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| Logo AGES | |
| Equine encephalomyelitides | |
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| 05.02.2025 13:02 Uhr | |

**Equine
encephalomyelitides**

**Western
Equine
Encephalomyelitis
(WEE),
Venezuelan
Equine
Encephalomyelitis
(VEE),
Japanese
Encephalitis
(JE)**

Last
change:
10.10.2023

**Profile**

Equine
encephalomyelitis
is
an
inflammation
of
the
brain
and
spinal
cord
in
hoofed
animals
caused
by
viruses.
Horses
(including
humans)
can
contract
the
infection
but
do
not
pass
on
the
virus.
Infections
in
horses
are
often
asymptomatic,
but
occasionally
severe
disease,
sometimes
fatal,
can
occur.

**Occurrence**

Eastern,
Western,
and
Venezuelan
encephalomyelitis
(EEE,
WEE,
VEE)
occur
in
the
Americas.
Japanese
encephalitis
is
widespread
in
Asia.
The
occurrence
of
the
above-mentioned
diseases
is
not
currently
expected
in
Europe.

**Host
animals**

Wild
birds;
lagomorphs,
rats
(western
encephalomyelitis);
rodents
(Venezuelan
encephalomyelitis);
pigs
(Japanese
encephalitis).

**Infection
route**

Predominantly
by
blood-sucking
insects

**Incubation
time**

In
Venezuelan
encephalomyelitis
1-5
days,
in
the
other
equine
encephalomyelitides
5-15
days.

**Symptoms**

In
horses,
the
infections
are
usually
clinically
inconspicuous;
in
a
small
percentage,
fever,
loss
of
appetite
and
depression
may
occur,
but
usually
go
unnoticed.
In
individual
animals,
non-specific
mild
to
severe
neurological
symptoms
may
occur,
mostly
when
the
fever
has
already
subsided.
Severe
courses
can
lead
to
death.

**Therapy**

A
specific
therapy
is
not
possible.
Diseased
horses
can
only
be
treated
symptomatically.

**Prevention**

The
best
prevention
of
infection
is
to
protect
horses
from
mosquitoes/insects.
There
are
approved
vaccines
in
the
affected
regions.

**Situation
in
Austria**

All
clinical
forms
of
equine
encephalomyelitis
are
notifiable
in
Austria.
So
far,
none
of
the
listed
diseases
has
been
detected
in
equidae
or
other
animals
in
Austria.

**Technical
information**

The
American
equine
encephalomyelitides
(Western,
Eastern,
Venezuelan
equine
encephalomyelitis)
are
caused
by
so-called
arboviruses
(insect-borne
-
arthropod-borne
-
viruses).
All
three
viruses
belong
to
the
family
Togaviridae
and
the
genus
Alphavirus.
They
are
all
enveloped,
spherical,
single-stranded
RNA
viruses
with
positive
polarity
and
a
diameter
of
70
nm.
**Eastern
Equine
Encephalomyelitis
(EEE)
-
EEE
virus**:
originally
4
subtypes,
lineages
2-4
have
recently
been
renamed
Madariaga
virus
**Western
Equine
Encephalomyelitis
(WEE)
-
WEE
virus**:
no
other
subtypes
**Venezuelan
Equine
Encephalomyelitis
(VEE)
-
VEE
virus**:
6
different
subtypes,
these
are
divided
into
enzootic
and
epizootic
strains
(the
latter
arise
from
mutations)
Transmission
is
predominantly
by
blood-sucking
insects
(mosquitoes)
with
horses,
humans
and
other
mammals
usually
acting
as
false
hosts.
Other
transmission
routes
have
also
been
observed
through
mites,
lice,
fleas
and
predatory
bugs.
However,
direct
contact
can
also
lead
to
transmission
(e.g.
in
the
case
of
EEE
by
birds
or
in
the
case
of
VEE
by
humans,
horses
and
rodents).
Oral
routes
of
transmission
are
known
in
birds
(e.g.,
cannibalism,
feather
pecking)
and
aerogenic
routes
of
transmission
from
VEE
to
humans
(e.g.,
during
mucking
out
of
laboratory
rodent
cages)
are
also
known.
The
pathogenesis
of
American
encephalomyelitis
is
similar.

Initial
viral
replication
occurs
after
the
mosquito
bite
in
regional
blood
and
lymph
vessels
and
lymph
nodes.
After
the
first
viremic
phase,
a
second
viral
replication
occurs
in
the
lymph
nodes
and
muscles,
after
which
the
second
viremic
phase
causes
hematogenous
infection
of
the
CNS.
In
the
CNS,
viral
replication
occurs
in
neurons,
glial
cells
as
well
as
endothelial
cells.
The
virus
triggers
immunopathological
reactions,
whereby
the
synthesis
of
inflammatory
mediators
is
stimulated
in
the
astrocytes
and
programmed
cell
death
(apoptosis)
increasingly
occurs
in
the
affected
neurons.

Accordingly,
the
CNS
is
dominated
by
the
pathomorphological
picture
of
a
non-pathological
encephalomyelitis,
characterized
by
perivascular
infiltrates
(lymphocytes,
histiocytes,
in
the
early
stage
also
neutrophilic
granulocytes),
neuronal
degeneration
and
neuronophagy.

**Japanese
encephalitis
(JE)**
is
also
caused
by
an
arbovirus.
The
causative
agent,
Japanese
Encephalitis
Virus
(JEV)
belongs
to
the
genus
Flavivirus
of
the
family
Flaviviridae.
JEV
is
an
enveloped,
spherical,
single-stranded
RNA
virus
with
positive
polarity
and
a
diameter
of
50
nm
and
belongs
to
the
so-called
Japanese
Encephalitis-Serocomplex-Group
with
other
known
members
in
this
complex,
such
as
the
West
Nile
Virus
(WNV),
the
St.
Louis
Encephalitis
Virus
(SLEV)
and
others.
The
disease
is
widespread
in
the
Asian
region,
but
has
also
recently
been
detected
in
western
India
and
the
Western
Pacific
region
(Indonesian
Islands,
Papua
New
Guinea,
Northern
Australia).

Transmission
is
by
mosquitoes,
with
wild
birds
(waders)
and
pigs
as
reservoirs.
Humans,
horses
and
dogs
are
considered
false
hosts
that
do
not
spread
the
virus.
Human
cases
often
correlate
with
areas
of
intensive
pig
farming,
irrigation
schemes
as
well
as
rice
fields.
The
pathogenesis
as
well
as
the
pathomorphologic
findings
in
horses
largely
resemble
those
of
American
equine
encephalomyelitis.
The
zoonotic
potential
of
all
four
diseases
cannot
be
neglected.

**Symptomatology**

The
clinical
symptoms
are
by
no
means
specific
and
vary
individually.
In
addition
to
clinically
inapparent
forms,
fever,
anorexia,
and
depression
may
occur
but
usually
go
unnoticed.
In
a
few
animals,
various
neurological
disorders
such
as
hypersensitivity,
involuntary
muscle
movements,
running
in
circles,
head
pressing,
difficulty
swallowing,
central
blindness,
ataxias,
paresis
to
paralysis/convulsions,
recumbency
with
paddling
movements
and
occasionally
pruritus
may
occur.
Death
or
euthanasia
due
to
severe
progression
occurs
after
2-4
days.
If
the
animals
survive,
they
usually
show
mental
deficits.
Diarrhoea,
constipation
and
weight
loss
are
also
occasionally
observed.

Sows
affected
by
JE
may
have
abortions
or
stillbirths,
but
do
not
have
specific
findings.
Fetuses
may
be
mummified,
and
the
weak
or
stillborn
piglets
may
show
hydrocephalus
(watery
head)
and
encephalitis
(inflammation
of
the
brain).
In
boars,
testicular
and
epididymitis
may
occasionally
be
observed.

**Diagnostic**

Fatal
progressive
encephalomyelitis
cases
are
investigated
neuropathologically
as
well
as
molecularly.
In
a
suspected
case,
appropriate
tissue
samples
(CNS
from
dead
or
euthanized
animals
and
CSF
and
blood
from
febrile
animals
with
clinical
signs
as
recommended
by
WOAH)
are
sent
to
the
European
Reference
Laboratory
(ANSES)
for
confirmation
and
further
characterization.
Suitable
organs
for
direct
detection
are
considered
to
be
the
brain
and
spinal
cord,
if
applicable.
Ideally,
the
horse's
head
and
cerebrospinal
fluid,
if
applicable,
should
be
sent
to
the
National
Reference
Laboratory.

If
JEV
is
suspected
in
pigs,
aborted
material
should
be
sent.

**Contact**

**National
reference
laboratory
for
equine
encephalomyelitis**

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