http://dx.doi.org/10.4314/ajtcam.v11i1.18

CLINICAL STUDY OF THE HYPOTHESIS OF ENDOGENOUS COLLATERAL WIND ON ACUTE CORONARY SYNDROME: A REVIEW

Xian Wang[1], Cong Zhang[2], Ran Yang[1], Haiyan Zhu[1], Huaibing Zhao[1] and Xiaoming Li[1].

[1]Department of cardiology, Dongzhimen hospital Affiliated to Beijing University of Chinese Medicine, Beijing, 100700, China, [2]Beijing University of Chinese Medicine, Beijing, 100029,China

***E-mail:** wangxian20130101@126.com

Abstract

Background: Acute Coronary Syndrome (ACS), is a serious threat to people's health, and life, and in recent years, the incidence has increased yearly. This study was to propose the hypothesis of “endogenous collateral wind” based on the patho-mechanism of thrombogenesis complicated by ruptured plaque on ACS, and the theory of traditional Chinese medicine.

Materials and Methods: Through successful coronary angiography (CAG), and intravascular ultrasound (IVUS)， patients with coronary artery disease were made the differential diagnosis such as blood stasis, blood stasis due to phlegm obstruction, and endogenous collateral wind. The levels of plasma inflammatory marker were measured to study on the characteristics of “endogenous collateral wind”. Luo heng dripping pills with promoting blood circulation to expel wind-evil, and remove wetness were made based on the hypothesis of “endogenous collateral wind” on ACS. Patients with unstable angina were randomly divided into 3, groups based on therapeutic methods: conventional therapy group, Luo Heng dripping pills group and Tongxinluo caps. Differences among groups were compared.

Results: There were great changes in number and degree of coronary arteriostenosis confirmed by CAG, the types of ACC/AHA lesion and Levin lesion confirmed by CAG, remodeling index, positive or negative remodeling percentage measured by IVUS, the plasma levels of plasma inflammatory marker measured by ELLSA in the patients with endogenous collateral wind, compared with patients with blood stasis and blood stasis due to phlegm obstruction. The total effective rate of improved angina in Luo Heng dripping pills group was significantly higher than those in other two groups. The levels of plasma inflammatory marker were significantly lower in Luo Heng dripping pills group.

Conclusion: There were some pathological basis which were found about the hypothesis of “endogenous collateral wind” on acute coronary syndrome. It provided evidences for patients with coronary artery disease treated by medicines with expelling evil-wind, and removing wetness.

Key words: The hypothesis of “endogenous collateral wind”, acute coronary syndrome, Luoheng dripping pills, coronary artery disease.

Introduction

Atherosclerotic diseases are the main cause of death, severely hazardous to people's health and life, and about 19000, thousand people die of atherosclerotic diseases globally per aunm, because of coronary atherosclerotic disease (CAD) (Anderson M et al., 1987).The artherosclerosis was known as a progressive linear procedure in the past few years, which is due to the lipid deposition, the plaque under the endarterium gradually intruding the lumen of blood vessel, leading to stenosis and ischemia, and in the long run cardiovascular and cranial vascular disease would occur. The acute coronary syndrome (ACS) mostly happens in the patients with mild and moderate coronary stenosis, the main cause is the rupture of plaque and thrombogenesis. The form of thrombogenesis depends on the vulnerability of plaque (Morteza Naghavi MD et al., 2003).Today percutaneous coronary intervention(PCI), and coronary artery bypass graft (CABG) are becoming mature and perfect, and intravascular ultrasound (IVUS), is considered as a better method for confirming the vulnerable plaque, and the study of statin has been taken as an important breakthrough in stabilizing and reversing the plaque. The progress involves inflammation, immunization, metabolism, coagulation from the stability of atherosclerotic plaque to the instability, and inflammation is the key factor for the instability of atherosclerotic plaque (Van der Wal AC et al., 2003).Effective drugs improving the stability of atherosclerotic plaque are in short supply. But until now, the ACS, is still the first killer seriously imperiling the health of people, so it has significant meanings of exploring the therapeutic medicine for the CAD, including the combination of Chinese traditional, and Western medicine.

The base for proposing the hypothesis of “endogenous collateral wind” on the acute coronary syndrome

There is no reports about the hypothesis of “endogenous collateral wind” on the acute coronary syndrome within the internal and

http://dx.doi.org/10.4314/ajtcam.v11i1.18

external .The author thinks that the ACS, similar to the wind syndrome of Traditional Chinese Medicine (TCM), characterized by swift changes, rapid movement of clinical manifestation; the location of disease is situated in the heart, and the pathogen is wind-cold intrusion into the heart, unbalanced or irregular diet and emotion, physically weak as old age and so on; mechanism of disease is that the phlegm, static blood and dampness are depressing, evaporating and degenerating, which coagulate and shape toxin, becoming heat and wind; or the disease intruded into collaterals. And both of the hollow collaterals and blood deficiency could generate wind. Based on these theories, the authors propose the hypothesis of "endogenous collateral wind" (Xian Wang et al., 2002a; Xian Wang et al., 2002b; Xian Wang et al., 2003).

Wind, a pathogenic factor characterized by its rapid movement, swift changes, ascending and opening actions, and rapid movement of clinical manifestation, is refered to as pathogenic wind. *SuWen FengLun* "The wind is the leadership of other pathogenic factors, whose changes will generate other diseases". The wind may develop into other diseases, with no limitation of time and location, but the changes are from the wind, so we may examine the etiopathogenisis. *SuWen ZhiZhenYaoDaLun* said that "The wind and the dizziness belong to the liver". The wind includes the internal and external wind, and the wind also includes the one invading the external, the viscera and bowels and main and collateral channels. The internal wind includes liver wind stirring the internal wind, extreme heat engendering wind and liver blood deficiency transforming into wind. *SuWen ZhiZhenYaoDaLun* said that "The exuberant wind will shake the body", including the shaking of head and limbs, with dizziness, so that the external, and internal shaking belongs to the wind. The clinical manifestation of thrombogenesis complicated by ruptured plaque on the ACS manifests with sudden breaking out, of fierce chest pain, which belongs to the internal wind. *SuWen MiuCiLun* said that "The pathogen attacks kidney meridian which make sudden heart pain", showing that the pathogen invading the collateral, cause the heart pain. *Treatise on the Pathogenesis and Manifestations of All Disease* said that "The heart collateral is invaded by the wind of cold and heat which leads to chronic heart pain, if the measles erupt and disappear timely, he will not die, but chronic heart pain is not easier to cure". So the angina belongs to the disease of heart collateral, and the writer proposes the concept of "collateral wind".

Coronary Artery disease (CAD), is sthenia in origin and asthenia in superficiality, and the sthenia origin includes the deficiencies of the heart, liver, spleen and kidney, if the heart's yang deficiency starts not warming other visceras; heart yin-blood deficiency not moistening other organs, the heart collateral will not be nourished, so the angina may break out. The asthenia superficiality includes the cold-coagulate qi-stagnate, phlegm–retardant, and sluggish blood which block the heart collateral, and the angina may also break out. The phlegm and stagnant blood have the same origin, and hold each other. The phlegm grows out of the pathogenic damp belonging to fluid and the stagnant blood. At the same time, the fluid-humor and blood have the same source, so the phlegm is closely related to the stagnant blood. The pathological tangible outcomes of phlegm and stagnant blood obstruct the heart collateral, so the disease of collateral has two forms: If the patient is of short duration, the disease is situated at collateral, and then the collateral pass the disease on to the meridian, finally the disease will harm the viscera and bowels. This does not include the diseases caused by injury and operations. If the patient has a very long duration，the viscera and bowels suffer the initial damage which can pass the disease on to the meridian, and then the meridian passes the disease on to the collateral. The main pathogen are the pathological tangible phlegm and stagnant blood, so the clinical manifestation is pain.*SuWen BiLun* said "The aeipathia will invade the body deeply, nutrient-defense-qi doesn't move smoothly，and meridian and collateral qi will stagnate, so the qi will be obstructed". Some studies on pharmacokinetics (Jianxin Chen et al., 2012a) suggested that the pathological tangible outcomes of phlegm and stagnant blood obstruct the heart collateral, so the artherosclerosis will be formed. The three theories on formed artherosclerosis are generally accepted: the theory of lipid－invading, thrombus－forming, and the theory of damage－reflecting. The theory of lipid－invading consider the artherosclerosis as the reflecting of blood vessel on lipid invading, the traditional Chinese medicine resolving phlegm and relieving dampness can improve the high lipid patients' clinical symptoms, so we can conclude the lipid invading may be closely correlated with the phlegm jamming the heart collateral (Xian Wang et al., 2002a); the viewpoint of stagnate blood blocking heart collateral is identical with the theory of thrombus -forming, the theory of damage－reflecting thinking that some damageable factors such as the blood shearing force caused by high cholesterol, hypertension may not transform the shape of endotheliocyte, but will change the permeability of endotheliocyte, the plasma lipoprotein easily permeate into the arterial wall, and promote the phlegm entering the collateral (Xian Wang et al., 2002a)[l]. The seriously damaged endothelium may result in endothelial cell desquamation; the collagen under endothelium will touch with the platelet and other blood ingredients, and then lead to the platelet aggregating, thrombus -forming, the stagnant blood blocking.

The deficiency of the healthy qi and excessiveness of the pathogenic qi block the collateral with the passing of time, the phlegm, stagnate blood and dampness is depressing ,evaporating and degenerating which condense into the toxin, bear heat and wind，or the phlegm and stagnant blood choke heart channels, the aeipathia invade collateral, the channels will be empty, the wind engendered from extreme heat and from liver blood deficiency both belong to the "collateral wind" (Xian Wang et al., 2002a; Xian Wang et al., 2002b; Xian Wang er al., 2003).The origin of "collateral wind" lies in the phlegm turbidity, stagnant blood and dampness turbidity which may evaporate into water qi ,condense into the

http://dx.doi.org/10.4314/ajtcam.v11i1.18

toxin, and then phlegm-dampness turbidity,stagnant blood, water qi and heat toxin may aggregate into soft plaque, the soft plaque is vulnerable, the ACS will be happen.

So the diagnosis standard of endogenous collateral wind syndrome is summarized as follows: the main symptoms are chest pain; chest tightness or discomfort; the secondary symptoms are; the pain is sudden or intremitent when it happens, and can spread from shoulder to arm; the tongue and pulse; the clour of the tongue is dark or we can see ecchymosis, and petechiae; the pulse is wiry and frequent pulse; or maybe weak. Diagnosis: one of the main syndromes and one of the secondary symptoms combining the tongue and pulse can make a diagnosis.

The study of coronary angiography on the hypothesis of "endogenous collateral wind" in the ACS

Our previous studies showed that the number of stenotic coronary branches increase in proper sequence from the syndrome of stagnant blood internal blocking, phlegm obstructing and stagnant blood silting, the significant difference were observed in the syndrome of "endogenous collateral wind" as compared with the other syndrome($P<0.01$).The degree of coronary stenosis for the syndrome of endogenous collateral wind was seriously stegnotic ($P<0.01$), as compared with the other syndrome, the degree of coronary stenosis for the other syndrome was similar. The stegnotic type and gradually complicated from the syndrome of stagnant blood internal blocking, to the syndrome of phlegm obstruction, and stagnant blood silting, and then the syndrome of "endogenous collateral wind" in accordance with the ACC/AHA, criterion, the stegnotic type of the patients with the syndrome of "endogenous collateral wind" showed almost B2 and C type, the stegnotic type of the patients with the syndrome of stagnant blood internal blocking, and the syndrome of phlegm obstructing and stagnant blood silting showed almost A and B1 type . Ridit analysis revealed the significant difference observed in the syndrome of "endogenous collateral wind" as compared with the other syndrome($P<0.01$). In brief, the CAD with "endogenous collateral wind" was much more stegnotic in coronary branches, the degree of coronary stenosis was seriously stegnotic, showed almost B2, and C, type. Our earlier studies showed the degree of coronary stenosis as almost ≥50%, in the group of blood-stasis syndrome and the group of phlegm obstruct-blood stasis syndrome, the two groups showed almost A type according to the ACC/AHA, standard, the significant difference were observed as compared with the group of "endogenous collateral wind" ($P<0.05$).The degree of coronary stenosis was almost 95-99%, in the group of "endogenous collateral wind", showed chiefly B2 and C type according to the ACC/AHA standard, the significant difference were observed as compared with the group of blood stasis syndrome and the group of phlegm obstruct-blood stasis syndrome($P<0.05$), but there was no significant difference between the group of blood stasis syndrome and the group of phlegm obstruct-blood stasis syndrome ($P>0.05$). The II type in the group of "endogenous collateral wind" exceed the other groups according to the Levin standard ($P<0.05$), the I and III type were almost observed in the group of blood stasis syndrome and the group of phlegm obstruct-blood stasis syndrome, significant difference were observed as compared with the group of "endogenous collateral wind", but there was no significant difference between the group of blood stasis syndrome, and the group of phlegm obstruct-blood stasis syndrome ($P>0.05$).The group of blood stasis syndrome and the group of phlegm obstruct-blood stasis syndrome was approximate in the degree of coronary stenosis, the pathological type according to the ACC/AHA, standard and Levin standard, which showed the phlegm was closely related to the stasis in the patient with CAD, also showed the atherosclerotic plaque was relatively stable in the patients with blood stasis syndrome and phlegm obstruct-blood stasis syndrome, the degree of coronary stenosis was mild ,the pathological type simple according to the ACC/AHA, standard and Levin standard. The degree of coronary stenosis was severe in the patient with "endogenous collateral wind" syndrome, the pathological type was complicated according to the ACC/AHA standard and Levin standard, the pathological type was cusp angle, niche, uneven in surface ,crater shape, which implies that the soft plaque increase much more in the endogenous collateral wind syndrome as compared with blood stasis syndrome and the phlegm obstruct-blood stasis syndrome, the plaque was inclined to be unstable, also fully prove the soft plaque and unstable plaque were closely related to the pathological production of phlegm- blood stasis, heat toxin,water retention,phlegm turbid.

The study of inflamed mechanism on the hypothesis of "endogenous collateral wind" in the ACS

The atherosclerotic plaque is decided by multiple factors, the inflammation inside plaque is a key factor which may result in unstable plaque, the rupturing plaque and anabrotic plaque exist together with the inflammation, the inflammation inside plaque is up-regulated in clinical unstable state (Van der Wal AC et al., 2003).The incidence of inflammation in superficial, is correlated with the degree of coronary stenosis, and the rupture of plaque, which confirms that it is the fibre cap not the inflammation deep inside plaque that play key role in the rupture of plaque (Bayes-Genis A et al., 2001) .Many proof indicate CRP as sensitive inflammation index is the strong predictor of cardiovascular event. Our study showed the level of plasma hs-CRP as having no significant difference in each group, but the level of plasma MMP-2 was closely correlated

http://dx.doi.org/10.4314/ajtcam.v11i1.18

with the group of endogenous collateral wind(r=0.486,P＜0.05) ,which clearly indicated CRP, and MMP-2 probably participate in the inflammatory reaction of atherosclerotic plaque, both affected the prognosis of patients with endogenous collateral wind. It was the main internal reason that the vascular smooth muscle cell composed extracellular matrix decrees, and albumen resolved degrade extracellular matrix increasingly ,the MMP gave full scope to the latter. Our study indicated (Schonbeck U et al., 1999) the level of plasma MMP-2 and MMP-9 in the group of endogenous collateral wind syndrome exceeded the group of blood stasis syndrome and the group of phlegm obstruct- blood stasis syndrome(P＜0.05),but the level of plasma MMP-2and MMP-9 did not differ between the group of blood stasis syndrome and the group of phlegm obstruct-blood stasis syndrome (P>0.05).The ascending level of plasma MMP-2 and MMP-9 in the group of endogenous collateral wind syndrome showed MMPs as inflammation factor participate in the progress of vulnerable plaque. In recent years，the study considered the soluble CD40, molecule and its CD40, ligand as the hinge of immunological and inflammatory reactions, its biological effect up-regulated the expression level of plasma MMP, and then affected the stability of plaque (Schonbeck U et al., 1999).Our study showed that the level of plasma CD40L was significantly different in each group, but the level of plasma CD40L was significantly positively correlated with the level of plasma PAPP-A(r=0.766,P＜0.001), which indicated CD40L might play a big role in mediators of inflammation by means of PAPP-A, in this field, we consulted the control of medicine (Jianxin Chen et al., 2011a) . PAPP-A was recently discovered as calcium and zinc dependent MMP, the level of plasma PAPP-A was obviously elevated in the peripheral blood, the matrix inside atherosclerotic plaque, the smooth muscle cell, monocyte at the patient with ACS(Bayes-Genis A et al., 2001). Our study showed(Schonbeck U et al., 1999) the level of plasma PAPP-A in the group of endogenous collateral wind syndrome significantly exceeded the group of blood stasis syndrome and the group of phlegm obstruct-blood stasis syndrome(P＜0.05),which indicated the elevated level of plasma PAPP-A was probable correlated with the happening of atherosclerotic plaque rupture. We observed the level of plasma hs-CRP,MMP-2,MMP-9,PAPP-A was correlated with each other in the endogenous collateral wind syndrome, the level of plasma MMP-2 was significantly and positively correlated with hs-CRP and PAPP-A(r1=0.486, r2=0.544,P＜0.05),the level of plasma CD40L was also significantly and positive;y correlated with PAPP-A(r=0.766,P＜0.001).This indicated that the atherosclerotic plaque rupture was related to the expression of PAPP-A stimulated by the elevated level of plasma hs-CRP,CD40L,MMP-2.The authors concluded that the inflamed mechanism hypothesis of “endogenous collateral wind” in the ACS was valid in view of the above：the phlegm-blood stasis,dampness turbid,water vapor,heat toxin in atherosclerotic plaque was impelled to be depressing, evaporating and degenerating, transforming into heat and engendering wind, which was the result of solo acting or interacting with each other by the inflamed mediators of hs-CRP,MMP-2,MMP-9,CD40L,PAPP-A.Or if the heat toxin gathers with the passing of time, and then the channels will become incapacitated , the wind will be engendered from blood deficiency, which was the hypothesis of “endogenous collateral wind” in the ACS.

The study of intravascular unltrasound(IVUS)on the hypothesis of “endogenous collateral wind” on ACS

The artery remodeling at atherosclerotic plaque is so much concerned recently, the artery remodeling was first reported by Glagov (Glagov S et al., 1987) and others at autopsy study, was subsequently confirmed at IVUS study. The artery remodeling was known as compensatory mechanism to prevent the reduction lumen of blood vessel at atherosclerotic early stage, the artery positive remodeling may increase the compensatory cross section area of lumen partial blood vessel, the artery negative remodeling may decrease the compensatory cross section area of lumen partial blood vessel, the recent study show the clinical manifestation of ACS, is correlated with the artery remodeling (Hermiller JB et al., 1993). Weizhong Zhu and others investigated 76 patients with ACS and 40 patients with stable angina (Weizhong Zhu et al., 2009), the outcome showed the exterior elastic membrane area, the plaque area and the artery remodeling rate significantly increased in the patients with unstable angina compared with the patients with stable angina, the patients with unstable angina showed that the artery positive remodeling (51% compared with 18%,P=0.002), but the patients with stable angina showed the artery negative remodeling (58% compared with 33%,P=0.002). According to Chen and others’ studies (Jianxin Chen et al., 2007; Jianxin Chen et al., 2010; Jianxin Chen et al., 2012b), our studies showed the remodeling index (RI) of endogenous collateral wind syndrome group significantly exceeded the group of blood stasis syndrome(1.407±0.134) compared with (0.916±0.115)](P=0.015), the group of endogenous collateral wind syndrome frequently showed artery positive remodeling (50% compared with 10%,P=0.040),the group of blood stasis syndrome frequently showed artery negative remodeling (70% compared with 27%,P=0.064).Taking RI as dependent variable, the group (the group of blood stasis syndrome and the group of endogenous collateral wind syndrome), as independent variable, the outcome of linear regression analysis showed significant regression coefficient between the grouping and the RI(P＜0.05),which indicated the group of blood stasis syndrome and the group of endogenous collateral wind syndrome might be the independent variable for forecasting the coronary artery remodeling.

Based on the relationship between the coronary artery remodeling and the blood stasis syndrome, the endogenous collateral wind

http://dx.doi.org/10.4314/ajtcam.v11i1.18

syndrome was useful for us to understand that the hypothesis of “endogenous collateral wind”, the high incidence of artery negative remodeling in the group of endogenous collateral wind syndrome indicate double-effect on the close relationship between the fibrosis, the contraction of blood vessel area. The patients with endogenous collateral wind syndrome had much more soft plaque（containing much more lipid）than the patients with blood stasis syndrome, have fewer calcific plaque and intermix plaque. The patients with endogenous collateral wind syndrome had larger remodeling plaque which was easy to be impacted by the mechanical force, and then leaded to the clinical manifestation of ruptured and unstable plaque. From the studies of coronary artery remodeling in IVUS, we could presume the effect of artery positive remodeling might decrease the stenosis of coronary artery, but at the same time might increase the possibility of ruptured plaque and cardiac incident in the patients with endogenous collateral wind syndrome, but the patients with blood stasis syndrome exhibited the effect of artery negative remodeling, the fiber change related with the effect of artery negative remodeling might increase the capability of anti-ruptured plaque, so the plaque in the patient with blood stasis syndrome is relatively stable. On the whole, the patients with endogenous collateral wind syndrome had the weightier loading, the higher incidence of risk plaque, the higher of RI, frequently had artery positive remodeling in the IVUS, which indicated the essence of hypothesis on “endogenous collateral wind” in the ACS was unstable plaque, higher possibility of ruptured plaque. The ruptured plaque is related to the absence of extracellular matrix in some part, often take place in the shoulder region (Ikeda U et al., 2003).The collagen affect the stability of plaque, the increased expression of MMP may adjust the coronary artery remodeling in the patient with ACS. Our study showed [9] the level of hs-CRP in the group of endo-grnouds collateral wind syndrome was relatively higher than the group of stable angina(P=0.033). Although the level of plasma MMP-2and MM0-9 in the group of positive remodeling was not significantly higher than the group of negative remodeling (P>0.05), according to the grouping of coronary artery remodeling. But according to the grouping of plaque character, the level of plasma MMP-2 and MMP-9 in the group with higher risk plaque was averagely higher than the group with no higher risk plaque (P=0.011,P=0.001), which indicated the inflammation mediator of hs-CRP,MMP-2 and MMP-9 may affect the progress of coronary artery remodeling. But taking RI as dependent variable, taking grouping and other inflammatory mediators as independent variable, the outcome from linear regression analyzed showed there was significant regression coefficient only existing between the grouping and RI (P＜0.05),which indicated the syndrome differentiation and grouping of TCM maybe the independent variable forecasting the cardiovascular remodeling. Our study also showed that the level of plasma CD40L and PPP-A was significantly higher in the group of endogenous collateral wind and the group of higher risk plaque, indicated that the CD40L along with MMP-2 and MMP-9,and other inflammatory mediators participate in the progress of cardiovascular remodeling through affecting the collagen in plaque fiber head cap.

LuoHeng dripping pills stabilizing the coronary artery atherosclerotic plaque

The strategy of prevention and cure for CAD emphasize the methods of activating blood and resolving stasis, and employ wind-medicinal aiming directly at the hypothesis of “endogenous collateral wind” in the ACS, at the same, we consulted the thoughts in medicine of JiangZhiNing (Jianxin Chen et al., 2012c; Jianxin Chen et al., 2011b). Endogenous collateral wind should be treated by activating blood to free the collateral vessels, resolving stasis to dispel wind. On the basis of《*TaiPingShengHuiFang*》“treating the cardiodynia, chest distress going to be dead by the pills of Cynanchum paniculatum one liang, Benzoin one liang”,the cynanchum paniculatum is principal drug, its function can activate blood to free the collateral vessels, resolve stasis to dispel wind, the ministerial drug is Borneol which can activate blood and relieve orifice, and along with others Chinese medicinal materials compose the Luo Heng dripping pills, the initial curative effect is good. The pharmacodynamic study confirmed the function of Cynanchum paniculatum is sedative, paregoric, antiinflammatory, and can decrease total cholesterol and β-lipoprotein, improve myocardial ischemia. Compared with the control group, the incidence of artherosclerosis is lower, the streakiness of mass plaque dispersed and fewer in the Cynanchum paniculatum group.The Chinese medicinal materials in the formula may prevent the ACS attack through the function of anti-inflammatory, adjusting- lipid ,anti-thrombus, sedative, analgesia effects. The 90, patients with unstable angina were randomly divided into 3, groups including the conventional therapy group (30 case),the LuoHeng dripping pills therapy group (30 case), the TongXinLuo therapy group (30 case), was separately given the LuoHeng dripping pills,TongXinLuo caps on the basis of conventional therapy. The study showed the total effective rate of relieving angina was 93, percent in the LuoHeng dripping pills therapy group which , was significantly higher than the TongXinLuo therapy group and the conventional therapy group (P＜0.05,P＜0.01). The improvement rate of electrokardiogramis was not significantly different among each group (P>0.05).The level total plasma cholesterol was significantly decreased in the LuoHeng dripping pills therapy group at post-treatment(P＜0.05),but in the conventional therapy group and the TongXinLuo therapy group the decrease was not significant (P>0.05). At prior treatment, the level of plasma CRP、MCP-1、TNF-α、IL-6 is averagely higher than the normal control group (P＜0.05), the inflammatory index level at post-treatment did not change significantly. the level

http://dx.doi.org/10.4314/ajtcam.v11i1.18

seen at prior treatment in the conventional therapy group and the TongXinLuo therapy group(P>0.05). But the inflammatory index level decreased significantly at post-treatment in LuoHeng dripping pills therapy group(P＜0.05). It could be concluded that the LuoHeng dripping pills group was superior to the TongXinLuo therapy group in relieving clinical symptoms of unstable angina, reducing the level of lipid, reducing inflammatory reaction, the LuoHeng dripping pills group might reverse the pathological production of phlegm-blood stasis, heat toxin, water retention, phlegm turbid which would be depressing, evaporating and degenerating ,and then stabilized the atheromatous plaque, treat the patients with unstable angina .

Acknowledgement

This study was supported by a Grant from the Foundation of Beijing TCM (NO.WX2002).

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