SHORT COMMUNICATIONS

PEMPHIGUS VULGARIS AND PREGNANCY

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Pemphigus vulgaris during pregnancy is extremely rare; 2 such immunopathologically confirmed cases were treated by us. Case 1 delivered normal child; in case 2 a macerated foetus was born with extensive features of Neonatal pemphigus vulgaris survived for 10 days.

Key Words : Pemphigus vulgaris with pregnancy, Transplacental transmission, Neonatal pemphigus vulgaris

Introduction

Pemphigus vulgaris with pregnancy is extremely rare condition, due to improvement of recent immunopathological testing so far 16 cases have been published.[1,2] Foetal mortality is high due to transplacental transmission of pemphigus antibody from mother to child.[3,4] Risk of using teratogenic immuno suppressive drug may produce defect in the foetus.

Case I. A 35-year-old women, para 6 was reported with flaccid bullae all over the body specially on abdomen, thighs & back with a duration of 2 months. Blood bio chemistry was normal. High WBC count 26,000 cmm. with raised ESR 160 mm was the only positive findings. Bacteriogical culture was positive for staphylococcus aureus. In histopathology typical suprabasal bullae with acantholysis was seen. (Fig.1) Material send for direct immunoflorescence was positive; IgG level 1 : 540. Patient was treated with systemic steroid (120 mgm per day), azathioprine (150 mgm per day), weekly dosage of cyclophosphamide (200 mgm). Pregcolor test was positive. Patient delivered normal baby, his chord blood was examined for pemphigus antibody found negative.

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Case II. A 40-years-old women para 8 reported with history of P. vulgaris in late pregnancy about 8 months duration. Vagina

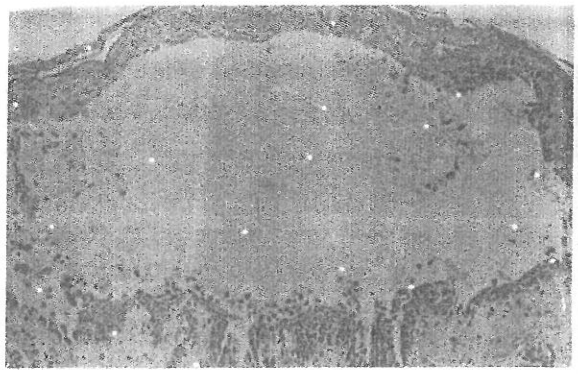

Fig. 1. Suprabasal bullae with cantholysis (H&E stain)

and oral mucous membranes were extensively involved with erosions & ulcers. No sign of toximea seen. Direct immunopathological testing showed florescence at the intracellular bridges (Fig.2) IgG level 1 : 640, with high WBC count 30,000 cmm with raised ESR 208 mm. Virological culture negative for cytomegalovirus. Bacteriological culture was positive for staphylococcus aureus. Treated with systemic steroid and immunosuppressive agents like previous case. Supportive therapy, blood transfusion, antibiotic cefatoxime was also given. At full term she delivered a

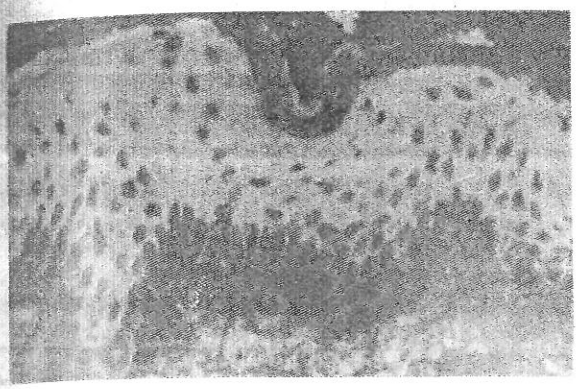

Fig. 2. Direct Immuno florescence- Intracellular bridges illuminated due to deposition of IgG

maccerated foetus which we apprehended from ultrasonographic foetal monitoring (Fig.3). Material send for direct immuno histopathological testing it was positive. IgG level chord blood was positive 1 : 6. The baby died after 10 days.

Comments

Role of circulating autoantibodies (mainly IgG) in the pathogenesis of P. vulgaris have long been debated.[5,6] Presence of circulating immunoglobuline is the cause of disease or secondary to some primary stimulus is difficult to ascertain. Autoantibody thus produced directed against attachment function between epidermal cells have been established. Passive transfer of P. vulgaris patient's serum to animals[7,8] and cultured human keratinocytes[9] & simultaneous development of acantholysis[10] attributed to role of immunoglobuline in the disease process. Co-relation of disease activity along with P. vulgaris antibody titre and amelioration of disease by plasma pheresis speak for role of IgG in such cases.[11] In our cases we were forced to use immuno-

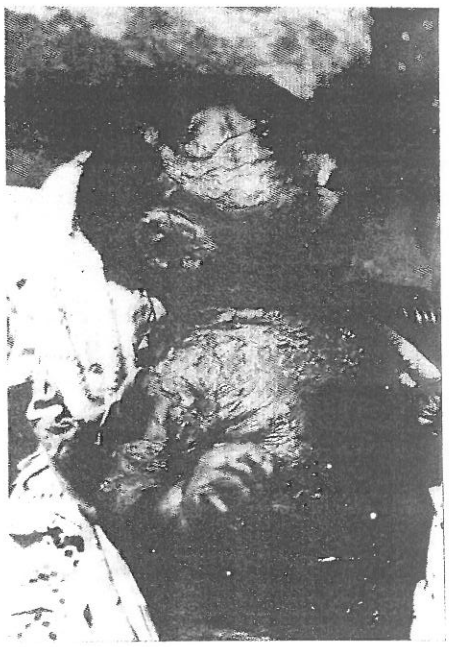

Fig. 3. Maccerateal foetus with extensive bullae, : Front view

suppressive agents because they didn't improve with simply systemic steroid and other supportive therapy.

IgG class antibody is capable to cross placental barrier to produce neonatal P. vulgaris or macerated foetus. Use of immunosuppresive agents may produce birth defect.

References

1. David Green, et al. Maternal pemphigus vulgaris with in vivo bound antibodies in the stillborn foetus. J Am Acad Dermatol 1992; 27 : 388.
2. Goldberg, et al. Pemphigus Vulgaris and pregnancy : Risk factors and recommendations. J Am Acad Dermatol 1993; 28 : 877.
3. Moncada B, Kettelsen S, Hernandez-Moctezumal JL, et al. Neonatal pemphigus vulgaris : role of passively transferred pemphigus antibodies. Br J Dermatol 1982; 106 : 465-8.

4. Wasserstrum N, Laros RK. Transplacental transmission of pemphigus. JAMA 1983; 249 : 1480-2.

5. Okano M, Takijiri C, Aoki T, et al. Pemphigus vulgaris associated with pregnancy : a case report from Japan. Acta Derm Venereol (Stockh) 1990; 70 : 517-9.

6. Terpstra H, de Jong MCJM, Klokke AH, et al. In vivo bound pemphigus antibodies in a stillborn infant. Arch Dermatol 1979; 115 : 316-9.

7. Chorzelski TP, Beutner EH, Jarzabek, M. Passive transfer of pemphigus in experimental animals. Int Arch Allergy App Immunol 1970; 39 : 106.

8. Holubar K, Chorzelski TP, Gauto M, et al. Studies in immunodermatology. III. Induct of intraepithelial lesions in monkeys intramucosal injections of pemphig antibodies. Int Arch Allergy Appl Immu 1973; 44 : 631-43.

9. Michel B, Ko CS. An organ culture model the study of pemphigus acantholysis. B Dermatol 1977; 96 : 295-302.

10. Schiltz JR, Michel B. Production of epiderm acantholysis in normal human skin in vitro the IgG fraction from pemphigus serum Invest Dermatol 1976; 67 : 254-60.

11. Fitzpatrick R, Newcomer V. The correlation disease activity and antibody titers in pemphig Arch Dermatol 1980; 116 : 285-90.