small facial mass. Clinical findings included a small triangular face with frontal bossing, blue sclerae, thin lips with down-turned corners, retromicrognathia, and limb asymmetry. Weight and height at admission were less than 5th percentile (Weight was 3.9 kg and size 56 cm); her head circumference grew normally along the 75th percentile.

Observation 2 A male Infant was hospitalised at the age of 1 year and 4 months. Physical examination showed growth deficiency: Weight: 7.1 Kg (<5th percentile), size: 72 cm, head circumference: 46 cm (25th percentile). The same facial features as the first case was found with body asymmetry, flat and narrow feet and cryptorchidism.

Conclusion The diagnosis of this syndrome is based on clinical findings. The treatment is purely symptomatic and growth hormone administration does not achieve the target size. The long-term prognosis, a part the growth, is good.

PO-0363 ASKIN TUMOUR:CASE REPORT

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Introduction Askin tumour is a primitive neuroectodermal tumour of the thoracopulmonary region. Pulmonary location is not common. We report a case of an Askin tumour in a child who presented with worsening breathlessness.

Observation A 9 years-old female child with no pathological history, was admitted with severe dyspnea up to one week. A Weight loss was reported. Physical examination showed silence in pulmonary auscultation of the left lung field. A chest X-ray found a left opacity occupying almost the entire of the left lung, mediastinal compression to the right side and nibbled aspect of the fourth rib. Chest Ultrasound showed pleural effusion with finely heterogeneous liquid. Chest CT showed a pulmonary solid tumour with aspect of massive necrosis of the left upper lobe with hemothorax, rib erosion in contact, without lymphadenopathy or vascular invasion or secondary lesion distance. A scano-guided biopsy was performed and immuno-histological examination found a small round cell tumour with morphological aspects favour the diagnosis of primitive peripheral neuroectodermal tumour corresponding to an Askin tumour. The child underwent an intensive chemotherapy and surgery.

Conclusion The management of Askin tumour is not well codified. The prognosis of these tumours remains very unfavourable because of their metastatic risk and potential local recurrence.

PO-0364 HISTOLOGICAL ASPECTS OF THE FETO-PLACENTAL INTERFACE IN THE PREGNANCY INDUCED HYPERTENSION

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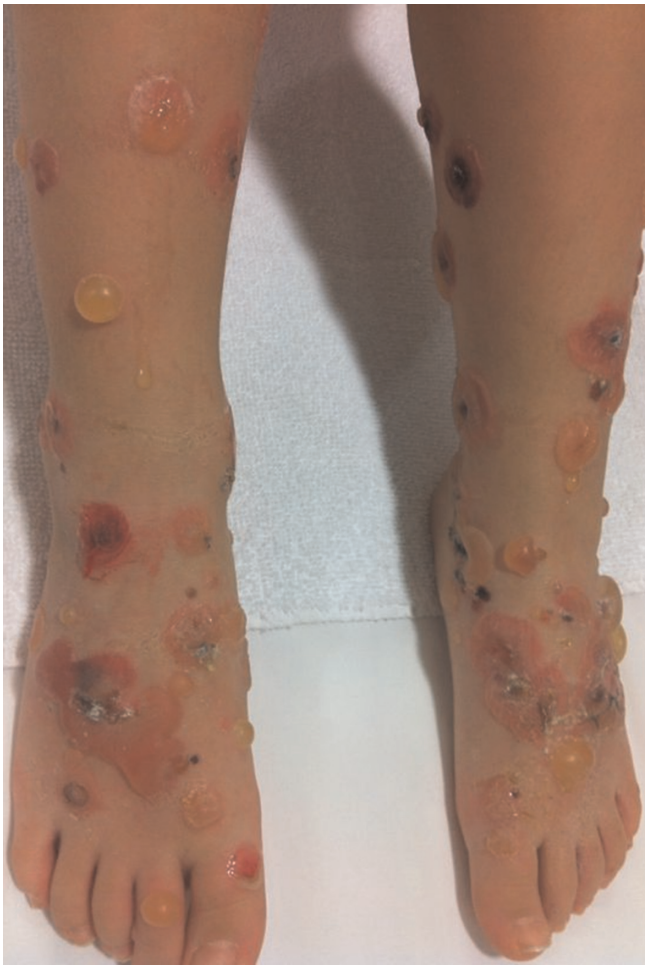
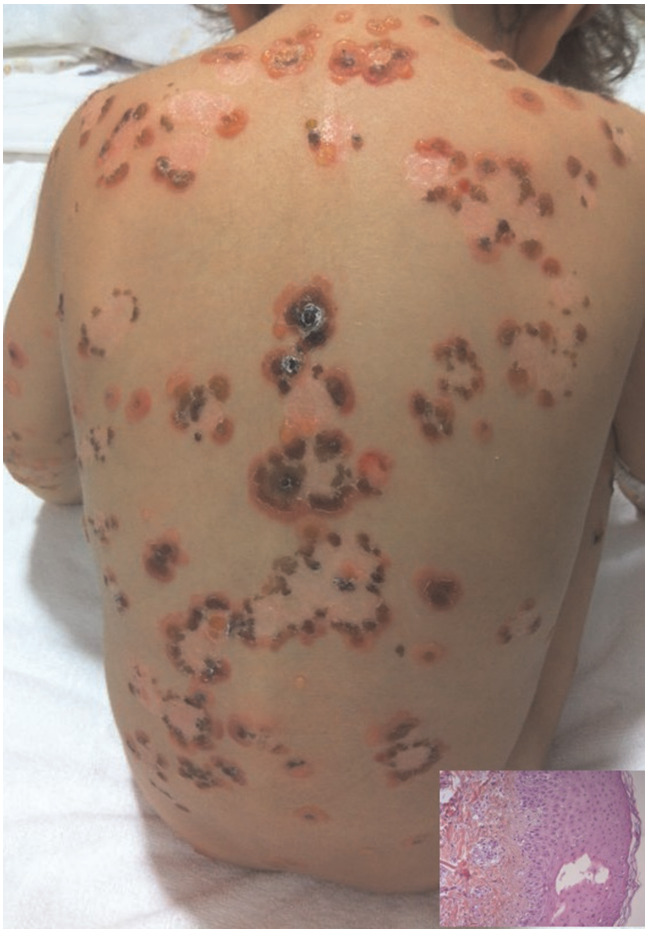


Abstract PO-0354 Figure 1



Abstract PO-0354 Figure 2

Abstract PO-0354 Table 1 Differential diagnosis of child subepidermal dermatosis

	Bullous Impetigo	Dermatitis herpetiformis	Bullous pemphigoid	Linear iga dermatosis
Clinical Features	Blisters of smooth walled content initially clear and later turbid	Pruritic papules and vesicles on the extensor surfaces of the limbs, buttocks, shoulders, nape of neck, scalp	Tense vesicles or bullae on erythematous base on the inner surface of the thighs, forearms, axillary folds, palms, soles	Vesicles or bullae on perigenital area, extremities, trunk, face
Aetiology	Staphylococcus aureus epidermolytic toxin	Inmunogenetic	Autoinmune	Autoinmune, drugs induced, infections, trauma and burns
Histology	Vesicular subcorneal pustules with accumulations of neutrophils	Subepidermal cavity with neutrophils in the dermal papillae	Subepidermal cavity with an inflammatory infiltrate, predominantly of eosinophils	Subepidermal cavity with neutrophils along the basement membrane vacuolar degeneration, eosinophils may be present
Antigen	None	Transglutaminase	230 kDa, 180 kDa	LAD-1 BP230 97-kDa, 120-kDa antigen
Dif	None	Granular deposition of IgA	Linear deposition of C3 and IgG along the basement membrane	Linear deposition of IgA along the basement membrane
Treatment	Topical and systemic antibiotic	Gluten free diet, oral dapsone	Topical and oral steroids. Oral dapsone	-Oral dapsone -Oral dapsone + oral steroids -Colchicine and intravenous immunoglobulins

Background and aims We studied the feto-placental interface, in Pregnancy Induced Hypertension (PIH), to present his specific structural modifications.
Method We have studied the microscopical modifications of 68 placentas obtained after delivery for two equal groups representing mothers with PIH and normotensive. The samples obtained by sections were specifically prepared for the study using three types of histological stains. We used optical microscopy for observing mainly the lumen of spiral arteriole and changes in its intimate and medial tunica.
Results We registered the following specific structural modifications in the pregnancies with PIH : fibrosis in the middle of the villosity, fibrinoid necrosis, condensation of stromal connective tissue, syncytial layer agglutinations of the villous or intervillous spaces (nodes, buds, or bridges), thrombosis and/or infarction of the spiral vessels and villous capillary endothelial atheromatosis.
Conclusions Our study was done to find a better understanding of the histo-logical changes of the preeclamptic feto-maternal interface concerning his role in PIH. The morphological modifications of the feto-placental interface in the PIH represent a marker of the fetal and postnatal hypoxia/ischemia with an immediate and late impact upon their cerebral development.

PO-0365 HISTOLOGICAL ASPECTS OF THE FETO-PLACENTAL INTERFACE IN THE GESTATIONAL DIABETES MELLITUS

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Background and aims We studied the feto-placental interface, in the Gestational diabetes mellitus (GDM), to present his specific structural modifications and his cellular injuries.
Method An optical microscopic analysis was performed on 30 placentas, obtained after delivery for two equal groups representing mothers with GDM and normal pregnancies. The samples obtained by sections were specifically prepared for the study using three types of histological stains. The histological observation centred upon the: trophoblast, villous stroma and fetal capillary. The statistical study of the data was performed using SPSS 17.0.
Results Through optical microscopy were identified varying degrees of lesions consisting of: villous oedema, proliferation and villous fibrosis of the capillaries, large number of syncytial knots, important fibrinoid necrosis, moderate fibrin thrombi, hyperplasia of the syncytiotrophoblast, chorangiosis, slightly thickened of the basement membrane of the feto-maternal interface.
Conclusion Histological changes in the placentas of women with GDM are significant factors contributing to fetal anoxia with impact on placental vascular permeability. A diabetic milieu causes vascular dysfunction, increasing angiogenesis in GDM is considerred to be the cause of the placental abnormalities and complications (miscarriage, stillbirth, macrosomia, and congenital anomalies).

PO-0366 PRADER-WILLI SYNDROME: 3 CASES STUDIES

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Backgroud and aims Prader–Willi syndrome is a highly variable genetic disorder affecting multiple body systems.
Aims Study the clinical diagnostic criteria and genetic testing confirmation of Prader–Willi syndrome.
Methods We report 3 cases of Prader-Willi syndrome over a period of 7 years between 2007 and 2013.
Results It's about 3 boys. The age of diagnosis was 15 days (first patient), 8 months (second patient) and 6 years (third patient). History of hypotonia with poor suck was found in 2 cases and recurrent respiratory tract infection in 1 case.