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Review On Silybum Marianum Exhibiting Hepato-Protective Activity

Aavany V.N[3],Aleena Babu[3],Anaswara.O[3]Anjana.K[3],Arathy.K.R[3`], Mathew George*[1] and Lincy Joseph[2]

Holy Queen College Of Pharmaceutical Sciences and Research, Thrissur-680623, Kerala.

1. Dr. Mathew George, Professor, Holy Queen College of Pharmaceutical Sciences and Research, Puthuruthy, Thrissur-680623, Kerala, India

*Corresponding Author: Dr. Mathew George, professor, Holy Queen College of Pharmaceutical Sciences and Research, Puthuruthy, Thrissur-680623, Kerala, India

2. Dr. Lincy Joseph, Professor, Holy Queen College of Pharmaceutical Sciences and Research, Puthuruthy, Thrissur- 680623, Kerala, India

3. Research Scholars, Holy Queen College of Pharmaceutical Sciences and Research, Puthuruthy, Thrissur-680623, Kerala, India

ABSTRACT

The use of medicinal plants in treating illnesses has been reported since ancestral times. In the case of hepatic diseases, Silymarin is a natural compound derived from the species Silybum marianum, which is commonly known as Milk thistle. This plant contains at least seven flavoligands and the flavonoid taxifolin. The hepatoprotective and antioxidant activity of silymarin is caused by its ability to inhibit the free radicals that are produced from the metabolism of toxic substances such as ethanol, acetaminophen, and carbon tetrachloride. The generation of free radicals is known to damage cellular membranes and cause lipoperoxidation. Silymarin enhances hepatic glutathione and may contribute to the antioxidant defense of the liver. It has also been shown that silymarin increases protein synthesis in hepatocytes by stimulating RNA polymerase I activity. A previous study on humans reported that silymarin treatment caused a slight increase in the compared with untreated controls.

Keywords: Silybum marianum, Hepatoprotector, Lipoperoxidation, Silymarin

1. INTRODUCTION

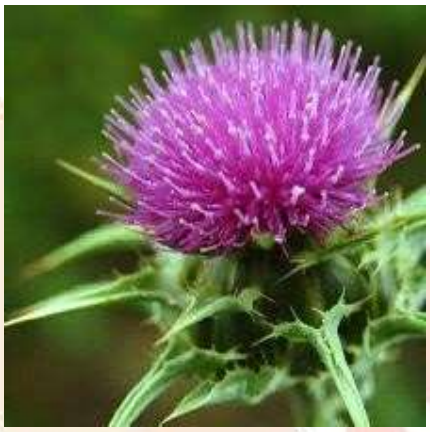
Silybum marianum is the scientific name for Milk thistle or St. Mary's thistle. It is a plant native to the Mediterranean region and belongs to the Asteraceae family. It is characterized by thorny branches and a milky sap, with its oval leaves reaching up to 30 cm. The flowers are bright pink and can measure up to 8 cm in diameter. Milk thistle grows in its wild form in southern Europe, northern Africa, and the Middle East. The plant is cultivated in Hungary, China, and South American countries such as Argentina, Venezuela, and Ecuador. In Mexico, Milk thistle is consumed as a supplementary food.[2] Silymarin is a natural compound that is present in species derived from Silybum marianum, which is commonly known as Milk thistle. The plant contains at least seven flavolignans and the flavonoid taxifolin. The most important flavolignans present include silybin, silydianin, and silychristine.

Silybin represents between 50% and 70% of the extract from silymarin. The following flavolignan isoforms are known: silbyna A, silbyna B, isosilbyna A, and isosilbyna B. Silymarin has been used worldwide for many years as a complementary alternative medicine because of the beneficial effects associated with the treatment of hepatic diseases. Silymarin belongs to the Aster family (Asteraceae or Compositae). The mature plant has large brilliant-purple flowers and abundant thorns. The plant grows in places with sufficient sun exposure.[1]

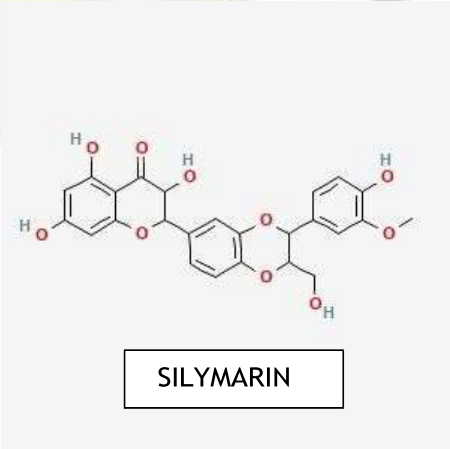
2. AIM

Here the aim is to review of hepatoprotective activity in silybum marianum and to identity the compounds which are responsible for the hepatoprotective activity.

3. MILK THISTLE[6]



According to Angelo et.al, the seeds of this plant contain many compounds such as silybin, silibinin A and B, silicristin, silidianin,apigenin, dehydrosilybin, deoxysilyn cristin, among other. Extract of dried seed of this plant contains up to 4% Silymarin. Silymarin is a combination of flavonoids such as Silibinin.[5]



PHARMACOLOGICAL ACTIVITY

In this study, laboratory animals (often rats) were divided into different groups. One group received only diclofenac to induce toxicity. Another group received both diclofenac and an extract of Silybum marianum. Control groups were also maintained—one received no treatment, and one received only Silybum marianum. The treatments were given over a certain period (commonly 7–28 days). Blood and tissue samples were collected at the end of the study for biochemical and histological analysis.The study used a

controlled experimental design. Diclofenac was administered to induce liver and/or kidney toxicity. Silybum marianum extract was given either before or along with diclofenac to test its protective effects. Researchers analyzed various markers of liver and kidney damage, such as ALT, AST, creatinine, and urea levels. Histopathological examination of liver and kidney tissues was also done to observe structural damage or healing.[7]

In this study, researchers used laboratory animals (typically rats) divided into groups:A control group received no treatment.A toxic group received a high dose of paracetamol to induce liver toxicity.Treatment groups received Silybum marianum extract either before or after paracetamol administration.Blood and liver tissue samples were collected. Researchers measured liver enzymes (like ALT, AST) and performed histopathological examinations of liver tissue to observe damage. They also assessed antioxidant levels and oxidative stress markers to understand the protective mechanism of Silybum marianum.[4]

The experimental methodology typically involves the use of laboratory animals, such as rats or mice, which are divided into several groups: a control group, an ethanol-treated group, and one or more groups treated with both ethanol and Silybum marianum extract. Ethanol is administered over a specific period to induce liver toxicity, mimicking the damaging effects of alcohol on the liver. The Silybum marianum extract, often standardized for its active compound silymarin, is given orally at measured doses either prior to, during, or after ethanol exposure to evaluate its protective or therapeutic effect. Blood samples are collected to assess biochemical markers such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), and oxidative stress markers. In addition, liver tissues are examined histologically to observe structural damage, inflammation, and cellular degeneration. These combined methods help determine whether Silybum marianum can counteract or reduce the harmful effects of ethanol on liver function and tissue integrity.[5]

The protective effects of Silybum marianum against thioacetamide (TAA)-induced toxicity, researchers typically use animal models, most often rats. The animals are divided into several groups: a normal control group, a TAA-only group, and one or more groups treated with both TAA and Silybum marianum extract. Thioacetamide is administered to induce liver damage, as it mimics liver cirrhosis and fibrosis similar to that found in chronic liver diseases in humans.The milk thistle extract, which contains silymarin as the active compound, is given either orally or by injection for several days or weeks, either before or after TAA administration. Researchers monitor the animals for signs of liver injury using blood tests that measure liver enzymes (ALT, AST, ALP), oxidative stress markers (like malondialdehyde or MDA), and antioxidant enzyme levels (such as glutathione, SOD, and catalase). Liver tissue is also examined under a microscope for structural damage or fibrosis.[3]

DISCUSSION

The study used a controlled experimental design. Diclofenac was administered to induce liver and/or kidney toxicity. Silybum marianum extract was given either before or along with diclofenac to test its protective effects. Researchers analyzed various markers of liver and kidney damage, such as ALT, AST, creatinine, and urea level,Histopathological examination of liver and kidney tissues was also done to observe structural damage or healing.

The group treated with Silybum marianum showed significantly lower levels of liver enzymes compared to the paracetamol-only group, indicating reduced liver damage. Histological analysis revealed that Silybum marianum preserved liver structure, reduced inflammation, and prevented necrosis. Additionally, antioxidant levels were higher, and oxidative stress was lower in treated animals.

The results indicated that ethanol administration led to significant liver damage, characterized by elevated levels of ALT and AST, increased oxidative stress, and visible structural damage in liver tissue. However, in animals treated with Silybum marianum, these effects were significantly reduced. The extract lowered liver enzyme levels closer to normal, increased antioxidant enzyme activity, and reduced lipid peroxidation, as evidenced by decreased MDA levels and increased GSH content. Histological analysis also revealed that liver cells in the Silybum marianum-treated group retained a more normal structure compared to the severe degeneration observed in the ethanol-only group. These findings demonstrate the hepatoprotective effect of Silybum marianum.

The group treated with thioacetamide alone usually shows significant liver damage. This includes high levels of liver enzymes, indicating cellular damage, increased oxidative stress, and histological signs of fibrosis and necrosis in liver tissue. In contrast, the groups treated with both TAA and Silybum marianum show clear improvement. Liver enzymes are significantly reduced, indicating less liver injury. Antioxidant enzyme levels are restored, and oxidative stress markers are reduced. Microscopically, the liver tissues show fewer signs of damage, reduced fibrosis, and better overall structure.

CONCLUSION

In the study of Ali Nouri et.al ,concluded that the prescence of silymarin compound ,exhibiting hepatoprotective activity aganist Diclofenac induced hepatotoxicity in tested often rats. Gabriel Fernando et.al,concluded the prescence of silymarin ,exhibited hepatoprotective activity against acetaminophen induced hepatotoxicity in tested rats,Karen et.al,concluded thath the prescence of silymarin compound ,exhibiting hepatorotective against ethanol intested rats.Najmeh Kabiri et.al,concluded that the prescence of silymarin ,exhibiting heptoprotective activity against thioacetamide induced hepatotoxicity in tested albino rats.

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