INTRODUCTION

*Vatsanabha* or *Mahavisha*, Aconitum ferox is a species of monk’s hood from the family Ranunculaceae is a deciduous perennial with tall and erect stems crowned by racemes of large eye catching blue, purple, white zygomorphic flowers with numerous stamens. [1] *Vatsanabha* is also identified as *Vatsanaga, Ksweda, Visa* and *Amrita.* [2] As the synonym reveals, this toxic plant can also be useful as *Amrita*. The root of this plant is extremely poisonous but useful in the treatment of various diseases such as fever, rheumatoid arthritis, sciatica, hypertension and also act as a *Rasayana* after *Shodhana* [3–5]. Classically, *Charaka* has categorized *Vatsanabha* under *Sthavara Visha*, *Susruta* under *Kanda Visha* and *Kaiyadeva nighantu* under *Visha Varga* [6]. The objective of this study is to review the state of knowledge of the drug as such along with its toxic effects on various systems, its shodhana and medico-legal importance.

**Toxic constituents:**

The tuber of *Vatsanabha* contains 0.4–0.8% diterpene alkaloids and the concentration of aconite in the fresh plant is between 0.3% and 2.0% in tubers and 0.2% and 1.2% in the leaves. The highest concentration of aconite is found in the winter. The major alkaloids are aconitine, pseudoaconitine, bikhaconitine, diacetyl pseudoaconitine, aconine, picro-aconine, veratry pseudoaconitine, chamaconitine, veratryl gama aconine, and di-Ac-Y-aconitine. [7].

**Necessity of Shodhana:**

The present review is designed to extensively discuss and understand the plant, its toxic effects, management and the medico-legal aspects involved.
Impure or impurely purified *Vatsnabha* if administered in any form will cause *Daha* (acute burning sensation) all over the body. It may also cause *Murcha* (Syncope), *Hrutrodana* (cardiac arrest) which may usually lead to *Mrutyu* (death) of patient. Use of *Vatsnabha* in higher dose may also lead to toxicity and death. All parts of the plants are poisonous. Tuber is chiefly used as a poison. Leaves handled or rubbed on the skin, produces tingling and numbness. The odor of the plant has a narcotic effect; its pollen causes pain and swelling in the eyes. It imparts a sensation of tingling & numbness to the tongue, lips & mouth when chewed.\[^9\]

**Toxic symptoms**^10-12^  
Susruta enumerates the toxic symptoms as *Greeva stambha* (neck stiffness) and *peeta vinmoorta netrata* (yellowish discoloration of feces, urine and eyes).  
*Rasa Vagbhata* enumerated *Ashta vega* (eight stages) of poisoning as:

<table>
<thead>
<tr>
<th>Stage</th>
<th>Symptom</th>
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<tbody>
<tr>
<td>1st</td>
<td><em>Twak vikara</em> (skin changes)</td>
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<tr>
<td>2nd</td>
<td><em>Vepathu</em> (tremors)</td>
</tr>
<tr>
<td>3rd</td>
<td><em>Daaha</em> (burning all over the body)</td>
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<tr>
<td>4th</td>
<td><em>Vikrtavastha</em> (deformities)</td>
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<tr>
<td>5th</td>
<td><em>Phenodgama</em> (froth from mouth)</td>
</tr>
<tr>
<td>6th</td>
<td><em>Skanda bhanga</em> (drooping of shoulders)</td>
</tr>
<tr>
<td>7th</td>
<td><em>Jadata</em> (comatose)</td>
</tr>
<tr>
<td>8th</td>
<td><em>Marana</em> (death)</td>
</tr>
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</table>

Externally, when applied to mucous membranes or to the skin for any length of time,aconite first stimulates and then depresses the ends of the sensory nerves, producing respectively tingling, numbness, and local anesthesia.

It acts as both Neurotoxic and Cardiotoxic. Generally symptoms occur immediately or within minutes of ingestion. Patient experiences Bitter-sweet taste in the mouth followed by severe burning and tingling sensation of lips, tongue, mouth & throat, which is again followed by numbness & anesthesia, Nausea, salivation, difficulty in swallowing, Pain in abdomen, vomiting and later tingling of the whole body.

CNS Symptoms include Vertigo, Restlessness, Difficulty in speech; Great prostration, Pain and weakness of muscles associated with twitching and spasm, Contraction and dilatation of pupil[finally dilated] and Diplopia or impaired vision.

CVS Symptoms include slow, feeble and irregular pulse, fall in BP, Respiration which at first is rapid and then slow, labored and shallow. Skin may become cold and damp and temperature will be usually sub normal.

**Mode of Action**^13^:

Its major alkaloid aconitine has the chemical formula C$_{34}$H$_{47}$NO$_{11}$, and is soluble in chloroform or benzene, slightly in alcohol or ether, and only very slightly in water. Aconitine can interact with the voltage-dependent sodium-ion channels, which are proteins in the cell membranes of excitable tissues, such as cardiac and skeletal muscles and neurons. These proteins are highly selective for sodium-ions. They open very fast to depolarize the cell membrane potential, causing the upstroke of an action potential.
Normally, the sodium channels close very rapidly, but the depolarization of the membrane potential causes the opening (activation) of potassium channels and potassium efflux, which results in repolarization of the membrane potential.

In short Aconitine increases the permeability of excitable membrane for sodium ions and prolong the sodium influx during the action potential as a consequence sensible nerve ending and motor endplates are first activated but later blocked.

**Fatal dose**[^14]:
- Indian aconite root: 1.3-2gm
- Tincture: 5ml
- Liniment: 1ml
- Pure aconitine: 2mg

<table>
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<tr>
<th>Fatal Period[^15]:</th>
</tr>
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<tr>
<td>Usually 1-5 hrs</td>
</tr>
<tr>
<td>Doses below Lethal Dose: produce alarming symptoms immediately</td>
</tr>
<tr>
<td>Non-fatal cases: numbness &amp; tingling sensation persist</td>
</tr>
</tbody>
</table>

**Recovery Period**[^16]:
Recovery time is dependent on amount of intoxication. Mildly intoxicated patients may take 1–2 days while patients with cardiovascular complications may take 7–9 days to recover.

**Cause of death**[^17]:
Death may be due to Respiratory failure or Ventricular fibrillation.

**Shodhana:**

<table>
<thead>
<tr>
<th>Sl No.</th>
<th>Process</th>
<th>Media</th>
<th>Duration</th>
</tr>
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<tbody>
<tr>
<td>01</td>
<td>Swedana[^18,19]</td>
<td>Aja dugdha</td>
<td>3 days</td>
</tr>
<tr>
<td>02</td>
<td>Sthapana Aatapa shoshana[^22,21,22,23]</td>
<td>Gomootra</td>
<td>3 days</td>
</tr>
<tr>
<td>03</td>
<td>Swedana[^24]</td>
<td>Go dugdha</td>
<td>3 days</td>
</tr>
<tr>
<td>04</td>
<td>Sthapana Aatapa shoshana Bhavana[^25]</td>
<td>Gomootra Tankana</td>
<td>3 days</td>
</tr>
</tbody>
</table>

**Changes occurring during Shodhana:**
Though treatment with cow urine, cow milk, or cow dung is the traditional method of *Shodhana*, it may not be feasible or acceptable to all. After *Shodhana* process, the total alkaloid content decreases[^26] but the contents of less toxic substances such as aconine, hypoaconine, and benzylhypoaconine increases[^27,28] possibly due to conversion of the toxic aconitine into aconine or hydrolysis of the alkaloids to their respective amino alcohols after *Shodhana* process[^29,30].

It has been reported that *Gomootra* converts Aconite to a compound with cardiac stimulant property, whereas, raw Aconite showed cardiac depressant properties.[^31,32,33,34] *Shodhana* by both *Gomootra* and *Godugdha* makes Aconite devoid of cardiac and neuromuscular toxic effects without affecting its antipyretic activity.[^35] Soaking and boiling during processing or decoction preparation will hydrolyze aconite alkaloids into less toxic and non-toxic derivatives.

The results of the toxicity study suggested that after Ayurvedic *Shodhana* process, TLC studies have shown that pseudoaconitine and aconitine were converted into far less toxic substances veratroyl pseudoaconine and benzoylaconine respectively.

**Management:**
According to *Rasa Vagbhata*, Upto 5th *vayga* management is possible. First *vamana*...
(vomiting) shall be induced using Ajak-sheera followed by Lepa, Kwatha or Anjana of Vishaghna gana.

If toxic symptoms is appears due to over dose of formulation contains aconitum or usage of improper / without purification of Vatsanabha in that condition Tankana bhasma along with Ghee is given\[36\]. Combination of honey, ghee and bark of Arjun (Terminalia arjuna) is also used as antidote. Whenever Vatsanabha is used in any formulation, Tankana bhasma (borax calx) is also used in the formulation to counteract the toxic effect of Vatsnabha and hence there will be no toxic effects\[37\].

Use of Emetics or Gastric lavage with a solution containing animal charcoal, tannic acid or milk is advised. Immediate attention should be given to vital organs. There should be close monitoring of BP & cardiac rhythm. Maintenance of body heat is essential and use of inotropic therapy in cases of hypotension. In cases of bradycardia, administration of 1mg atropine is indicated. For cardiac arrhythmias, 50ml of 0.1 percent Novocain given intravenously, in a slow manner is useful. Oxygen & artificial respiration may be resorted to, if necessary. Glucose saline may be administered intravenously to combat collapse \[38\].

**Post Mortem Appearance** \[39\]:
Post-mortem appearances are not characteristic. Fragments of root may be found in the stomach contents. Mucus membrane of stomach and small intestines may be congested and inflamed. The bronchial tree shows frothy mucus.

**Medico-legal Importance** \[40\]:
Accidental poisoning is not rare as aconite root is often consumed after mistaking it for horse radish root. Tincture has been swallowed in overdoses and liniment is taken internally by mistake. Inhalation of the dust while powdering of root has caused toxic symptoms.

Used of aconite as suicidal and homicidal poisoning is common in India, where aconite is given along with betel leaf to mask its taste. Aconitine added in Indian liquors to increase the intoxicating effect, and causes poisonous symptoms followed occasionally by death. In rare cases, aconite root has been used as an abortifacient. Root of aconite is occasionally used as cattle poison. Roots of aconite are used as an arrow poison.

**Identification** \[41\]:
Elimination of aconitine is mainly in the urine and is best tested by acidifying it with diluted acetic acid. Traces have also been found in the saliva, sweat and bile.

**Vatsanabha: An Ideal Homicide** \[42\]:
Aconitine is extremely unstable and is destroyed by putrefactive process. Hence it is often difficult to detect it after its death. It is also decomposed by an alkali. Wood ashes, which are often added to a vomit, destroy aconite owing to the presence of an alkali.

**CONCLUSION:**

*Vatsnabha* (Aconitum ferox) is categorized under the *sthavara visha* (plant origin) while Aconite is a Greek word which means arrow referring to the then use of the plant. As our Acharyas have clearly mentioned, even a strong poison can become an excellent medicine if administered properly; on the other hand even the most useful medicine act as a poison if not handled correctly. *Vatsnabha* is highly toxic in nature even then it is used as a medicine after purification and in therapeutic dose. Active constituents of many plant drugs may exert severe toxic effect at high concentrations. The purification processes are basically intended to reduce the toxicity level to a
body sustainable limit and to reduce the toxic constituents to some extent or by potentiating their chemical transformation to nontoxic or relatively less toxic substances by enhancing their biological efficacy.

Pharmacologically, it improves digestion, relieves coldness, nutritive. It is used in the treatment of vision problems, night blindness, eyes infections, inflammation, otitis and headache. It is useful in treatment of sciatica and backache. It is also used as antidote for poisoning due to jangama visha (animal origin poison).

Due to some properties like Ashukaritwa, Ushna, Teekshna vish dravya get spread rapidly in the body. So for the quick action of medicines many Ayurvedic formulations contain these vishadravyas like Vatsanabha as their ingredient. By utilizing these properties of vishadravyas medicines can be made more effective. In spite of being a Mahavisha, it is a basic ingredient of various ayurvedic formulations which thus proves its synonym Amrita.

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