AGRI MAGAZINE
(International E-Magazine for Agricultural Articles)
Volume: 03, Issue: 10 (October, 2026)
Available online at http://www.agrimagazine.in
©Agri Magazine, ISSN: 3048-8656

# Managing Ranikhet Disease Outbreaks in Poultry Farms

*A. Pavan Kumar[1], P. Venkat Sai Kumar[2] and B. Ramya[3]
[1]Ph.D Scholar, Department of Veterinary Pathology, College of Veterinary Science, Rajendranagar, Telangana, India
[2]PG Scholar, Department of Poultry Science, College of Veterinary Science, Rajendranagar, Telangana, India
[3]Associate Professor, Department of Veterinary Pathology, College of Veterinary Science, Rajendranagar, Telangana, India
*Corresponding Author's email: pavankumar.a465@gmail.com

Ranikhet disease (RD), known elsewhere as Newcastle disease, remains one of the most destructive viral infections of chickens in India. Virulent strains can kill most of an unvaccinated flock within days. Outbreaks still occur in vaccinated flocks, owing partly to genotype XIII viruses that differ from older vaccine strains and partly to lapses in vaccine handling and biosecurity. As no drug cures RD, management rests on early detection, laboratory confirmation, supportive care, control of secondary infections and preventing spread.

**Keywords:** Ranikhet disease; Newcastle disease; poultry; vaccination; biosecurity; outbreak management

## Introduction

Sudden deaths, birds circling with twisted necks and a sharp fall in egg production are familiar to field veterinarians in India. This is Ranikhet disease (RD), called Newcastle disease (ND) elsewhere, first described in 1926 in Java and near Newcastle-upon-Tyne, England [1], and named in India after the hill town of Ranikhet. Virulent strains can still kill almost every bird in an unprotected flock, and because ND is listed by the World Organisation for Animal Health (WOAH), outbreaks can also restrict trade [2]. Most frustrating for farmers, outbreaks continue in vaccinated flocks.

## The Virus in Brief

RD is caused by virulent strains of avian orthoavulavirus 1 (species *Orthoavulavirus javaense*, family *Paramyxoviridae*), an enveloped RNA virus. Its haemagglutinin-neuraminidase (HN) protein attaches the virus to cells and agglutinates chicken red blood cells, the basis of haemagglutination (HA) and haemagglutination-inhibition (HI) tests; the fusion (F) protein allows cell entry [3]. In virulent strains the F protein cleavage site carries several basic amino acids, so enzymes in almost all tissues can activate it and the virus spreads throughout the body. WOAH defines virulent ND virus by this cleavage-site pattern or an intracerebral pathogenicity index (ICPI) of 0.7 or more [2]. Strains are described as lentogenic (mild), mesogenic (moderate) or velogenic (severe); velogenic strains are viscerotropic (intestinal haemorrhages) or neurotropic (respiratory and nervous disease) [3]. There is one serotype but many genotypes [4], and virulent genotype XIII viruses have repeatedly been found in Indian outbreaks, including in vaccinated flocks [5,6].

## How the Disease Enters and Spreads

Birds are infected by inhaling virus in droplets and dust or by swallowing contaminated feed, water and droppings [1,3]. Between farms the virus travels mainly with people and objects:

market birds, returned unsold birds, crates, egg trays, feed bags, vehicles, and the shoes, clothes and hands of workers, traders and vaccinators. Incubation is usually 2–6 days, and birds shed virus before they look ill [1], so infection has often spread through the shed before the first deaths. Missed or poorly given vaccinations, a broken cold chain, immunosuppression (infectious bursal disease, Marek's disease, chicken infectious anaemia, mycotoxins), multi-age sites and an uncontrolled farm gate all make outbreaks more likely.

## Recognising Ranikhet Disease on the Farm

### *Clinical signs*

Signs depend on the strain, age and, above all, immunity [3]. The viscerotropic form causes sudden deaths, dullness, ruffled feathers, greenish watery diarrhoea and swollen heads and wattles, with mortality approaching 100% in non-immune flocks. The neurotropic form causes coughing and gasping, then tremors, twisted neck (torticollis), circling and paralysis. Layers show a sharp fall in production, with pale, thin-shelled, misshapen or shell-less eggs. Partly vaccinated flocks may show only mild signs that are easily misdiagnosed.

### *Post-mortem and histopathology*

Typical viscerotropic lesions are pin-point haemorrhages on the tips of the proventricular glands, haemorrhagic or necrotic ulcers over intestinal lymphoid patches, swollen dark caecal tonsils and a congested trachea [3,6]; layers may have flaccid ovarian follicles and free yolk. Highly pathogenic avian influenza produces almost identical lesions, while neurotropic and vaccinated cases may show very few, so a clean carcass does not rule out RD. Microscopically, there is lymphoid necrosis, vascular damage and, in nervous cases, non-suppurative encephalitis [3]; even less virulent strains can inflame the air sacs and heart [7]. Histopathology supports but cannot confirm the diagnosis.

## Confirming the Diagnosis

Laboratory confirmation is essential [8]. Send oropharyngeal or tracheal and cloacal swabs from sick birds, and trachea, lungs, spleen, intestine, caecal tonsils and brain from fresh carcasses, chilled and without delay. Real-time RT-PCR detects the virus within hours, and F-gene testing or cleavage-site sequencing shows whether the strain is virulent. Virus can also be isolated in 9–11-day-old embryonated eggs and identified by HA and HI tests. HI and ELISA mainly assess vaccine response; a sharp titre rise between paired samples is suspicious. Rule out avian influenza, infectious bronchitis, infectious laryngotracheitis, fowl cholera and egg drop syndrome, and, with nervous signs, Marek's disease and vitamin E deficiency.

## Managing an Outbreak

There is no specific treatment for RD, and antiviral drugs are not advised [9]; farmers should be wary of products sold as cures. The aims are to limit losses, prevent secondary infections and stop spread.

### *Immediate actions*

1. Isolate the affected shed; no birds, eggs, manure, equipment or staff should move from it to other sheds.
2. Inform the veterinary officer or State Animal Husbandry Department, as ND is notifiable, and send samples to a laboratory.
3. Remove dead birds several times daily for deep burial with lime or incineration. Never dump carcasses or sell sick birds.
4. Record daily mortality, feed and water intake and egg production.

### *Supportive care and secondary infections*

Birds still eating and drinking can recover with good care. Provide clean, cool water with electrolytes and water-soluble vitamins (A, D3, E, C) for general support, ensure good ventilation and avoid unnecessary handling. Secondary *E. coli* and *Mycoplasma* infections are common, and antimicrobials may reduce losses, mainly with less virulent strains [9]. Use them only on veterinary prescription, never routinely, and observe withdrawal periods.

***Vaccination during an outbreak***

In endemic areas, unaffected sheds on the same premises are sometimes revaccinated with a live lentogenic vaccine early in an outbreak. Birds already incubating infection will not benefit, and careless vaccination can spread virus, so healthy sheds should be done first by separate teams and equipment.

***Cleaning and restocking***

After depopulation, remove litter and manure for burial or composting, wash surfaces with detergent and apply an approved disinfectant as per the label. The enveloped ND virus is readily killed once organic matter is removed. Leave the shed empty and dry, then restock with a single age group on an all-in, all-out basis.

## Prevention: The Long-Term Strategy

***Vaccination programme***

Vaccination is the mainstay of RD control in India. Properly given, current vaccines prevent disease and deaths but not infection [9,10]. Live lentogenic vaccines (Hitchner B1/F1, LaSota) are given by eye or nasal drop, drinking water or spray, and mass application needs about 85% of birds to receive vaccine [9]. Live mesogenic R2B (Mukteswar) vaccine is injected into older birds but is too reactive for chicks [10]. Inactivated oil-emulsion vaccines give primed breeders and layers high, long-lasting immunity, and recombinant vector vaccines carrying the F gene can be given in ovo or at hatch but take 3–4 weeks to protect [9]. Thermostable vaccines such as I-2 suit village chickens without a cold chain [11].

**Table 1. An example of a Ranikhet vaccination programme commonly followed in India**

| Age of birds | Vaccine | Route |
|---|---|---|
| 5–7 days | Live F1 (B1) or LaSota | Eye or nasal drop |
| 21–28 days | Live LaSota booster | Drinking water or eye drop |
| 8–10 weeks (layers, breeders, backyard birds) | Live R2B (Mukteswar) or inactivated vaccine | Subcutaneous or intramuscular injection |
| 16–18 weeks (layers, breeders) | Inactivated oil-emulsion vaccine, with or without a live booster | Intramuscular or subcutaneous injection |
| During lay (every 6–8 weeks, as advised) | Live LaSota booster | Drinking water or spray |

*Note:* This is only an example; broilers usually receive just the first two doses. The local veterinarian should set the schedule based on maternal antibodies, local disease situation and manufacturer's instructions.

***Vaccine handling and monitoring***

Store vaccines at 2–8 °C and carry them in an ice box, out of the sun. For water vaccination, use chlorine-free water with skimmed milk powder (about 2 g per litre), withhold water for 1–2 hours so birds drink all vaccine within two hours, and vaccinate only healthy birds. Periodic HI testing of layers and breeders shows whether vaccination has worked and when boosters are due.

***Biosecurity***

Fence the farm, keep a visitors' register, use separate footwear and clothing for each shed, keep foot dips fresh and disinfect vehicles, crates and egg trays before entry. Exclude pigeons, wild birds and backyard poultry, buy chicks from reliable hatcheries, isolate new birds for two to three weeks, control immunosuppressive diseases and avoid mouldy feed.

## Why Do Vaccinated Flocks Still Suffer Outbreaks?

Vaccines do not fully prevent infection, so vaccinated birds with low antibody levels can still be infected and shed virus [10]. Most Indian vaccines are based on older genotype II strains, whereas recent outbreaks involve genotype XIII viruses, and investigations in vaccinated flocks have partly attributed failures to this mismatch [5,6]. The commonest and most preventable causes, however, are errors in vaccine handling and administration, and

immunosuppression. Field-strain-matched vaccines have reduced virus shedding experimentally, but their field performance is still being evaluated [10]. Herbal products and immunomodulators lack evidence to replace vaccination and biosecurity.

## Public Health Note

In humans the virus may cause mild, self-limiting conjunctivitis, mostly in heavily exposed laboratory workers or vaccinators [1]. Properly cooked chicken and eggs pose no known risk, but eye protection and hand washing during mass vaccination are sensible.

## Conclusion

Ranikhet disease cannot be treated, but it can be prevented and contained. Farms with the lowest losses vaccinate correctly, protect the cold chain, control immunosuppressive diseases and enforce strict biosecurity. Treat any sudden rise in deaths, nervous signs or fall in egg production as possible RD until the laboratory rules it out. In an outbreak, isolate the shed, report it, support the birds and disinfect thoroughly before restocking.

## Conflict of Interest

The authors declare no conflict of interest.

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