Parvovirus B_{19} Caused Fetal Death : A Case Report in Thailand

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Abstract

This was a case of an intrauterine parvoviral B_{19} infection resulting in hydrops fetalis and enlarged placenta. Histologically, the virus was found to be in nucleated red cells of the fetus which was confirmed by electron microscopy. Careful placental examination at the gross and microscopic levels yielded the correct diagnosis.

Key word : Hydrops fetalis, Placenta Infection, Parvovirus B_{19}

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Most causes of dead fetus *in utero* are uncertain[1]. In Thailand death *in utero* due to Bart's hemoglobin disease is about 9.5 per cent of fetal deaths[1]. Intrauterine viral infection is rarely found by autopsy due to autolysis of organs and lack of viral serologic study.

This is a case of hydrops fetalis caused by intrauterine parvoviral infection, demonstrated by histology of placenta and electron microscopy.

CASE REPORT

A 36 week female dead fetus *in utero* from a 35 year old Thai, G_1P_0 mother was reported. In this first pregnancy, she had 6 visits at the antenatal clinical without any complications. The serologic studies such as : VDRL, Anti-HIV, HBsAg. Ab and rubella titer revealed negative results. At about 36 weeks of gestation, the mother complained that the fetal movement seemed to be inactive and she developed labour pain. Ultrasonogram revealed a single fetus in oblique lie, breech presentation, but without fetal heart sound.

Induction of labour was performed and a dead female fetus *in utero* was delivered by breech extraction, with birth weight of 2,880 g, and a 1,000 g placenta.

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Her chromosome study revealed 46XX with normal general appearance.

At autopsy, the infant was found to be a normally developed female with anasarca. Marceration was generalized to indicate an intrauterine death. All internal organs, including the central nervous system, showed normal appearance regarding their locations, measurements, shapes and sizes.

The lungs were unaerated. The other organs showed variable degree of autolysis from mild to moderate degree. (Fig. 1)

The submitted placenta showed complete features with intact maternal surface, measuring 22x21x3 cm and weighing 1,000 g. It was red-tan, bulky with friable parenchyma. The fetal membrane was mildly edematous, white and cloudy in appearance. The umbilical cord was 33 cm long and 1.5 cm in diameter. It eccentrically inserted at 6 cm. from the margin. The cut surface showed three normal vessels. (Fig. 2)

Light microscopic findings :

All internal organs were autolysed to a variable degree and without inflammatory reaction.

Microscopically, the placenta showed focal chronic villitis and features compatible with hydrops fetalis. Some areas showed focal villous necrosis. Viral inclusions were in the nucleated red blood cells of the villous capillaries. They contained central clear to eosinophilic bodies with peripheral chromatin condensation. The inclusion was typical for parvovirus inclusion. (Fig. 3)

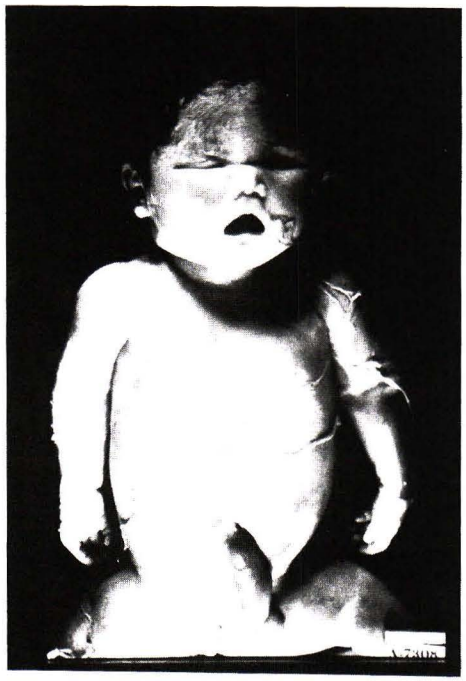


Fig. 1. Showing anasarca with marcerated skin of the fetus.

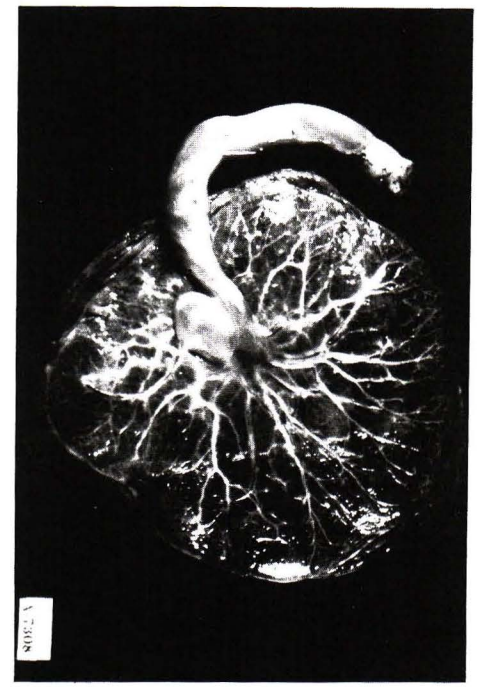


Fig. 2. Showing enlarged placenta but intact membrane and placental tissue with edematous umbilical cord.

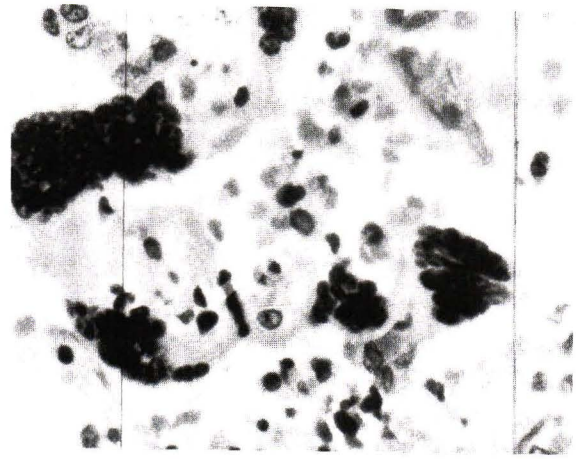
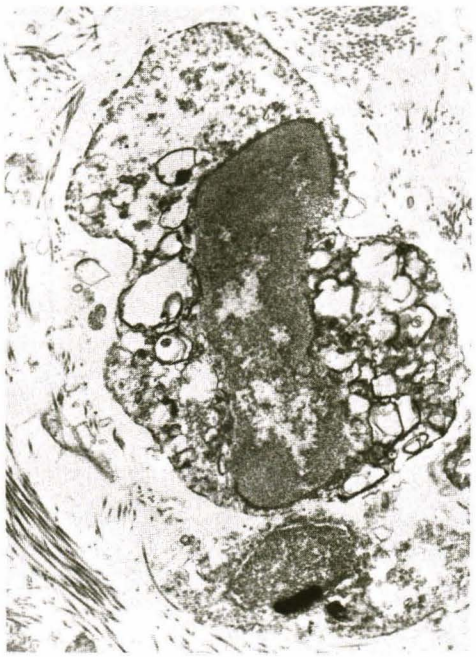


Fig. 3. Section of the placenta reveals intranuclear inclusion (arrow) in the nucleated red cell.



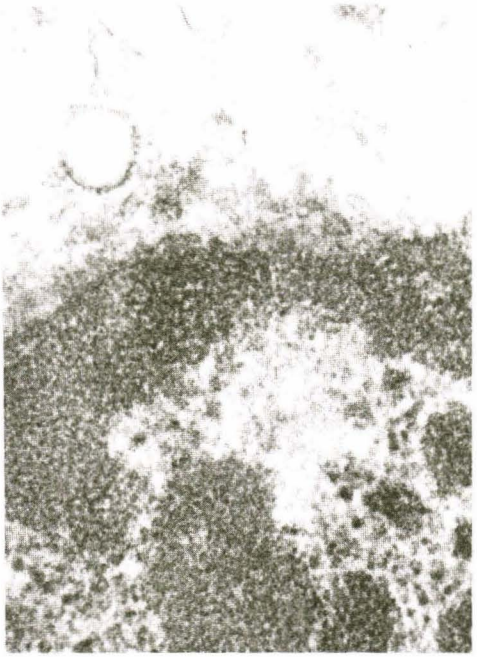


Fig. 4, 5. Showing intranuclear inclusion of non-envelope particles measuring 18-20 nm in diameter compatible with parvovirus virions(6).

Electron microscopically, some of the nucleated red cells contained a large number of clusters of round, 18-20 nm in diameter of virions within their nuclei. They showed characteristics of parvovirus B_{19} virus. (Fig. 4 and 5)

The infected nucleated red blood cells were noted in the submitted placenta and liver tissues.

DISCUSSION

The pathological diagnosis was based on the histological and electron microscopic findings of parvovirus infection which was previously described. Many studies in pregnancies of women which serologically confirmed B_{19} infection have shown the risk of fetal infection in 33 per cent and fetal death was 9 per cent which most commonly occured in 10-12 weeks of gestation(3). Some 12 per cent had spontaneous abortions or fetal deaths occuring between 11-18 weeks of gestation(4).

In the present case, the placental weight was about one half of the birth weight. In this case, features of hydrops fetalis were the heavier weight of placenta and mild anasarca of the fetus.

Parvovirus B_{19} is the smallest DNA virus. It has non enveloped, single stranded DNA surrounded by two capsid proteins, VP-1, VP-2 which are species specific(4).

Major clinical manifestations of parvovirus B_{19} infection are;(4)

1. erythema infectiosum, affecting children more than adults,
2. arthropathy,
3. transient aplastic crisis showing chronic hemolytic anemia,
4. chronic anemia in immunocompromised patients including AIDS,
5. hydrops fetalis and fetal death in fetus (especially < 20 weeks),
6. neurologic, vasculitis, cardiac : rare reports in various hosts(3)

It is transmitted *via* respiratory secretion and person to person contact. Vertical transmission from mother to fetus can result in hydrops fetalis due to anemia in the fetus caused by short lived red blood cells, rapidly expanding red cell volume, and possibly ineffective immune response, which can lead to chronic anemia. Myocarditis in fetus

due to B_{19} contributes to heart failure. Vasculitis within the placental villi has also been reported[2].

The hydrops state and cause of death are most likely multifactorial[3].

Clinically, about 1 week after infection is a period of viremia. The virus is also detected in nasal washes and gargles. The first phase of the illness consists of mild nonspecific signs and symptoms, such as; fever, headache, myalgias and pruritis[3].

The second phase of the illness begins about 3 weeks after infection, manifested by an erythematous rash, joint involvement resulting in stiffness of the hands, knees and ankles with frank swelling of the fingers and knuckles that persists for several days after the rash has recovered. Self limited anemia usually occurs before the rash or artheropathy. Patients with increased red blood cells destruction, or with decreased RBC production can develop severe anemia.

The rash of erythema infectiosum and arthritis occurs at the same time as the development of B_{19} specific antibodies.

Definitive diagnosis relies on the detection of B_{19} IgM, IgG and viral DNA[3].

IgM is detectable within 3 days of onset of symptoms. The level of IgM begins decreasing at about 1 month and is often undetectable by 2-3 months, but IgG is still present several days after viremia and persists for years and perhaps for life[3].

Histologic findings in tissue of the fetus reveal intranuclear inclusions in erythroid precursors present in many organs including the liver. The intranuclear inclusions show the characteristic central clear to eosinophilic area with peripheral chromatin condensation. They are more frequently seen in liver of the fetus than in the placental vessels.

Electron microscopy can also be used to detect B_{19} virions in fetal tissue[2].

In general, the cause of death in almost 30 per cent of fetal deaths is not known,[1] due to many factors; severe autolysis, no placental examination etc. The placenta is the diary of intrauterine life that sometimes contained the information of the cause of fetal death. Careful placental examination, therefore, cannot be overemphasized.

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สาเหตุการตายของทารกในครรภ์เนื่องจากพาร์โวไวรัส บี 19 : รายงานผู้ป่วยรายแรกในประเทศไทย

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รายงานผู้ป่วยทารกเสียชีวิตในครรภ์ เนื่องจากการติดเชื้อพาร์โวไวรัส บี 19 ทารกมีลักษณะบวมน้ำทั้งตัว (ไฮดรอป ฟีทาลีส) และมีรกขนาดใหญ่ การตรวจด้วยกล้องจุลทรรศน์พบเชื้อไวรัสในนิวเคลียสของเม็ดเลือดแดงในรกและในตับของทารก

คำสำคัญ : บวมน้ำทั้งตัว, การอักเสบติดเชื้อในรก, พาร์โวไวรัส บี 19

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