Evaluation of anti-osteoarthritic activity of *Vigna mungo* in papain induced osteoarthritis model

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**ABSTRACT**

**Aim:** This study was carried out to evaluate the effect of *Vigna mungo* hydroalcoholic extract (VMHA) by papain induced osteoarthritis (OA) in the rat model.

**Materials and Methods:** OA was induced by intra-articular injection of papain (4% w/v) along with cysteine (0.03 M) on day 1, 4 and 7 in rats and VMHA was administered orally in three doses (100, 200 and 400 mg/kg) after last papain injection. The anti-osteoarthritic activity was evaluated by measuring knee joint diameter, grip strength, locomotion activity and hanging time. Histopathological analysis and acute toxicity study were also performed.

**Results:** VMHA improved inflammatory condition with all the doses, but significant ($P < 0.05$) attenuation of inflammation was present only with 400 mg/kg dose. The grip strength, locomotion activity and hanging time were also significantly ($P < 0.05$) improved at dose level of 100 mg/kg however other two doses (200 mg/kg and 400 mg/kg) were not found to be effective. VMHA did not show any mortality or any toxic clinical signs after oral administration of 2 g/kg dose.

**Conclusion:** VMHA improved arthritic condition by significantly reducing pain and inflammation.

**KEY WORDS:** Grip strength, inflammation, osteoarthritis, papain

**Introduction**

Osteoarthritis (OA) is a musculoskeletal system disorder following mechanical and biological events that destabilize normal coupling between degradation and synthesis within articular cartilage.[1] Among many types of arthritis, OA is most commonly occurring joint degenerative disorder,[2] characterized by chronic inflammation, pain, joint stiffness and loss of mobility.[3] The prevalence of OA in Indian population is 22–39%[4,5] and thus it could be the major cause of disability in the near future. The pathophysiology of OA is not yet fully understood but it is believed to be mediated through various factors including tumor necrosis factor α (TNF-α), interleukin β (IL-β), matrix metalloproteinases (MMP), inducible nitric oxide synthase and C reactive protein.[6]

The widely used synthetic therapy with nonsteroidal anti-inflammatory drugs and disease-modifying anti-rheumatic drugs is the currently available as treatment option for OA, but toxicity, side-effects and reappearing of symptoms on discontinuation limits its usage.[6] Due to limitation of pharmacological treatment various alternative treatments would be of much importance. This alternative treatment mainly includes use of herbal remedies and other therapies.[7]

*Vigna mungo* Linn. (VM) is commonly known as black gram and is mainly cultivated in India and Pakistan. Traditionally, VM is mentioned for its beneficial effects in many of the ailments like ostalgia, abscess, inflammation, rheumatism and asthma.[8] Moreover, the seeds are also diuretic, emollient, appetizer, thermogenic, nervine tonic, laxative, aphrodisiac, astringent, styptic and galactagogue. They are also advantageous in managing epistaxis, schizophrenia, scabies, hemorrhoids, gonorrhea, leukoderma, asthma, heart problems, indigestion, anorexia, pains, constipation, hepatopathy, agalactia, neuropathy, hysteria, nervous debility, partial paralysis, facial paralysis and weakness of memory.[9]

Apart from these properties seed are also used in erectile dysfunction and premature ejaculation. Seeds also maintain as well as improve hair growth and protect them from dandruff.[9]
In addition to these VM has been reported pharmacologically to possess anti-inflammatory activity, analgesic activity, anti-cancer activity and hepatoprotective activity. It has also been described for its MMP inhibiting activity. Thus, the rationale of selecting VM was to explore its role as anti-osteoarthritic Herb. Several in-vivo animal models are available for screening of anti-osteoarthritic activity, but papain induced OA is one of those models, which mimic the conditions found in human OA. Thus, the present study was designed with an objective of validating the role of VM for its facilitatory effect on OA in appropriate animal models (Papain induced OA), which could pave the way for discovery of new leads with novel biodynamic actions.

Materials and Methods

Plant Material

The seeds of VM were collected from the local market of Kamothe, Navi Mumbai, Raigadh district, India. Samples were authenticated by Dr. Ganesh Iyer, Ruia College Matunga, Mumbai. Samples were preserved at the Department of Botany of Ruia College for further reference.

Extraction of Plant Drug

Seeds of the VM were grounded to a fine powder. Dried fine powder of plant drug was first defatted with petroleum ether to remove fatty materials and other pigmentation. Powder was dried and again used for extraction with 50% v/v ethanol by soxhlet extraction process. Ethanolic content from extract was removed by distillation while water content was removed by using a tray drier at 60°C. The yield of the extract for VM was 6–8%. Dried extract was stored at 4°C in the refrigerator for further use during the experiment.

Characterization of Plant Extract

Various physicochemical parameters including extractive values, ash values and loss on drying values were studied by standard procedure along with preliminary phytochemical screening.

Animals

Female Wistar rats weighing 180–200 g obtained were used for the experiment. The animals were housed in polycarbonate cages at room temperature (25 ± 2°C) and humidity (75 ± 5%) with 12:12 h light-dark cycle. The animals were acclimatized for 1 week before starting experimental work. The present study was approved by the Institutional Animal Ethics Committee formed as per the guidelines of Committee for the Purpose of Control and Supervision on Experiments on Animals.

Chemicals and Instrumentation

Papain was obtained as gift sample from advanced enzyme technologies, Mumbai. Cysteine (Papain activator), hematoxylin and eosin stain were purchased from Hi-media, Mumbai. Celecoxib capsules (GSK Pharmaceuticals) were purchased from local market pharmacy store.

Digital Vernier caliper (Unitech instrumentation and control) was used to measure knee joint diameter. Grip strength meter (Columbus instrument) was used to measure grip strength of animals. Activity meter was used to (Columbus instrument) was used to measure locomotor activity. Histopathology slides were prepared using tissue processor (Lieca instruments), tissue embedder (Lieca instruments), microtome (Lieca instruments) and auto-stainer (Lieca instruments). Histopathology slides were read using digital microscope (Lieca instruments).

Acute Toxicity Study

The acute toxicity study of VM hydro alcoholic extract (VMHA) was evaluated in rats using Organization for Economic Co-operation and Development guideline 420. The extract was administered orally at dose of 2000 mg/kg. All the clinical signs and mortality were observed for 1 h after dosing, periodically during first 24 h and daily for a total of 14 days.

Induction of Osteoarthritis and Drug Administration

Albinio Wistar rats were divided into six groups consisting nine animals per group where control group received only vehicle and disease control group with other treatment group received 4% papain and 0.03 M cysteine prepared in distilled water. 0.2 ml of papain and 0.1 ml of cysteine were administered intra-articularly three times (1, 4 and 7 day) to induce OA. VMHA in three different doses (100 mg/kg, 200 mg/kg and 400 mg/kg) and celecoxib (30 mg/kg) were administered orally once in a day after last papain injection. Three animals from each group were sacrificed at the last day (28th) of study.

Anti-Osteoarthritic Activity Evaluation

Knee Joint Diameter

Inflammation of the knee joint of animals was measured using digital Vernier Caliper. To measure the knee joint diameter, hairs from the knee joints of the animals were clipped for the proper palpitation of knee, followed by disinfection with sterilium before measuring joint diameter. Knee joint diameter of all animals was measured before the papain injection and once in a week after last papain injection.

Wire Hang Test

Wire hang test was performed using wire screen to evaluate the physical disability (pain) of animals in OA. The square area of the screen was built in a way to restrict animal in 50 cm × 50 cm section of the screen. The animal was placed on the screen and screen was waved gently in the air three times to force the rat to grip the wires. The screen was then immediately turned upside down above a large rodent housing cage. Latency to falling was recorded before papain injection and once in a week. Rats that fell in <10 s were given a second trial while cut off time was set to 10 min. Animals were observed once in a week for a change in hanging time.

Grip Strength Meter

In order to assess neuromuscular pain grip strength meter was used to sense the peak amount of force applied while grasping specially designed pull bar assemblies. Hind limbs of animals were handled in such a way that only its hindlimbs will be free for holding the pull bar. They were pulled slowly away from the gauge until the paws get released. The movement was in a horizontal direction, and the force applied by rats was measured before papain injection and each sacrifice. Force applied by each animal of all groups was measured and observed.

Activity Meter

The activity meter consists of a pair of sensors along with an emitter, which sends out pulsed infrared beams, and a detector;
which picks up these optical pulses. The animal was placed in the cage for 10 min and total distance travelled by an animal was measured. Baseline reading were taken before papain injection. Distance travelled by each animal of all groups was measured and observed before each sacrifice.

**Histopathology**

Gross observation of knee joints of animals was done before sacrifice. Animals were sacrificed using CO₂ overdose method and right knee of each animal from all groups was collected. The specimens were fixed in 10% neutral buffered formaldehyde at room temperature for 1 week. Decalcification was performed in Goodings and Stewart reagent for 7 days. Then the tissue blocks were dehydrated in a graded series of ethanol, cleared in xylene and embedded in paraffin. Sections of 5–7 μm were cut with a sliding microtome and stained with Harris hematoxylin and eosin. The morphological differences at cellular levels observed in the slides were evaluated according modified Mankin scoring system.

**Statistical Analysis**

Results were expressed as mean ± standard error of the mean. Data were analyzed as a completely randomized design using one-way analysis of variance using a graph pad prism (version 5.0) (GraphPad Software, Inc, La Jolla, USA). Any significant difference between means was assessed by the Dunnet’s post-hoc test. 95% level of significance (P < 0.05) was used for the statistical analysis.

**Results**

**Characterization of Plant Extract**

The total ash and acid soluble ash for VMHA were found to be 6.2% and 0.28%, respectively. Limits of detection of the VMHA were found to be 9.50%. The water soluble extractives and alcohol soluble extractives for VMHA were found to be 17.82% and 6.9%. Moreover, the preliminary phytochemical screening of VMHA showed the presence of carbohydrates, proteins, glycosides, flavonoids, and phenolic compounds.

**Acute Toxicity Study**

Animals observed for 14 days after single dosing of 2000 mg/kg of VM showed neither any abnormal clinical signs nor mortality. As a result of this study 2000 mg/kg dose was considered safe.

**Anti-Osteoarthritic Activity Evaluation**

**Knee Joint Diameter**

The effect of oral administration of VMHA on knee joint diameter is shown in Table 1. Intra-articular injection of papain produced increase in knee joint diameter on day 7 which continued up to day 24. With VMHA treatment (200 and 400 mg/kg) there was significant (P < 0.05) decrease in joint diameter compared to disease control group on day 14 and day 24 which was found to be more effective than celecoxib treatment. The knee joint diameter of animals treated with VMHA was comparable to normal control group on day 24.

**Wire Hang Test**

The changes in hanging time observed after oral administration of VMHA and celecoxib are shown in Table 2. Intra-articular injection with papain in animals decreased the hanging time on day 7, which continued up to day 24. Oral administration of VMHA at dose of 100 mg/kg produced significant (P < 0.05) increase in hanging time on day 14 and day 24 which was almost comparable with standard treatment.

**Grip Strength**

The effect of oral administration of VMHA on the grip strength at different time periods is shown in Table 3. Injection

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**Table 1:**

| Group | Day | Control | Positive control | Standard control | VM (100 mg/kg) | VM (200 mg/kg) | VM (400 mg/kg) |
|-------|-----|---------|------------------|------------------|---------------|---------------|---------------|
| 0     | 8.11±0.11 | 8.08±0.10 | 8.05±0.07 | 7.89±0.12 | 7.94±0.09 | 8.04±0.14 |
| 7     | 8.28±0.05 | 9.62±0.15* | 9.54±0.20* | 9.59±0.09* | 9.60±0.27* | 9.34±0.24* |
| 14    | 7.78±0.09 | 9.17±0.12* | 8.76±0.26* | 8.66±0.2* | 8.52±0.13* | 8.30±0.12* |
| 24    | 8.03±0.28 | 9.07±0.15 | 8.50±0.16 | 8.80±0.25 | 8.42±0.25 | 8.03±0.07* |

*Significantly different at P<0.05 versus control and *significantly different at P<0.05 versus positive control. Each values represent mean±SEM (n=9 in each group) for the number of animals utilized during experiment. SEM=Standard error of mean, VM=Vigna mungo

**Table 2:**

| Group | Day | Control | Positive control | Standard control | VM (100 mg/kg) | VM (200 mg/kg) | VM (400 mg/kg) |
|-------|-----|---------|------------------|------------------|---------------|---------------|---------------|
| 0     | 45±2.64 | 49±2.46 | 49±2.46 | 49±5.55 | 55±2.90 | 42±3.02 |
| 7     | 65±3.70 | 38±2.32* | 46±3.73* | 36±3.02* | 36±1.45* | 35±1.84* |
| 14    | 56±3.35 | 35±1.95* | 57±4.14* | 64±5.94* | 47±3.58 | 44±2.02 |
| 24    | 80±2.88 | 39±3.21 | 63±3.33* | 63±3.35* | 54±7.75 | 54±4.78 |

*Significantly different at P<0.05 versus control and *significantly different at P<0.05 versus positive control. Each values represent mean±SEM (n=9 in each group) for the number of animals utilized during experiment. SEM=Standard error of mean, VM=Vigna mungo
with papain produced decrease in grip strength on day 14 which lasted till day 24. Oral treatment of VMHA at 100, 200 and 400 mg/kg dose exhibited significant improvement on day 14 and 24, which was found more effective than celecoxib treatment.

**Activity Meter**

Locomotion activity was decreased in all papain treated animals on day 14 and day 24. Oral treatment of VMHA at the dose of 100 and 400 mg/kg dose significantly improved locomotion activity on day 14 and 24 which was more effective than standard treatment celecoxib [Table 4].

**Histopathology**

Table 5 demonstrates the effect of oral administration of VMHA on histological changes at cellular level of the knee joint.

Intra-articular injection of papain produced mild muscle degeneration, slight erosion of cartilage and mild fibrillation [Figure 1] in joint tissues of positive control group compared with normal control group. Oral treatment of VMHA at dose of 100 mg/kg showed sub-maximal effect with mild muscle degeneration, slight erosion of cartilage and fibrillation [Figure 1] however other two doses (200 and 400 mg/kg) showed prominent effect with only slight muscle degeneration and erosion of cartilage [Figure 1]. The effect of VMHA at dose of 200 and 400 mg/kg was found almost comparable with standard treatment.

**Discussion**

Intra-articular injection of papain significantly increased knee joint diameter and thus produced swelling on knees. Inflammation results from the release of a number of chemical mediators including cytokines and leukotriene.[22] Papain - a proteolytic enzyme when administered to cartilage cause its breakdown, producing inflammatory cytokines (TNF-α and IL-β).[13] Along with these inflammatory cytokines, there are also elevated levels MMP with increased free radicle generation.[22] These all together shows arthritic like symptoms on intra-articular injection of papain in rats.[13]

Oral administration of VMHA at the dose of 200 and 400 mg/kg significantly attenuated the inflammation. Phytochemical screening revealed the presence of poly-phenolic and flavonoids in the VMHA. As already known, these poly-phenolic and flavonoids inhibit cyclooxygenase pathway

**Table 3:**

Effect of VMHA and celecoxib on grip strength

| Group          | Day | Control   | Positive control | Standard control | VM (100 mg/kg) | VM (200 mg/kg) | VM (400 mg/kg) |
|----------------|-----|-----------|------------------|------------------|----------------|----------------|----------------|
|                | 0   | 160±2.23  | 151±3.64         | 153±2.01         | 159±2.36       | 156±2.27       | 168±3.40       |
|                | 7   | 138±1.34  | 127±2.57*        | 127±2.74*        | 127±1.76*      | 129±0.84*      | 128±2.10*      |
|                | 14  | 150±3.79  | 132±4.14*        | 138±2.10         | 171±4.85*      | 146±6.00*      | 153±3.38*      |
|                | 24  | 176±5.48  | 124±3.08         | 145±5.11         | 175±1.54       | 169±15.14      | 147±27.67      |

*Significantly different at P<0.05 versus control and *significantly different at P<0.05 versus positive control. Each values represent mean±SEM (n=9 in each group) for the number of animals utilized during experiment. SEM=Standard error of mean, VM=Vigna mungo

**Table 4:**

Effect of VMHA and celecoxib on locomotion activity

| Group          | Day | Control   | Positive control | Standard control | VM (100 mg/kg) | VM (200 mg/kg) | VM (400 mg/kg) |
|----------------|-----|-----------|------------------|------------------|----------------|----------------|----------------|
|                | 0   | 2437±86.9 | 2134±68.8        | 2257±69.3        | 2159±83.3      | 2427±80.8      | 2425±67.7      |
|                | 7   | 2362±80.6 | 1903±77.6*       | 1806±71.7*       | 1919±73.2*     | 2064±80.6*     | 1638±47.6*     |
|                | 14  | 2437±71.3 | 1985±71.7*       | 1873±63.1*       | 2390±41.9*     | 2184±72.1      | 2176±70.2      |
|                | 24  | 2382±50.4 | 1845±71.6        | 1909±27.0        | 2105±71.6*     | 2148±76.8      | 2346±58.7*     |

*Significantly different at P<0.05 versus control and *significantly different at P<0.05 versus positive control. Each values represent mean±SEM for the number of animals utilized during experiment (n=9 in each group). SEM=Standard error of mean, VM=Vigna mungo

**Table 5:**

Effect of VMHA on histopathological findings

| Groups         | Parameter          | Control | Positive control | Standard control | VM (100 mg/kg) | VM (200 mg/kg) | VM (400 mg/kg) |
|----------------|--------------------|---------|------------------|------------------|----------------|----------------|----------------|
|                | Muscle degeneration| N       | ++               | +                | ++             | +              | +              |
|                | Fibrillation       | N       | ++               | +                | ++             | +              | +              |
|                | Erosion of cartilage| N     | ++               | +                | +              | +              | +              |
|                | Chondrocyte death  | N       | +                | +                | +              | +              | +              |

N=Normal, ++=Slight, +++=Mild, VM=Vigna mungo
and capable of neutralizing free radicle and thus reduce inflammation. Thus, presence of flavonoids could be attributed to anti-inflammatory activity of VMHA in rats.

In addition, the MMPs are the enzymes which upon release dissolves the cartilage thus ultimately enhance the inflammatory process by releasing more cytokines and MMPs, thus the reduction in knee joint diameter could also be attributed to MMPs inhibition.

Osteoarthritis is a major cause of disability throughout the world. It causes pain due to inflamed knee joints.

There is substantial indication displaying that certain cytokines are responsible for not only for initiating, but also in persisting the pathological pain through direct activation of nociceptive sensory neurons. Moreover, inflammatory cytokines also cause nerve-injury or inflammation-induced central sensitization, and are accountable for the development of contralateral hyperalgesia. Intra-articular injection caused intense pain in knee joints which significantly reduced grip strength, hanging time and locomotion activity of animals due to severe pain. Oral administration of VMHA at the dose level of 100 mg/kg significantly improved grip strength, hanging time and locomotion activity. As already discussed earlier that flavonoids present in VMHA may inhibit peripheral inflammatory pathways and thus compromise the release of cytokines as well as prostaglandins, reducing inflammatory pain in animals. There is also the possibility of contribution of central pathways through involvement of opioid receptors. However, further studies are required for exploring the involvement of flavonoids in producing analgesia through central pathways of analgesia.

Osteoarthritis is the condition which involves progressive degeneration of articular cartilage, synovitis, formation of osteophyte, increased fibrillation due to increased denaturation and loss of collagen fibers. These deteriorating changes at cellular level in cartilage are may be outcomes of elevated release of cytokines and MMPs. It is also known that increase free radicle generation may cause cell (chondrocytes) death.

More precisely, these focal changes are mainly due to increased production of IL-β and TNF-α with reactive oxygen species. VMHA exhibited its effect in dose-dependent manner. Histopathological observation suggests oral administration of VMHA was found to be effective in improving muscle degeneration, fibrillation, erosion of cartilage and chondrocyte death due to the inhibition potential of flavonoids on cytokines and MMPs.

Conclusion

The data obtained from the present study demonstrated the favorable anti-inflammatory activity and analgesic activity aiding to its anti-osteoarthritic activity and thus confirms its traditional use in rheumatic conditions and bone disorders. Further experimentations are required to investigate the possible mechanism of action by which these chemical constituents show its action.

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