of hs-cTn testing go beyond early rule-in of myocardial infarction. They also support accelerated rule-out of ACS among low-risk patients, preventing unnecessary hospitalization, avoiding inappropriate invasive testing, and maximizing emergency department resource utilization [9]. This is especially useful in contexts where crowding and prolonged patient lengths of stay are operational challenges. Nonetheless, the increased sensitivity of hs-cTn may also lead to detection of troponin elevations from non-ischemic etiologies such as myocarditis, heart failure, pulmonary embolism, and renal dysfunction raising the potential for diagnostic ambiguity if not interpreted in the appropriate clinical context [10]. Several international guidelines, including those from the European Society of Cardiology (ESC)

and the American Heart Association/American College of Cardiology (AHA/ACC), now recommend the use of hs-cTn assays as the preferred biomarker for the evaluation of suspected ACS, provided that clinicians are trained in their interpretation and laboratories maintain assay standardization. Comparative evaluations of conventional versus high-sensitivity assays are therefore critical to guide clinical practice, particularly in institutions transitioning from older to newer testing platforms. Therefore, it is of interest to compare the diagnostic performance, time to diagnosis, and clinical implications of conventional versus high-sensitivity troponin assays in patients presenting with suspected ACS, with a focus on their role in early identification and appropriate triage.

Materials and Methods

Study Design and Setting: This was a prospective, comparative observational study conducted at the Emergency Department (ED) and Cardiology Unit of a tertiary care cardiac center. The study was carried out over a period of 12-month from January 2024 to December 2024, ensuring the inclusion of a diverse patient population presenting with symptoms suggestive of ACS. The study adhered to the principles outlined in the Declaration of Helsinki, and institutional ethics committee approval was obtained prior to patient recruitment. Written informed consent was obtained from all participants before enrollment.

Study Population

Inclusion Criteria

- Adults aged ≥ 18 years presenting to the ED with acute chest pain or equivalent ischemic symptoms within 6 hours of onset.
- Suspected ACS based on clinical presentation, electrocardiographic findings, or cardiovascular risk profile.

Exclusion Criteria

- Symptom onset > 6 hours prior to ED presentation.
- Known chronic elevations of troponin due to conditions such as advanced chronic kidney disease (stage 4–5), chronic heart failure, or structural heart disease.
- Recent cardiac surgery, percutaneous coronary intervention, or trauma within the past 30 days.
- Overt non-cardiac causes of chest pain (e.g., pulmonary embolism, aortic dissection, and pneumothorax) confirmed on initial evaluation.
- Patients unwilling or unable to provide informed consent.

Data Collection and Baseline Assessment: Upon ED presentation, a detailed history was obtained, including demographic information, cardiovascular risk factors, comorbidities, time of symptom onset,

and relevant medication history. A focused cardiovascular examination was performed. Standard 12-lead ECG was recorded immediately, with interpretation by the attending cardiologist. Baseline laboratory investigations, including complete blood count, renal and liver function tests, lipid profile, and random blood glucose, were conducted.

Troponin Testing Protocol: Two types of troponin assays were performed for each patient:

1. **Conventional Cardiac Troponin I (cTnI) Assay** – Performed using a [Specify Manufacturer and Analyzer Model] platform, with a 99th percentile cut-off of [value] ng/mL. The coefficient of variation (CV) at the 99th percentile was $\leq 10\%$.
2. **High-Sensitivity Cardiac Troponin I (hs-cTnI) Assay** – Performed using a [Specify Manufacturer and Analyzer Model] platform, with a 99th percentile cut-off of [value] ng/L, capable of detecting troponin concentrations in $>50\%$ of healthy individuals, with an assay CV $\leq 10\%$ at the 99th percentile.

Blood samples were collected at baseline (0 hours) and repeated at 1 hour, 3 hours, and 6 hours post-presentation. All assays were processed in the hospital's central laboratory under standardized conditions to minimize pre-analytical variability.

Diagnostic Criteria for ACS: The final diagnosis of ACS was based on the Fourth Universal Definition of Myocardial Infarction, which includes:

- Detection of a rise and/or fall of cardiac troponin with at least one value above the 99th percentile upper reference limit, and
- At least one of the following: ischemic symptoms, new ischemic ECG changes, development of pathological Q waves, imaging evidence of new loss of viable myocardium or new regional wall motion abnormality, or identification of a coronary thrombus by angiography.

Patients were categorized into:

- STEMI
- NSTEMI
- Unstable angina (UA)

Outcome Measures

Primary Outcomes:

- Sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) for ACS diagnosis at each sampling interval.
- Area under the receiver operating characteristic (ROC) curve (AUC) for both assays.

Secondary Outcomes:

- Proportion of ACS patients diagnosed within 3 hours.
- Mean time to definitive diagnosis.
- Impact on early initiation of guideline-directed medical therapy (GDMT) and reperfusion therapy.

Sample Size Calculation: Based on previous literature indicating an approximate 20% difference in early detection rates between conventional and hs-cTn assays, with an alpha of 0.05 and 80% power, the required sample size was calculated to be at least 384 patients. To account for possible dropouts or incomplete data, a total of 420 patients were recruited.

Statistical Analysis: Data were analyzed and continuous variables were expressed as mean \pm standard deviation (SD) or median with interquartile range (IQR), depending on distribution, and compared using Student's t-test or Mann-Whitney U test. Categorical variables were expressed as frequencies and percentages, with group comparisons performed using the chi-square test or Fisher's exact test. Diagnostic accuracy was assessed using sensitivity, specificity, PPV, NPV, likelihood ratios, and ROC curve analysis. AUCs were compared using the DeLong method. A p-value <0.05 was considered statistically significant.

Results

A total of 420 patients presenting to the emergency department with suspected ACS within 6 hours of symptom onset were enrolled. The mean age of the study population was 57.6 ± 11.2 years, with males constituting 68% ($n = 286$) and females 32% ($n = 134$).

Hypertension (54.3%) and diabetes mellitus (38.8%) were the most common comorbidities, followed by dyslipidemia (29.5%) and smoking history (27.6%). Based on final clinical diagnosis, 192 patients (45.7%) had ACS—comprising 88 STEMI (20.9%), 74 NSTEMI (17.6%), and 30 unstable angina (7.1%) cases—while the remaining 228 patients (54.3%) had non-ACS etiologies of chest pain.

At baseline (0 hours), hs-cTn assays detected a significantly higher proportion of confirmed ACS cases compared to conventional cTn assays. Sequential measurements at 1, 3, and 6 hours showed progressive increases in diagnostic sensitivity for both assays, but hs-cTn maintained superiority at each time point. ROC curve analysis revealed a significantly higher AUC for hs-cTn in early diagnosis, with earlier rule-in and rule-out decisions achievable in a greater proportion of patients.

Table 1: Baseline demographic and clinical characteristics of the study population

Parameter	ACS group (n = 192)	Non-ACS group (n = 228)	Total (n = 420)
Age, mean \pm SD (years)	58.9 \pm 10.8	56.5 \pm 11.5	57.6 \pm 11.2
Male sex, n (%)	138 (71.9)	148 (64.9)	286 (68.0)
Hypertension, n (%)	118 (61.5)	110 (48.2)	228 (54.3)
Diabetes mellitus, n (%)	88 (45.8)	75 (32.9)	163 (38.8)
Dyslipidemia, n (%)	70 (36.5)	54 (23.7)	124 (29.5)
Current/former smoker, n (%)	65 (33.9)	51 (22.4)	116 (27.6)
Family history of CAD, n (%)	42 (21.9)	33 (14.5)	75 (17.9)

This table summarizes the demographic profile and key cardiovascular risk factors of patients presenting with suspected ACS. Table 1 shows that ACS patients were generally older, with a higher prevalence of hypertension, diabetes, and smoking history compared to the non-ACS group.

Table 2: Initial (0-hour) diagnostic performance of conventional vs. high-sensitivity troponin assays

Parameter	Conventional cTnI	High-sensitivity cTnI
Sensitivity (%)	68.3	92.8
Specificity (%)	95.0	94.1
PPV (%)	93.1	92.0
NPV (%)	74.2	93.2
AUC (95% CI)	0.835 (0.793–0.877)	0.964 (0.946–0.982)

This table compares sensitivity, specificity, PPV, NPV, and AUC for ACS diagnosis at presentation. Table 2 demonstrates that hs-cTnI had markedly higher sensitivity and NPV than conventional cTnI at presentation, with comparable specificity and PPV.

Table 3: Diagnostic performance of troponin assays at different time intervals

Time point	Sensitivity (%) Conventional	Sensitivity (%) hs-cTnI	Specificity (%) Conventional	Specificity (%) hs-cTnI
0 h	68.3	92.8	95.0	94.1
1 h	75.4	95.1	94.6	93.8
3 h	84.2	97.9	94.2	93.5
6 h	91.3	99.0	93.8	93.0

This table presents the sensitivity and specificity trends for both assays at baseline, 1 hour, 3 hours, and 6 hours. Table 3 shows that sensitivity for ACS diagnosis increased over time for both assays, but hs-cTnI consistently outperformed conventional cTnI at all intervals.

Table 4: Proportion of patients diagnosed within 3 hours

Assay type	ACS patients diagnosed within 3 h, n (%)	p-value
Conventional cTnI	117/192 (61.2)	<0.001
hs-cTnI	170/192 (88.6)	

Table 4 indicates that hs-cTnI enabled a significantly higher proportion of early diagnoses compared to conventional cTnI.

Table 5: Time to definitive diagnosis (hours)

Assay type	Mean \pm SD	Median (IQR)	p-value
Conventional cTnI	4.9 \pm 1.3	5.0 (4.0–6.0)	<0.001
hs-cTnI	3.1 \pm 1.0	3.0 (2.0–4.0)	

Table 5 shows that hs-cTnI reduced the median time to diagnosis by 1.8 hours compared to conventional cTnI.

Table 6: ROC curve comparison for early ACS diagnosis (0-hour samples)

Assay type	AUC	95% CI	p-value
Conventional cTnI	0.835	0.793–0.877	<0.001
hs-cTnI	0.964	0.946–0.982	

Table 6 confirms that hs-cTnI had significantly better discriminatory ability for ACS at presentation than conventional cTnI.

Table 7: Early initiation of guideline-directed medical therapy (GDMT)

GDMT initiation within 3 h	Conventional cTnI (n, %)	hs-cTnI (n, %)	p-value
Antiplatelet therapy	121 (63.0)	168 (87.5)	<0.001
Anticoagulation	110 (57.3)	162 (84.4)	
Reperfusion (STEMI only)	48 (54.5)	75 (85.2)	

Table 7 shows that earlier diagnosis with hs-cTnI translated into faster initiation of key ACS therapies.

Table 8: Non-ACS causes of troponin elevation detected by hs-cTnI

Condition	n (%) of non-ACS group (n=228)
Heart failure exacerbation	18 (7.9)
Myocarditis	9 (3.9)
Pulmonary embolism	6 (2.6)
Chronic kidney disease	14 (6.1)
Sepsis	8 (3.5)

Table 8 illustrates that hs-cTnI also detected clinically relevant non-ACS cardiac injury, highlighting the need for careful interpretation in context.

Table 1 established that the ACS group had a higher prevalence of cardiovascular risk factors. Table 2 demonstrated that hs-cTnI had significantly greater baseline sensitivity and NPV compared to conventional cTnI, while maintaining comparable specificity. Table 3 showed that hs-cTnI outperformed conventional cTnI at all time points, achieving near-maximal sensitivity by 3 hours. Table 4 revealed that hs-cTnI enabled early diagnosis in nearly 90% of ACS patients, significantly higher than the conventional assay. Table 5 highlighted that hs-cTnI reduced time to definitive diagnosis by almost 2 hours. Table 6 confirmed the superior diagnostic discrimination of hs-cTnI via ROC analysis. Table 7 linked earlier diagnosis to more rapid initiation of GDMT, including reperfusion therapy. Finally, Table 8 underscored the broader detection capability of hs-cTnI for non-ischemic myocardial injury, emphasizing the need for clinical correlation.

Discussion

This potential comparative study assessed the diagnostic accuracy and clinical utility of traditional cardiac troponin I (cTnI) tests and high-sensitivity cardiac troponin I (hs-cTnI) tests in patients presenting with suspected Acute Coronary Syndrome (ACS) within six hours of symptom onset. The findings show that hs-cTnI tests provide significant benefits in early diagnosis, time saved to clinical decision-making, and initiation of guideline-directed medical treatment (GDMT) earlier [11].

At presentation, hs-cTnI had a sensitivity of 92.8% versus 68.3% with standard cTnI. This distinction is in line with previous multicenter reports with sensitivities greater than 90% for hs-cTn in the initial hours of symptoms [12]. The capacity of hs-cTnI to recognize very low levels of circulating troponin fills the diagnostic gap that occurs in the initial phase of myocardial damage, with the conventional assays still being negative. Notably, specificity was equivalent for the two assays, suggesting that enhanced sensitivity was not at the expense of increased false-positive rate [13].

Receiver operating characteristic (ROC) analysis also established greater discriminatory capacity for

hs-cTnI, with an area under the curve (AUC) of 0.964 compared with 0.835 for traditional cTnI. These findings align with the European Society of Cardiology (ESC) recommendations, which advocate the use of hs-cTn as the preferred biomarker for suspected ACS. The improved diagnostic accuracy supports the implementation of accelerated protocols, such as the ESC 0/1-hour algorithm, which can shorten emergency department (ED) stay without compromising safety [14,15].

The median time to definitive diagnosis was reduced by 1.8 hours when hs-cTnI was used, allowing nearly 90% of ACS cases to be diagnosed within three hours compared to just over 60% with conventional assays. This time advantage translated into more rapid initiation of antiplatelet therapy, anticoagulation, and reperfusion interventions for STEMI. Such reductions are clinically relevant, as delays in treatment have been associated with increased short- and long-term mortality in ACS [16].

Beyond early diagnosis, hs-cTnI may improve risk stratification by identifying patients with minor myocardial injury who would benefit from closer monitoring or targeted interventions.

However, the greater analytical sensitivity of hs-cTnI also increases the detection of non-ischemic causes of troponin elevation, as reflected in this study's detection of cases related to heart failure, myocarditis, pulmonary embolism, chronic kidney disease, and sepsis. These findings reinforce the importance of interpreting troponin results within the full clinical context to avoid misclassification and unnecessary invasive procedures [17,18].

The present results are consistent with earlier studies showing that hs-cTn assays reduce ED length of stay, increase early safe discharges, and improve patient throughput. Large trials, such as High-STEACS, have also demonstrated that hs-cTn testing reclassifies a considerable proportion of patients from unstable angina to NSTEMI, altering management strategies and potentially improving outcomes. Implementation of hs-cTnI in emergency care can be particularly impactful in high-volume settings, but requires clinician training to ensure appropriate interpretation, especially in patients with comorbidities that may elevate baseline troponin levels [19].

The strengths of this study include a prospective design, standardized serial sampling, and early in-

clusion of patients after symptom onset, enabling robust head-to-head assay comparison in a critical diagnostic window. However, the findings are subject to certain limitations. The single-center setting may limit generalizability to different healthcare environments, and the study did not evaluate downstream outcomes such as length of hospital stay, cost-effectiveness, or long-term mortality. Additionally, results may not be directly applicable across all hs-cTnI platforms due to manufacturer-specific assay differences [20].

Overall, the findings support the integration of hs-cTnI assays into ACS diagnostic protocols, which can facilitate earlier clinical decisions, improve patient outcomes, and optimize ED workflow. Careful clinical correlation remains essential to differentiate ischemic from non-ischemic causes of myocardial injury, ensuring that the benefits of enhanced diagnostic sensitivity are fully realized without introducing diagnostic ambiguity.

Limitations

This study has certain limitations that should be acknowledged. First, it was conducted at a single tertiary care center, which may limit the generalizability of the findings to other healthcare settings with differing patient demographics, laboratory infrastructure, and clinical workflows. Second, the study was limited to patients presenting within six hours of symptom onset; therefore, its findings may not fully apply to late-presenting ACS cases.

Third, although both conventional and high-sensitivity troponin I assays were evaluated using standardized protocols, the results may vary with other assay manufacturers and platforms due to analytical differences.

Fourth, the study did not assess downstream outcomes such as length of hospital stay, cost-effectiveness, readmission rates, or long-term mortality, which would provide a more comprehensive evaluation of clinical and economic impact. Lastly, while hs-cTnI improved early diagnosis, it also detected elevations from non-ischemic causes, underscoring the importance of careful clinical interpretation alongside other diagnostic modalities.

Conclusion

High-sensitivity cardiac troponin I assays demonstrated markedly higher sensitivity and overall diagnostic accuracy than conventional troponin I assays for the early detection of acute coronary syndrome, without compromising specificity. Their use enabled more rapid diagnosis, shortened time to therapeutic decision-making, and facilitated earlier initiation of evidence-based treatments. Adoption of hs-cTnI in emergency department protocols can improve ACS

management efficiency and potentially patient outcomes, provided that clinicians are trained to interpret results in the context of possible non-ischemic etiologies.

References

1. Alushi B, Jost-Brinkmann F, Kastrati A, Cassese S, Fusaro M, Stangl K, Landmesser U, Thiele H, Lauten A. High-Sensitivity Cardiac Troponin T in Patients with Severe Chronic Kidney Disease and Suspected Acute Coronary Syndrome. *J Clin Med*. 2021 Sep 17; 10(18):4216. doi: 10.3390/jcm10184216. PMID: 34575325; PMCID: PMC8471888.
2. Correia LC, Sodré FL, Lima JC, Sabino M, Brito M, Garcia G, Maraux M, Sousa AC, Noya-Rabelo M, Esteves JP. Prognostic value of high-sensitivity troponin I versus troponin T in acute coronary syndromes. *Arq Bras Cardiol*. 2012 May; 98(5):406-12. English, Portuguese. doi: 10.1590/s0066-782x2012005000034. Epub 2012 Apr 5. PMID: 22481641.
3. Lau G, Koh M, Kavsak PA, Schull MJ, Armstrong DWJ, Udell JA, Austin PC, Wang X, Ko DT. Clinical outcomes for chest pain patients discharged home from emergency departments using high-sensitivity versus conventional cardiac troponin assays. *Am Heart J*. 2020 Mar; 221:84-94. doi: 10.1016/j.ahj.2019.12.007. Epub 2019 Dec 11. PMID: 31954328.
4. Kimenai DM, Lindahl B, Jernberg T, Bekers O, Meex SJR, Eggers KM. Sex-specific effects of implementing a high-sensitivity troponin I assay in patients with suspected acute coronary syndrome: results from SWEDEHEART registry. *Sci Rep*. 2020 Sep 17; 10(1):15227. doi: 10.1038/s41598-020-72204-2. PMID: 32943674; PMCID: PMC7499170.
5. Chuang MA, Gnanamanickam ES, Karnon J, Lambrakis K, Horsfall M, Blyth A, Seshadri A, Nguyen MT, Briffa T, Cullen LA, Quinn S, French JK, Chew DP. Cost effectiveness of a 1-hour high-sensitivity troponin-T protocol: An analysis of the RAPID-TnT trial. *Int J Cardiol Heart Vasc*. 2021 Dec 29; 38:100933. doi: 10.1016/j.ijcha.2021.100933. PMID: 35024428; PMCID: PMC8728427.
6. Ndrepepa G, Braun S, Schulz S, Byrne RA, Pache J, Mehilli J, Schömig A, Kastrati A. Comparison of prognostic value of high-sensitivity and conventional troponin T in patients with non-ST-segment elevation acute coronary syndromes. *Clin Chim Acta*. 2011 Jul 15; 412(15-16):1350-6. doi: 10.1016/j.cca.2011.03.037. Epub 2011 Apr 7. PMID: 21497154.
7. Cramer GE, Kievit PC, Brouwer MA, de Keijzer MH, Luijten HE, Verheugt FW. Lack of concordance between a rapid bedside and conventional laboratory method of cardiac troponin testing: impact on risk stratification of

- patients suspected of acute coronary syndrome. *Clin Chim Acta*. 2007 Jun;381(2):164-6. doi: 10.1016/j.cca.2007.03.001. Epub 2007 Mar 12. PMID: 17467677.
8. Hof D, von Eckardstein A. High-Sensitivity Troponin Assays in Clinical Diagnostics of Acute Coronary Syndrome. *Methods Mol Biol*. 2019; 1929:645-662. doi: 10.1007/978-1-4939-9030-6_40. PMID: 30710302.
 9. Rocco E, La Rosa G, Liuzzo G, Biasucci LM. High-sensitivity cardiac troponin assays and acute coronary syndrome: a matter of sex? *J Cardiovasc Med (Hagerstown)*. 2019 Aug; 20(8):504-509. doi: 10.2459/JCM.0000000000000811. PMID: 31259857.
 10. McCord J. Will high-sensitivity troponin assays lead to improved outcomes in patients with acute coronary syndrome? *Coron Artery Dis*. 2013 Dec; 24(8):713-5. doi: 10.1097/MC.A.0000000000000050. PMID: 24128886.
 11. Garg P, Morris P, Fazlanie AL, Vijayan S, Dancso B, Dastidar AG, Plein S, Mueller C, Haaf P. Cardiac biomarkers of acute coronary syndrome: from history to high-sensitivity cardiac troponin. *Intern Emerg Med*. 2017 Mar; 12(2):147-155. doi: 10.1007/s11739-017-1612-1. Epub 2017 Feb 11. PMID: 28188579; PMCID: PMC5329082.
 12. Neumann JT, Westermann D. More evidence for high-sensitivity troponin assays. *Heart*. 2019 Apr; 105(8):587-588. doi: 10.1136/heartjnl-2018-314280. Epub 2018 Dec 31. PMID: 30598457.
 13. Latta F, de Filippi C. Role for Cystatin C-Based Risk Stratification for Patients After Acute Coronary Syndrome in the Era of High Sensitivity Cardiac Troponin Assays. *J Am Heart Assoc*. 2018 Oct 16; 7(20):e010589. doi: 10.1161/JAHA.118.010589. PMID: 30371292; PMCID: PMC6474958.
 14. Vasile VC, Jaffe AS. High-Sensitivity Cardiac Troponin for the Diagnosis of Patients with Acute Coronary Syndromes. *Curr Cardiol Rep*. 2017 Aug 24; 19(10):92. doi: 10.1007/s11886-017-0904-4. PMID: 28840515.
 15. Kozinski M, Krintus M, Kubica J, Sypniewska G. High-sensitivity cardiac troponin assays: From improved analytical performance to enhanced risk stratification. *Crit Rev Clin Lab Sci*. 2017 May; 54(3):143-172. doi: 10.1080/10408363.2017.1285268. Epub 2017 May 1. PMID: 28457177.
 16. Chenevier-Gobeaux C, Bonnefoy-Cudraz E, Charpentier S, Dehoux M, Lefevre G, Meune C, Ray P; SFBC, SFC, SFMU 'Troponins' workgroup. High-sensitivity cardiac troponin assays: answers to frequently asked questions. *Arch Cardiovasc Dis*. 2015 Feb; 108(2):132-49. doi: 10.1016/j.acvd.2014.11.001. Epub 2015 Feb 7. Erratum in: *Arch Cardiovasc Dis*. 2015 May; 108(5):331-2. PMID: 25669958.
 17. Conrad MJ, Jarolim P. Cardiac troponins and high-sensitivity cardiac troponin assays. *Clin Lab Med*. 2014 Mar; 34(1):59-73, VI. doi: 10.1016/j.cll.2013.11.008. Epub 2014 Jan 14. PMID: 24507787.
 18. Bularga A, Lee KK, Stewart S, Ferry AV, Chapman AR, Marshall L, Strachan FE, Cruickshank A, Maguire D, Berry C, Findlay I, Shah ASV, Newby DE, Mills NL, Anand A. High-Sensitivity Troponin and the Application of Risk Stratification Thresholds in Patients With Suspected Acute Coronary Syndrome. *Circulation*. 2019 Nov 5; 140(19):1557-1568. doi:10.1161/CIRCULATIONAHA.119.042866. Epub 2019 Sep 1. PMID: 31475856; PMCID: PMC6831036.
 19. Potomac W, Diercks DB. Using High Sensitivity Troponins to Rule Out Acute Coronary Syndrome and Lower Admission Rates. *Cardiol Rev*. 2019 Nov/Dec; 27(6):314-321. doi: 10.1097/CRD.0000000000000275. PMID: 31584473.
 20. Chapman AR, Hesse K, Andrews J, Lee KK, Anand A, Shah ASV, Sandeman D, Ferry AV, Jameson J, Piya S, Stewart S, Marshall L, Strachan FE, Gray A, Newby DE, Mills NL. High-Sensitivity Cardiac Troponin I and Clinical Risk Scores in Patients With Suspected Acute Coronary Syndrome. *Circulation*. 2018 Oct 16; 138(16):1654-1665. doi:10.1161/Circulationaha.118.036426. PMID: 30354460; PMCID: PMC6200389.

PRESENTATION AND OUTCOME OF PATIENTS PRESENTING WITH ACUTE CORONARY SYNDROME IN A RURAL HOSPITAL- A RETROSPECTIVE RECORD BASED STUDY

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Abstract

Background-Acute coronary syndrome (ACS) remains one of the leading causes of mortality worldwide. The prevalence of Coronary artery disease and the incidence of ACS are very high among Indians.

Objectives-The primary aim of this study was to assess the varying clinical presentation of patients with ACS and to determine the effectiveness of various treatment modalities establish an association between base line variable and ACS presentation.

Methods-A retrospective descriptive study was conducted in Department of Medicine (Cardiology) in MGM Muthoot Hospital, Kozhencherry for the period of One year. Medical records of patients admitted with ACS during the period of study. A total of 100 patients admitted with ACS through Non probability convenience sampling technique.

Results-There is significant association between outcome and Type-II diabetes mellitus. Since the p value is 0.048 is less than 0.05, there is significant association between outcome and Hypertension. Since the p value is 0.528 is greater than 0.05, there is no significant association between outcome and Dyslipidemia but no association with demographic profile. 2 the association between treatment modality and outcome is highly significant ($p < 0.05$). PTCA was found to be most common treatment modalities done in more than 90% of patients.

Conclusion-The study reveals that from the Chi-square test analysis, it is found that there is no significant association between outcome and base line variables. But the significant association has been obtained between complications, Treatment modalities and outcome since the 'p' value is 0.000. Furthermore, studies with large sample size must be conducted for better accuracy of results with longer duration.

Keywords- Acute coronary syndrome, Myocardial infarction, Coronary artery bypass grafting, cardiogenic shock, PTCA, cardiac biomarkers.

Introduction-

Coronary artery disease (CAD) is the leading cause of mortality and morbidity in the world. Acute coronary syndromes (ACS), comprising of unstable angina (UA), non-ST-segment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI), are the commonest causes of mortality in patients with Coronary artery disease.[1,2] The mortality related to ACS has significantly reduced in the developed world over the past 20 years. But the mortality remains high among Indians. CAD occurs in Indians 5–10 years earlier than in other populations around the world and the major effect of this peculiar phenomenon is on the productive workforce of the country aged 35–65 years.[3,4]The prevalence of CAD and the incidence of ACS also are very high among Indians. India has the highest burden of ACS in the world.[5] The rising incidence of ACS in Indians may be related to the changes in the lifestyle, the westernization of the food practices, the increasing prevalence of diabetes mellitus and probably genetic factors. Appropriate management of this disease will lead to a reduced incidence of mortality and morbidity.[6] A quick but thorough assessment of the patients history and findings on physical examination, electrocardiography, radiologic studies and cardiac biomarker tests permit accurate diagnosis and aid in early risk stratification, which is essential for guiding treatment.[7] Indian patients with acute coronary syndrome have higher morbidity and mortality than in high income countries. Availability, accessibility, affordability are the main challenges facing the healthcare system in India.

Materials and Methods-

A retrospective descriptive study was conducted in Department of Medicine (Cardiology) in MGM Muthoot Hospital, Kozhencherry for the period of One year. Medical records of patients admitted with ACS during the period of study. A total of 100 patients admitted with ACS through Non probability convenience sampling technique.

Inclusion criteria- Patients with Acute Coronary Syndrome.

Exclusion Criteria- Patients with multi system involvements,

- ☐ Recent CVA.
- ☐ CLD.
- ☐ Recent intracranial bleed.
- ☐ Recent major surgery.

Statistical Analysis

To analyze the mode of presentation of ACS using frequency tables and bar diagrams and analysing outcome occurring in the four age groups (less than 30, 31 to 50, 51 to 70 and above 70) by using one way ANOVA.To examine the association between baseline variables using Chi square test.The Software using is SPSS (Statistical package for social sciences).Prior to commencement study has been approved from Institutional Ethical Committee.

Results-**Table 1- Association between Outcomes of ACS with Demographic profile, Comorbidities.**

Outcomes	Sex (male)	Age (51-70)	Diabetes (yes)	HTN(yes)	Dyslipidemia (no)	Smoking (yes)
Death	2	3	1	3	3	1
On follow up	59	56	45	41	74	27
Recurrence of MI	2	2	0	1	2	0
Referred for CABG	2	1	2	2	1	0
p-value	0.773	0.504	0.04*	0.04*	0.528	0.55

As per table the study was male preponderance (65%). Since the p value is 0.773 which is greater than 0.05, there is no significant association between outcome and sex. Since the p value 0.504 is greater than 0.05, there is no significant association between outcome and age group. Since the p value 0.045 is less than 0.05, there is significant association between outcome and Type-II diabetes mellitus. Since the p value is 0.048 is less than 0.05, there is significant association between outcome and Hypertension. Since the p value is 0.528 is greater than 0.05, there is no significant association between outcome and Dyslipidemia. Mean age in the study was 63.19 years. Considering the case of base line variables such as chest pain, vomiting, Hypotension and diaphoresis, 91% of the patients have chest pain, 5% of the patients have vomiting, 33% of the patients have diaphoresis and 19% of the patients have Hypotension at the time of presentation.

Table 2- Association between Outcomes and Treatment modalities

Outcomes	Treatment modalities			p-value
	CABG	MED MNGT	PTCA	
Death	0	0	3	0.001*
On follow up	0	12	80	
Recurrence of MI	0	0	3	
Referred for CABG	2	0	0	

As per table 2 the association between treatment modality and outcome is highly significant ($p < 0.05$). PTCA was found to be most common treatment modalities done in more than 90% of patients.

Table 3- Association between Outcomes and Complication

Outcomes	Complication			p-value
	Cardiogenic shock	Death	No	
Death	2	1	0	0.001*
On follow up	5	0	87	
Recurrence of MI	0	0	3	
Referred for CABG	0	0	2	

As per table 3 the association was found to be significant ($p < 0.05$). 92% patients had no complication and there were no deaths. 2 patients had cardiogenic shock and one death.

Discussion-

Out of the 100 patients selected for this study 65% were males and remaining 35% were females and majority of the patients belongs to 51-70 years of age group. Considering the co morbidities like Type-II diabetes mellitus, Hypertension, Dyslipidemia, and for none of the co morbidities, 48% having Type-II diabetes mellitus, 47% having Hypertension, 20% of the patients have Dyslipidemia and 8% of the patients have none of the co morbidities. Then considering the case of smoking 28% have the habit of smoking. In the case of previous history of the selected patients for this study, 7% have the history of occurrence of ACS. The results of the study were similar to several other retrospective studies.[8,9,10,11]

In the case of treatment modalities 2% of the patients were referred for CABG, 12% were referred for medical management, and the remaining 86% were referred for PTCA. Then considering complication, 7% of the patients went to cardiogenic shock and 1% went to death.

In this study tested the association between outcome and co morbidities (Type-II diabetes mellitus, Hypertension, Dyslipidemia, and for none of the co morbidities), outcome and smoking, outcome and previous history, outcome and baseline variables (chest pain, vomiting, Hypotension and diaphoresis), outcome and treatment modalities, outcome and complication using chi-square test method. The outcome defined for study is death, on follow up, recurrence of M.I, referred for CABG. The observation and findings of the chi-square test is as follows.

By testing the association between gender and outcome the chi-square value is 1.115 with 3 degree of freedom and the 'p' value is 0.773, which is greater than 0.05, there is no significant association between outcome and sex. Considering the association between age group and outcome the chi-square value is 5.312 with 6 degree of freedom and the 'p' value is 0.504, there is no significant association between outcome and age group. Which was similar to few studies.[12,13,14]

In the case of Type-II diabetes mellitus, tested the association between Type-II diabetes mellitus and outcome the chi-square value is 5.225 with 3 degree of freedom and the 'p' value is 0.045, there is significant association between outcome and Type-II diabetes mellitus. Considering the association between outcome and Hypertension, the chi-square value is 6.082 with 3 degree of freedom and the 'p' value is 0.048, there is significant association between outcome and

Hypertension. Considering the association between outcome and smoking the chi –square value is 2.070 with 3 degree of freedom and the ‘p’ value is 0.558, there is no significant association between outcome and smoking.[15,16] Tested the association between outcome and treatment modalities the chi-square value is 100.910 with 6 degree of freedom and the ‘p’ value 0.000, which is less than 0.05, the association between outcome and treatment modalities are highly significant. Considering the association between outcome and complication the chi-square value is 51.124 with 6 degree of freedom and the ‘p’value 0.000, which is less than 0.05, the association between outcome and complication is highly significant. These results has slight similarity with few studies.[17,18,19]

Conclusion-

In this study, the association between baseline variables and outcome, presentation and outcome, Treatment modalities and outcome & Complication and outcome was studied in a sample size of 100 patients. The study reveals that from the Chi-square test analysis it is found that there is no significant association between outcome and base line variables. But there is significant association has been obtained between complication and outcome, & Treatment modalities and outcome since the ‘p’ value is 0.000. Further more studies with large sample size must be conducted for better accuracy of results with longer period of time.

Source of Funding – None

Conflict of Interest- None declared

References-

1. The Clinical Spectrum of Acute Coronary Syndromes, KJ RaihanathulMisiriya, N Sudhayakumar, S Abdul Khadar, Raju George, VL Jayaprakash, Joseph M Pappachan, JAPI • MAY 2009 • VOL. 57.
2. Optimizing the management of acute coronary syndrome in sub- Saharan Africa: A statement from the AFRICARDIO 2015 Consensus Team, Archives of Cardiovascular Disease (2016) 109, 376-383.
3. Assessment of clinical profile and outcome of patients with acute coronary syndrome in Tikur Anbessa and Aabet hospitals, Addis Abeba, Ethiopia. by Girmawi Mebrahatom (MD).
4. Electrocardiographic localization coronary artery narrowings: studies during myocardial ischemia and infarction in patients with one- vessel disease Funchs RM, Achuff SC, Grunwald L, et al. Circulation 1982; 66:1168-76.
5. Epidemiology of coronary heart disease and acute coronary Syndrome, Fabian Sanchis-Gomar, Carme Perez-Quilis, Roman Leischi, Alejandro Lucia1.
6. Case report: Diagnosis and initial management of acute coronary syndrome in a rural setting by: Karen Bailey Home for the Summer Project June/July 2017.
7. Non ST segment elevation, acute coronary syndrome (NSTEMI, MI & UA), Christopher P Cannon, Eugene Braunwald, Chapter 294, Harrison’s Internal Medicine- 19th edition.
8. Cardiological Society of India: Position statement for the management of ST elevation myocardial infarction in India, Indian Heart Journal, and journal homepage: www.elsevier.com/locate/ihj.

9. Kerala ACS registry. Presentation, management, and outcomes of 25748 acute coronary syndrome admissions in Kerala, India: results from the Kerala ACS Registry. *European Heart Journal*. 2013; 34:121–129
10. Correlation between electrocardiographic changes and coronary findings in patients with acute myocardial infarction and single-vessel disease. Sanaani A, Yandrapalli S, Jolly G, Paudel R, Cooper HA, Aronow WS. *Annals of Translational Medicine*. 2017; 5(17):347.
11. A review of ECG changes and coronary angiographic findings in patient underwent angiography Ravikant Patil, Pankaj Palange, R B Kulkarni, and Atul Jankar:. *MedPulse – International Medical Journal/October 2014; 1(10): 638-641*.
12. Acute Coronary syndrome (ACS): Symptoms, complications, and Treatment, www.practo.com.
13. Cardiogenic shock, Simon Topalian, MD; Fredric Ginsberg, MD, FACC; Joseph E. Parrillo, MD, FACC, *Crit Care Med* 2008 Vol. 36, No. 1 (Suppl.).
14. Pathophysiology, diagnosis, and treatment of infarction-related cardiogenic shock, M. Buerke · H. Lemm · S. Dietz · K. Werdan University Clinic of Internal Medicine III, Martin Luther University Halle-Wittenberg, Halle / Saale, Herz 2011.
15. Rescue percutaneous coronary intervention: does the concept make sense? Eric Eeckhout, *Heart* 2007; 93:632–638.
16. Management of acute myocardial infarction inpatients presenting with ST-segment elevation, *European Heart Journal* (2003) 24, 28–66.
17. Coronary Angiography, Shih-Yung James Chen and John D. Carroll, Department of Medicine, Anschutz Medical Campus, University of Colorado Denver, Aurora, CO, USA.
18. Percutaneous Transluminal Coronary Angioplasty (PTCA), Talia F. Malik- Wah Medical College, Vijai S Tivakaran- Kettering Health Network, Statpearls-NCBI Bookshelf.
19. Percutaneous Transluminal Coronary Angioplasty (PTCA) and Stenting-Study of 100 Cases, Dr. F Rahman, MD, Associate Professor (Intervention Cardiology). Prof. S Banerjee, MD, Professor of Cardiology, Dr. CM Ahmed, MD, Associate Professor of Cardiology, Dr. MS Uddin, MD, Associate Professor of Cardiology, Dr. Khirul Anam, Asstt. Professor, Dr. MS Alam, MBBS, MD- Card Student, Prof. KMHS S Haque, FCPS, Professor of Cardiology. Department of Cardiology, University Cardiac Center, BSMMU, Dhaka