Hemlock (Conium Maculatum) Poisoning In A Child

Bir Çocuk Hastada Baldıran Otu (Conium Maculatum) Zehirlenmesi

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SUMMARY
Poison hemlock (Conium maculatum) is a plant that is poisonous for humans and animals. Accidental ingestion of the plant may result in central nervous system depression, respiratory failure, acute rhabdomyolysis, acute renal failure and even death. The main treatment of hemlock poisoning is supportive care. The case of a 6-year-old girl who was admitted to the emergency department with complaints of burning sensation in mouth, hypersalivation, tremor in hands and ataxia after ingestion of poison hemlock is presented here with clinical and laboratory features. In this case, we aim to report that accidental ingestion of plants resembling vegetables that are consumed daily can lead to serious complications and even death.

Key words: Conium maculatum; childhood; poisoning; hemlock.

ÖZET

Anahtar sözcükler: Conium maculatum; çocukluk çağı; zehirlenme.

Introduction
Poisonings are commonly seen during childhood and are associated with a high mortality rate. The vast majority of poisonings in children are due to taking medications; however, they may rarely be caused by petroleum products, caustic substances, and weeds. Conium maculatum, known popularly as hemlock in our country, is a poisonous plant belonging to the family Apiaceae, which grows in grasslands in damp places. Conium maculatum is poisonous not only for humans, but also for cattle, sheep, goats, pigs, rabbits, deer, poultry, and insects.[1] Poisoning in humans can be caused by ingestion of the plant, but may also be caused by ingestion of meat from the aforementioned animals.[2] Piperidine alkaloids, such as coniine and γ-coniceine, contained within the plant play an important role in the development of poisoning.[2,3] Coniine is an alkaloid which is neurotoxic for humans and which causes death via respiratory paralysis. Famous philosopher Socrates died from hemlock poisoning in 399 BC.[3]

We present the case of a six-year-old female patient who was referred to our clinic with symptoms of hypersalivation and...
ataxia due to ingestion of hemlock. The hemlock was mis-
taken for parsley and consumed. The aim of this report was
to emphasize that accidental ingestion of some plants in our
environment which closely resemble edible vegetables may
produce serious signs of poisoning and even lead to death.

Case Report
In this report, we present the case of a six-year-old female
patient. She ingested a weed found in a garden assuming it
was parsley. Two hours later, she was referred to our emer-
gency service due to complaints of burning in the mouth, in-
creased salivation, trembling hands, and walking imbalance.
Gastric lavage was performed, and activated carbon was
administered in our emergency department. She was then
hospitalized in the intensive care unit. The plant, which the
family brought with her, was sent to the Faculty of Science,
Department of Biology and was identified as *Conium macu-
latum*, known popularly as hemlock. In her first physical ex-
amination, she was fully conscious but agitated, and coop-
eration could not be established. The increased amount of
saliva was too much for her to swallow. The patient’s vitals
were as follows: arterial blood pressure, 105/65 mmHg; heart
rate, 125/min; pulse, filiform; and peripheral oxygen satu-
ration (SpO₂), 97%. Her neurological examination revealed a
tremor in both hands, loss of motor function in both lower
limbs (4/5), limitations in the movement of tongue, ataxia,
dilation in both pupils, and decreased response to light.

The patient was monitored, intravenous (IV) crystalloid infu-
sion was started, and emergent laboratory tests were stud-
ied immediately. Complete blood count, arterial blood gas-
es, and biochemical tests did not reveal any pathologies. The
results of the first coagulation tests were: PTT, 20 seconds;
INR, 2; and APTT, 50 seconds. Control coagulation test results
were as follows: PTT, 25 seconds; INR, 2.3; and APTT, 54 sec-
onds. Thus, a unit of fresh frozen plasma was administered
to the patient. After transfusion, her coagulation test results
were normal. Her ECG revealed no abnormalities except si-
nus tachycardia. Secretion crackles were observed during
auscultation in both lungs, however, her chest radiography
revealed no abnormalities. Repeated laboratory tests dem-
onstrated no deterioration, and the patient did not develop
respiratory distress. The tremor in her hands recovered after
6-8 hours, and the increased salivation diminished after 12-
14 hours. Limitations in tongue movement disappeared on
the second day, and she started talking again. Difficulty in
walking and ataxia disappeared. The symptoms which she
presented at admission disappeared completely, her physi-
cal examination findings became normal, and she was dis-
charged from the hospital three days after admission with
full recovery.

Discussion
*Conium maculatum* poisoning occurs due to some of its pi-
peridine alkaloid contents which have nicotinic effects, such
as coniceine, conine, N-methyl coniine, conhydrine, and
pseudoconhydrine. While every part of the plant is toxic, the
highest alkaloid concentration exists in the seeds.[4] Ataxia
and headache are the symptoms observed in the early stage
of poisoning. Increased salivation, tachycardia, and pupil-
lar dilation develop due to the effects of the plant on the
autonomic ganglia. Muscle weakness or paralysis, bradycar-
dia, and central nervous system depression may develop in
some of the patients due to increased cholinergic stimuli.[5]
Rhabdomyolysis and acute renal failure have also been re-
ported in some cases as a consequence of *Conium macula-
tum* poisoning.[4] Each of these piperidine alkaloids is a kind
of peripheral neurotoxin; the neurotoxins show curare-like
effects in neuromuscular junctions and create nicotinic ef-
fects in the autonomic ganglia.[7]

A previous report of two patients who were poisoned by
accidental ingestion of the plant demonstrated that early
stage symptoms recovered with symptomatic treatment.[8]
However, two additional studies conducted in our country
observed that respiratory depression developed imme-
diately after early stage symptoms, and patients required me-
chanical ventilation.[9,10] In the present study, nicotinic sym-
ptoms, such as hand tremors, difficulty in walking, ataxia, and
excessive salivation were observed on admission. However, our patient did not develop acute kidney failure, rhabdomyolysis, or respiratory failure. Our patient exhibited prolonged coagulation tests, which have not been previously reported in the literature. This abnormality, observed in the early stage, may have developed due to the deterioration of factors in the intrinsic and extrinsic coagulation cascade or in the common pathway coagulation cascade. We did not have the opportunity to measure the patient’s blood factor levels and, therefore, could not reach a definitive conclusion. However, careful examination of coagulation tests in similar future cases may be useful.

No specific antidote exists for the treatment of this toxin. Therefore, symptomatic treatment is the main treatment. Basic life support should be provided, followed by the administration of gastric lavage and activated carbon to reduce absorption. Fluid supplements should be administered to the patient via an intravenous access. Benzodiazepines or barbiturates should be used in the treatment if convulsions develop. Fluid supplementation and urine alkalinization should be provided in case of rhabdomyolysis or myoglobinuria. Hemodialysis and hemoperfusion are not reported to be useful in the treatment of rhabdomyolysis.[11] Close electrolyte and biochemical examinations and monitoring should be performed during intensive care unit follow-up. Death occurs mainly due to respiratory failure and paralysis of the respiratory muscles, thus continuous respiration should be ensured and mechanical ventilation support should be provided if necessary. Some previous studies in the literature have reported that children and adult patients required mechanical ventilation support after hemlock poisoning.[9,10,12] Our patient recovered completely without sequelae after receiving only supportive treatment.

In conclusion, the aim of the present report was to emphasize that accidental ingestion of some plants in our environment which closely resemble edible vegetables may produce serious signs of poisoning and even lead to death.

Conflict of Interest

The authors declare that there is no potential conflicts of interest.

References