

Cytomegalovirus infection in a pig in South Africa

M G Collett^{a†} and D C Roberts^{b‡}

ABSTRACT

An 8-week-old piglet with dyspnoea, bilateral mucopurulent nasal discharge and mouth breathing was euthanased and a necropsy was performed. Apart from histological evidence of diffuse rhinitis, large intranuclear inclusion bodies, pathognomonic for porcine cytomegalovirus infection, were detected within mucous glands on the nasal turbinates. This is the first such case to be diagnosed in South Africa.

Key words: cytomegalovirus, inclusion body rhinitis, porcine, suid herpesvirus 2.

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INTRODUCTION

Porcine cytomegalovirus (PCMV) (suid herpesvirus 2) infection, also known as inclusion body rhinitis, was first described by Done in the UK in 1955³. The causative virus, a beta herpesvirus, is distinct from 2 other herpesviruses affecting pigs: suid herpesvirus 1 (the cause of Aujeszky's disease) and bovine herpesvirus 1 (the cause of infectious bovine rhinotracheitis), both of which are alpha herpesviruses^{9,12}.

The disease causes sneezing, snuffling, nasal discharge, dyspnoea, anorexia, runting and occasional death in suckling (1–5 weeks old) and recently weaned piglets^{3,12}. The nasal discharge is initially seromucinous but later a yellowish-white purulent exudate becomes caked around the nostrils^{1,3}. Although the morbidity is high, mortality is generally low^{1,3}. Older pigs, especially boars and non-pregnant sows, generally do not show clinical signs. Susceptible pregnant sows, however, can be severely affected, with the main signs being coughing, anorexia, pyrexia, and dysgalactia, as well as abortion, stillbirths, mummification, embryonic death and infertility (the so-called SMEDI syndrome), and sudden deaths in neonatal piglets^{1,7,12,13,15}.

Experimental infection of neonatal piglets causes lethargy, anorexia, pallor, subcutaneous oedema of the throat and

hocks, dyspnoea and death⁷. Experimentally infected sows, on the other hand, show anorexia and lethargy and nasal and/or cervical viral excretion has been demonstrated⁷.

Gross lesions at necropsy include the presence of pus in the nasal cavity and sometimes the sinuses, pulmonary oedema, enlarged bronchial lymph nodes, transudates in body cavities, anaemia, and petechiation of the subcapsular surface of the kidneys, the heart and elsewhere^{2,6,11,12}. The outstanding microscopic lesion is the presence of large, basophilic, intranuclear cytomegalic inclusions in the tubulo-alveolar mucous acini and ducts, and, to a lesser extent, in the surface epithelium of the nasal turbinates, together with the infiltration of lymphocytes, plasma cells and neutrophils into the lamina propria¹³. Nasal inclusions have been described in a 1-day-old piglet¹³. Apart from the characteristic nasal lesions, there have been a number of reports detailing generalised lesions with intranuclear inclusions in cytomegalic renal tubular and interstitial cells, pulmonary alveolar macrophages, tracheal and small intestinal epithelium, oesophageal mucous glands, and salivary, Harderian and lachrymal glandular epithelium, as well as adrenal, testis and epididymis^{2,6,12,16}. Smaller herpetic inclusions can be seen in endothelial cells and macrophages in the choroid plexus, glomeruli, liver, spleen, lymph nodes and bone marrow^{2,6,10–12,16}. The lungs reveal an interstitial pneumonia¹². Disseminated foci of necrosis may be seen in parenchymatous organs, while foci of non-suppurative encephalitis have also been described^{2,6,16}. Foci of extramedullary haematopoiesis may be seen in

the liver and spleen^{2,16}. Complications include sinusitis, otitis media, pneumonia and atrophic rhinitis^{1,2,16}.

Lesions in aborted fetuses include ascites, multiple necrotic foci in the liver, and renal cortical and epicardial petechiae¹⁵. In affected embryos and fetuses, virus localises in the leptomeninges and inclusions may be detected in endothelial cells, hepatocytes, and splenic and pulmonary macrophages^{7,8,15}. The placenta is not a primary site of viral replication⁸.

The disease is believed to have a worldwide distribution⁵. As far as the authors are aware, however, no cases have ever been confirmed previously in South Africa.

CASE HISTORY

The affected pig came from a 40 Large White sow unit that was situated not far from Onderstepoort, near Pretoria, Gauteng. The pigs were housed in poorly fenced pens consisting of an open space and a wood and corrugated iron shack enclosure. The feed consisted of hotel swill that was fed in galvanised iron troughs and old motor car tyres cut circumferentially. Water was supplied in similar containers. The management could be described as rudimentary. A new boar had been introduced during February.

In July, 10 piglets were farrowed by a sow in her third parity. Two piglets died 2 days later and the other 8 were weaned 6 weeks later. Two weeks after weaning, a further piglet died. Unfortunately, none of the carcasses were submitted for necropsy. According to the owner, however, the last animal had a purulent nasal discharge and showed respiratory distress before death. A week later, the owner brought in a male littermate to the Outpatients Clinic of the Veterinary Academic Hospital, Faculty of Veterinary Science, University of Pretoria at Onderstepoort. The owner's chief complaints were small litter sizes, neonatal deaths (piglets dying within 3 days of birth) and that some 7–8-week-old piglets developed purulent nasal discharges and died. The clinical signs shown by this pig included prostration, dyspnoea, bilateral mucopurulent nasal discharge and mouth breathing.

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The animal was euthanased by barbiturate overdose for a necropsy and diagnostic investigation.

Pathology

Grossly, lesions were limited to the respiratory tract. A whitish, tenacious mucopurulent exudate was present at both nostrils and within the nasal cavity. The nasal mucosa was slightly redder than normal and the turbinate structures appeared normal and symmetrical on cross-section of the maxilla at the level of the third premolar teeth. In the lungs, a few small, greyish-white areas of increased consistency were present in the borders of the cranioventral lobes. Unfortunately, only nasal turbinate tissue was sampled for further investigation.

Histopathological examination of nasal turbinate tissue revealed squamous metaplasia of the surface epithelium and severe mucosal thickening due to a diffuse infiltration of predominantly lymphocytes and plasma cells into the lamina propria. Pus was present in some glandular lumens. In many areas, clusters of mucous glandular epithelial cells showed cytomegaly and contained conspicuously large, irregular, basophilic, nuclear inclusion bodies (visible at low magnification) (Fig. 1). Some of the affected nuclei had lost the distinctiveness of the nuclear membrane, giving the inclusion and nucleus a smudged appearance (Fig. 2). Affected cells often contained large cytoplasmic vacuoles and some had sloughed.

Transmission electron microscopy of affected cells revealed that inclusions contained many viral nucleocapsids (Fig. 3) and, in the cytoplasm, enveloped capsids (virions), which occurred singly or within discrete cytoplasmic vacuoles (Fig. 4).

A nasal swab yielded *Corynebacterium kutscheri*.

DISCUSSION

From a diagnostic viewpoint, the presence of the characteristic inclusion bodies in nasal mucosal epithelial cells is pathognomonic for PCMV infection³. In live animals, inclusion bodies can be detected in nasal scrapings prepared for cytology⁴. A method for rapid diagnosis, using electron microscopy and a negative staining technique, has been described¹⁴. Confirmation by virus isolation is problematic, however, as specialised tissue culture systems are required⁵.

In piglets younger than 3 weeks, congenital or neonatal PCMV infection can cause high mortality, and the clinical signs and generalised lesions (oedema, petechiae and anaemia) are probably

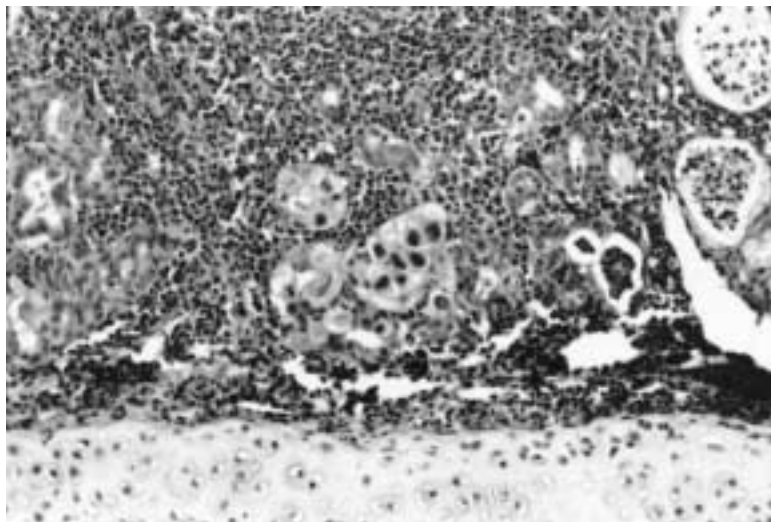


Fig. 1: Conspicuous intranuclear inclusions are present in the glands in the centre. Note the pronounced non-suppurative inflammatory infiltrate in the surrounding lamina propria. Turbinate cartilage is present at the bottom. HE, $\times 50$.

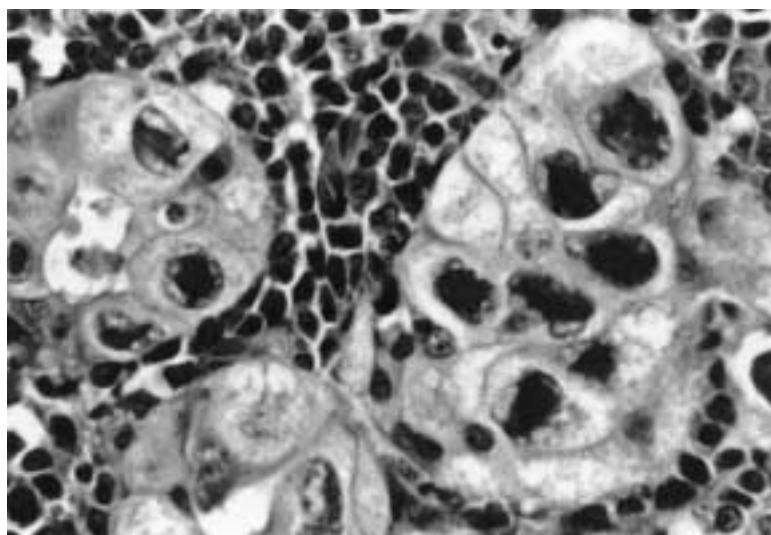


Fig. 2: Three mucous acini with cytomegalic epithelium and prominent nuclear inclusions especially in the larger one on the right. HE, $\times 200$.

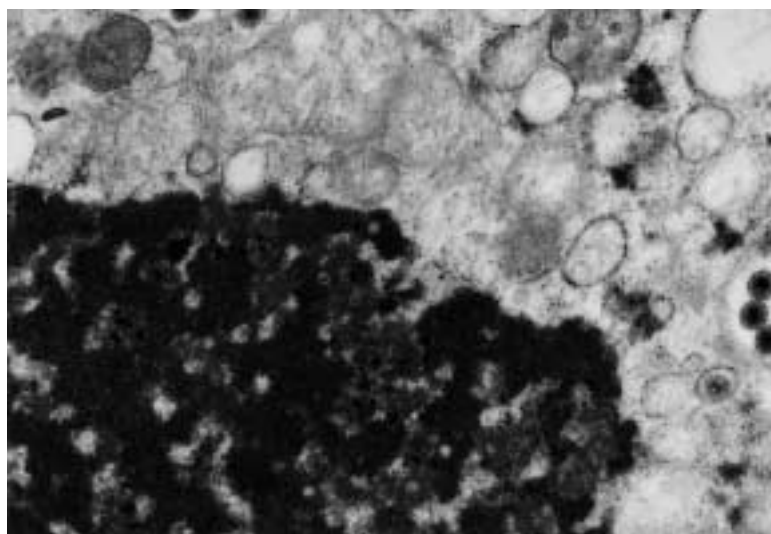


Fig. 3: Electron micrograph of nuclear inclusion (lower left) showing indistinct mass that contained numerous nucleocapsids. Note the three enveloped capsids in the cytoplasmic vacuole on the right. $\times 28\ 300$.

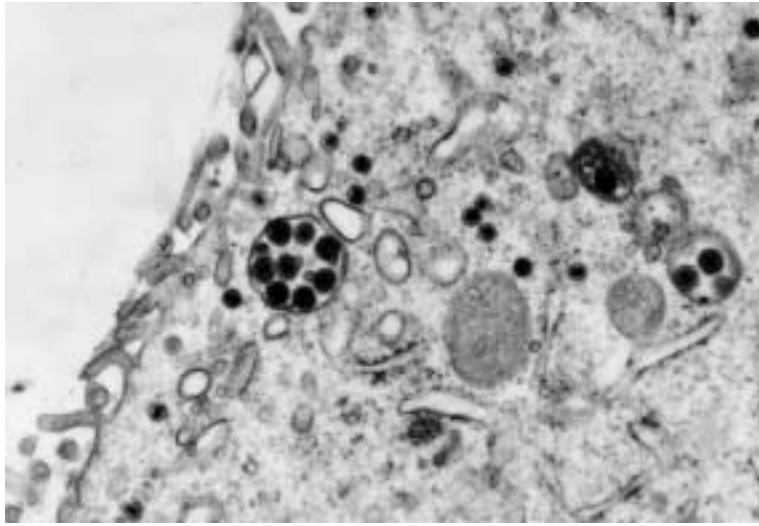


Fig. 4: **Electron micrograph showing a group of enveloped capsids in cytoplasmic vacuoles on the left (larger) and right (smaller), respectively. x28 300.**

correlated to viral replication in vascular endothelium and the mononuclear phagocytic system, particularly lung macrophages. In piglets older than 3 weeks, the distribution of virus and inclusions is a reflection of epithelial cell infection (especially nasal and renal); consequently clinical signs and lesions are much milder^{6,12}. Expression and severity of clinical signs is dependent on husbandry and hygiene. As is the case with many other herpesviruses, latent infections occur; cells of the mononuclear phagocytic system are likely to harbour the virus for indefinite periods^{5,11}.

Although further diagnostic investigation was precluded for financial reasons, it is interesting to speculate that the problems that the owner had identified (small litter sizes, neonatal deaths, rhinitis and deaths in the early post-weaning period) could all have been due to PCMV infection. It is likely that the boar introduced in February was the source of the infection, transmitted either by nasal shedding of virus or *via* the urine at coitus.

It is possible that the prevalence of PCMV infection in South Africa (and else-

where) is higher than expected. Swine practitioners are alerted to the possibility of PCMV infection being present in piggeries and being a factor to consider in the SMEDI (stillbirths, mummification, embryonic death and infertility) syndrome, as well as in cases of neonatal death, runtting, respiratory disease and interstitial nephritis in young pigs¹⁰.

Other causes of foetal death include classical swine fever virus, the viruses associated with SMEDI and suid herpesvirus 1 (Aujeszky's disease)⁷.

The viral ultrastructure resembled that previously described^{11,12,14,16}.

Corynebacterium kutscheri is normally isolated from rodents and is of low pathogenicity (M Henton, Onderstepoort Veterinary Institute, pers. comm.).

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