(e) ISSN Online: 2321-9599

(p) ISSN Print: 2348-6805

ORIGINAL ARTICLE

A comparative study of Enoxaparin and fondaparinux in unstable coronary artery disease

Pratik Kumar Soni

Assistant Professor, Department of General Medicine, L.N. Medical College & Research Centre, Bhopal, Madhya Pradesh, India

ABSTRACT:

Background: Coronary heart disease is the leading cause of death in the US general population. With the obesity pandemic and the expected worsening of cardiovascular risk factors in the general population, the incidence and the prevalence of heart disease is expected to rise. The present study compared Enoxaparin and fondaparinux in unstable coronary artery disease. **Materials & Methods:** 82 patients of unstable angina of both genders were divided into 2 groups of 41 each. Group I patients were given Enoxaparin in the dose of 1 mg/kg body weight, subcutaneously, twice daily and group II was given Fondaparinux in a dose of 2.5 mg/kg body weight, once daily, subcutaneously. In both groups, risk factors and treatment outcome was compared. **Results:** Group I had 30 males and 11 females and group II had 28 males and 13 females. Common risk factors were smoking in 24 in group I and 23 in group II, diabetes in 30 in group I and 36 in group II, hypertension in 22 in group I and 26 in group II, family history 10 in group I and 7 in group II and >2 factors 15 in group I and 11 in group II. The recovery was seen in 36 at day 9 and 39 at day 30 in group I and 38 in group I at day 9 and 40 at day 30 in group II, recurrent MI was seen in 2 at day 9 in group I and 2 and 1 at fay 9 and day 30 in group II, hemorrhage was seen in 3 and 2 at day 9 and day 30 in group I and 1 at day 9 in group II. No mortality was reported in either of the group. The difference was non- significant (P>0.05). **Conclusion:** Both drugs were comparable in treatment outcome in patients with unstable angina.

Key words: Enoxaparin, Fondaparinux, Unstable angina.

Corresponding author: Dr. Pratik Kumar Soni, Assistant Professor, Department of General Medicine, L.N. Medical College & Research Centre, Bhopal, Madhya Pradesh, India

This article may be cited as: Soni PK. A comparative study of Enoxaparin and fondaparinux in unstable coronary artery disease. J Adv Med Dent Scie Res 2016;4(2):196-199.

INTRODUCTION

Coronary heart disease is the leading cause of death in the US general population. With the obesity pandemic and the expected worsening of cardiovascular risk factors in the general population, the incidence and the prevalence of heart disease is expected to rise.[1] Coronary artery disease (CAD) is the leading cause of death in patients with chronic kidney disease (CKD): Of the more than 320,000 patients with ESRD that requires dialysis or kidney transplantation in the United States, half will die from cardiovascular causes, and patients with milder degrees of CKD are more likely to die of CAD than to develop kidney failure that requires renal replacement therapy.[2]

Thrombosis is of prime significance, as was indicated by its presence at the event site, in unstable CAD and by improvement in clinical outcome, after antithrombotic therapy.[3] Platelet activation and coronary vasoconstriction are other events that contribute to the initiation of unstable CAD. Over the last two decades, major improvements has been achieved in the management of unstable coronary artery disease by anti- platelet agents, anticoagulants, thrombolytic therapy, combined with mechanical revascularization or reperfusion.[4]

Unfractionated heparin (UFH) is commonly used in patients with unstable CAD. However, UFH exhibits an unpredictable anticoagulant effect which requires frequent monitoring and it has low bioavailability due to high protein binding and induced thrombocytopaenia.[5] FD sodium, which is a synthetic, sulfated pentasaccharide, selective factor Xa inhibitor, is indicated for preventing thrombus formation in patients with acute coronary syndromes, including those with ST-segment Elevation Myocardial Infarction (STEMI), non-STEMI (NSTEMI), or unstable angina.[6] The present study compared Enoxaparin and fondaparinux in unstable coronary artery disease.

MATERIALS & METHODS

The present study was conducted among 82 patients of unstable angina of both genders. All were informed regarding the study and their written consent was obtained.

Data such as name, age, gender etc. was recorded. Patients were divided into 2 groups of 41 each. Group I patients were given Enoxaparin in the dose of 1 mg/kg body weight, subcutaneously, twice daily and group II was given Fondaparinux in a dose of 2.5 mg/kg body weight, once daily, subcutaneously. In both groups, risk factors and treatment outcome was compared. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Groups	Group I	Group II
Drug	Enoxaparin	Fondaparinux
M:F	30:11	28:13

Table I, graph I shows that group I had 30 males and 11 females and group II had 28 males and 13 females.

Table II Assessment of risk factors

Risk factors	Group I	Group II	P value
Smoker	24	23	0.12
Diabetic	30	36	
Hypertension	22	26	
Family history	10	7	
>2 factors	15	11	

Table II, graph I shows that common risk factors were smoking in 24 in group I and 23 in group II, diabetes in 30 in group I and 36 in group II, hypertension in 22 in group I and 26 in group II, family history 10 in group I and 7 in group II and >2 factors 15 in group I and 11 in group II. The difference between both groups was non-significant (P>0.05).

Graph I Assessment of risk factors

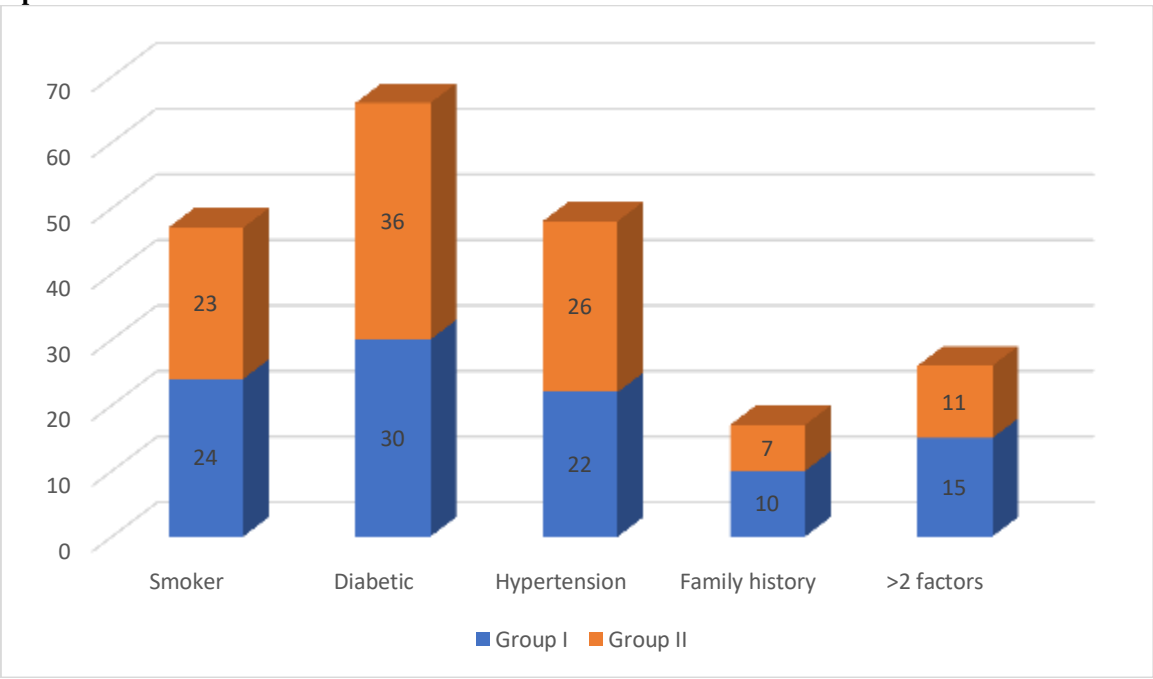
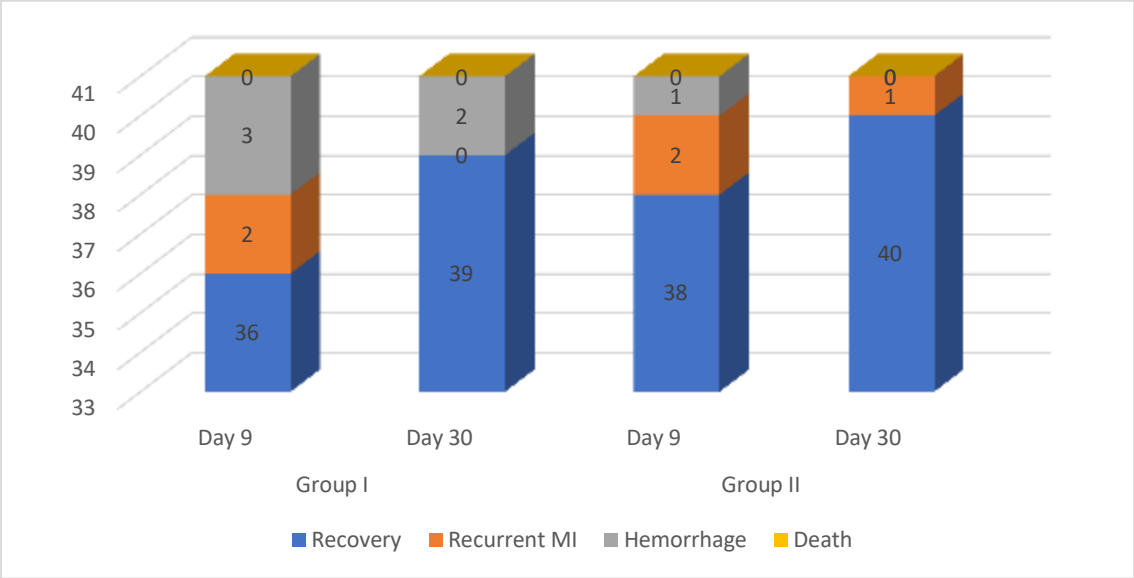


Table III Treatment outcome

Outcome	Group I		Group II		P value
	Day 9	Day 30	Day 9	Day 30	
Recovery	36	39	38	40	0.14
Recurrent MI	2	0	2	1	
Hemorrhage	3	2	1	0	
Death	0	0	0	0	

Table III, graph II shows that recovery was seen in 36 at day 9 and 39 at day 30 in group I and 38 in group I at day 9 and 40 at day 30 in group II, recurrent MI was seen in 2 at day 9 in group I and 2 and 1 at fay 9 and day 30 in group II, hemorrhage was seen in 3 and 2 at day 9 and day 30 in group I and 1 at day 9 in group II. No mortality was reported in either of the group. The difference was non- significant (P>0.05).

Graph I Treatment outcome



DISCUSSION

Cigarette smoking, another risk factor, alters LDL, reducing the endothelium-dependent relaxation induced by acetylcholine, in a similar way to oxidized LDL, without altering the non-endothelium dependent relaxation.[7] Both active and passive smoking are associated with the development of several clinical forms of CAD and diffuse atherosclerosis in men and women. Stable angina is repeated anginal attacks, which can develop over months or years are referred to as stable angina.[8] An increase in myocardial oxygen consumption usually due to physical exertion or emotions, triggers ischemia. It is relieved by rest or the use of vasodilators. The threshold for angina may vary. When the ventricular function is normal, as it frequently is, stable angina is compatible with an acceptable quality of life.[9] The present study compared Enoxaparin and fondaparinux in unstable coronary artery disease.

In present study, group I had 30 males and 11 females and group II had 28 males and 13 females. We found that common risk factors were smoking in 24 in group I and 23 in group II, diabetes in 30 in group I and 36 in group II, hypertension in 22 in group I and 26 in group II, family history 10 in group I and 7 in group II and >2 factors 15 in group I and 11 in group II. Shah et al[10] compared the safety and efficacy of Enoxaparin (EX) and Fondaparinux (FD) in patients with unstable coronary artery disease (UCAD). Recovery, recurrence, major and minor bleeding and biochemical investigations were evaluated and compared among two arms. The baseline demographic characteristics were similar in both groups, with mean age of 56.05 and 56.05 years in EX and FD group respectively. Recovery was equal in two arms. Recurrent MI or angina was seen numerically more in EX group, but it did not statistically vary from that in the FD group. Incidence of haemorrhage was similar in both groups at 9 days, but at 30 days, EX showed a higher incidence.

We found that recovery was seen in 36 at day 9 and 39 at day 30 in group I and 38 in group I at day 9 and 40 at day 30 in group II, recurrent MI was seen in 2 at day 9 in group I and 2 and 1 at fay 9 and day 30 in group II, hemorrhage was seen in 3 and 2 at day 9 and day 30 in group I and 1 at day 9 in group II. No mortality was reported in either of the group. Heart failure associated with CAD is mainly due to two conditions: ischemic cardiomyopathy and LV akinesia/dyskinesia (aneurysm). The ischemic cardiomyopathy is the ventricular dysfunction with diffuse hypocontractility and dilation of ischemic cause.[11] It can be a consequence of the replacement of fibrosis in areas where multiple, small infarctions have occurred, or of global myocardial dysfunction due to chronic ischemia and hibernation. The state of myocardial hibernation 91-93 is of great practical importance, for it indicates the presence of persistent ischemia with viable myocardium, therefore liable to recovery by restoring the blood flow.[12]

About 3% of the normal population and 30% of asymptomatic patients experience silent ischemia (SI) following an infarction of the myocardium. Most angina patients experience multiple ischemic episodes of painless ischemia, which amounts to 75% to 92% of the total ischemic load.[13] SI can be diagnosed by an ergometric test (ET) and Holter monitor. The prognosis depends mainly on the size of the risk area. SI is particularly troublesome because patients do not experience the classic warning sign: pain. Therefore, they may be at serious risk without knowing it.[14]

CONCLUSION

Author found that both drugs were comparable in treatment outcome in patients with unstable angina.

REFERENCES

1. Lewis HD Jr, Davis JW, Archibald DG, Steinke WE, Smitherman TC, Doherty JE 3rd, et al. Protective effects of aspirin against acute myocardial infarction and death in men with unstable angina. Results of a Veterans Administration Cooperative Study. N Engl J Med. 1983; 309(7): 396-403.
2. Lafeber M, Spiering W, van der Graaf Y, Nathoe H, Bots ML, Grobbee DE, et al. The combined use of aspirin, a statin, and blood pressure-lowering agents (polypill components) and the risk of vascular morbidity and mortality in patients with coronary artery disease. Am Heart J. 2013; 166(2): 282-89.
3. Warkentin TE, Levine MN, Hirsh J, Horsewood P, Roberts RS, Gent M, Kelton JG, et al. Heparin-Induced Thrombocytopenia in patients Treated with Low-Molecular Weight Heparin or Unfractionated Heparin. N Engl J Med. 1995; 332: 1330-35.
4. Ross R - The pathogenesis of atherosclerosis. An update. N Engl J Med 1986; 314: 488-500.
5. Moncada S, Higgs A - The L-arginine-nitric oxide pathway. N Engl J Med 1993; 329: 2002-12.
6. Berliner JA, Navab M, Fogelman AM, et al - Atherosclerosis: basic mechanisms. Oxidation, inflammation, and genetics. Circulation 1995; 91: 2488-96.
7. Simionescu N, Mora R, Vasile E, Lupu F, Filip DA, Simionescu M - Prelesional modifications of the vessel wall in hyperlipidemic atherogenesis. Extracellular accumulation of modified and reassembled lipoproteins. Ann N Y Acad Sci 1990; 598: 1-16.
8. Morel DW, DiCorleto PE, Chisolm GM - Endothelial and smooth muscle cells alter low density lipoprotein in vitro by free radical oxidation. Artheriosclerosis 1984; 4: 357-64.
9. Bevilacqua MP - Endothelial-leukocyte adhesion molecules. Annu Rev Immunol 1993; 11: 767-804.
10. Shah S, Khajuria V, Tandon VR, Gillani ZH, Lal M. Comparative evaluation of efficacy, safety and haemostatic parameters of enoxaparin and fondaparinux in unstable coronary artery disease. Journal of clinical and diagnostic research: JCDR. 2014 Jan;8(1):31.
11. Gimbrone MA Jr - Monocyte rolling, arrest and spreading on IL-4-activated vascular endothelium under flow is mediated via sequential action of L-selectin, beta 1-integrins, and beta 2-integrins. J Cell Biol 1994; 125: 1417-27.
12. Jang J, Lincoff AM, Plow EF, et al - Cell adhesion molecules in coronary artery disease. J Am Coll Cardiol 1994; 24: 1591-61.
13. Murota SI, Fujita H, Morita I, Wakabayashi Y - Adhesion molecule mediated endothelial cell injury elicited by activated leukocytes. Ann N Y Acad Sci 1995; 748: 133-47.
14. Steinberg D, Parthasarathy S, Carew TE, et al - Beyond cholesterol: modifications of low-density lipoprotein that increase its atherogenicity. N Engl J Med 1989; 320: 915-24.