

## DIARRHEA, VOMING AND PAIN ABDOMEN : A CASE REPORT OF SCHMIDT'S SYNDROME

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**Abstract:** Schmidt's syndrome is a condition where primary hypothyroidism is combined with autoimmune primary adrenocortical insufficiency. A drowsy lady on thyroxine who presented with severe abdominal pain, diarrhea and vomiting, is reported as rare case, presumably first time, in Nepal.

### CASE REPORT

A 50 year old lady presented with stupor, vomiting, diarrhea and abdominal pain for two days; 5 years ago she was diagnosed as Sheehan's non complaint with replacement therapy of 200 µg/day thyroxine. Examination revealed blood pressure 80/60 mm Hg, pulse 60/min, pallor, patchy hair loss in occiput and hyperpigmentation of face, arms, knuckles (Fig.1,2) and oral cavity. The skin was coarse and cold with sparse auxiliary and public hair. Reflexes were slow and delayed; her serial biochemical work up is tabulated below (table).

She was managed with dextrose saline and thyroxine. In order to correct sodium and blood pressure, initially hydrocortisone followed by fludrocortisone, was administered. Gradually, she recovered with normal biochemistry and vitals. She was diagnosed to have type II polyglandular deficiency syndrome having adrenocortical insufficiency, hypothyroidism and alopecia areata.

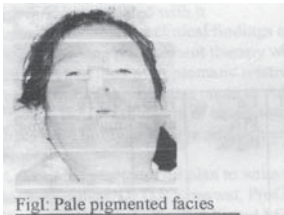


Fig1: Pale pigmented faces



Fig II: Hyperpigmented knuckles.

Table : Investigations

Date	Na + mEq/l	K+ mEq/l	Corlisol	FT3 pg/dl / fT4ng/dl/ TSH uIU/ml	FSH/LH/ U/l	Ca2+ / PO42- Mg%	FBS mg%	Tc/T g mg%	Ur/Cr mg%
24.3.00	108	5.1	<5	1/0.2/22	76.8/41.3	10.2/5.8	82	256/198	39/1.8
30.3.00	119	5.2					112		27/1.5
10.4.00	13	4.8		2.2/1.8/9			95		19/0.9

Low sodium/sugar and high urea/creatinine were normalized after hydrocortisone and fludrocortisone therapy. Low FT3 (normal : 1.4-4.2 pg/ml) and FT4(normal :0.8-2 ong/dl) and high TSH (normal ; 0.4-5 uIU/ml) were normalized after thyroxine (table) indicating primary hypothyroidism. Synacthen test revealed <5 ug/dl plasma cortisol before and after injection indicating primary adrenocortical insufficiency. Serum ACTH as well a thyroid and adrenal antibody tests were unavailable. FSH/LH (upper normal FSH(1 miu/ml) and LH (90 miu/ml level was high for age. Electrocardiography showed low voltage, bradycardia and first degree heart block. Ultrasonography of pelvis and upper gastrointestinal endoscopy including jejunal histology were normal

### DISCUSSION

Multiple endocrine and non endocrine glandular failure, in an individual

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patient or his family, had been recognized during the last century. Thomas Addison first described adrenocortical insufficiency features in pernicious anaemia patients. Martin Benno Schmidt in 1926, documented association between adrenocortical failure and thyroiditis Carpenter et. al. expanded Schmidt's syndrome to include insulin dependent diabetes. Schmidt's Type II autoimmune poly glandular syndrome (APS) infrequently occurs recessively in children. Besides thyroid and adrenocortical insufficiency, diabetes mellitus, alopecia areata, vitiligo, primary ovarian failure also occur in 40% of cases before the age of 50 years. Addisonian pernicious anemia occurs in 4% females. Two or more combinations of mainly Hashimoto's, Addison's disease, diabetes mellitus and infrequently alopecia areata, vitiligo, dermatitis herpetiformis, chronic active hepatitis, celiac disease, myasthenia gravis and Sjogren's syndrome constitute this syndrome. Clinical manifestations of such diseases serve as important clues to early diagnosis.

Dysfunction of T lymphocytes especially suppressor activity and development of thyroid, adrenal ovarian, melanocyte, pancreatic – B cells and gastric parietal cell autoantibodies lead to their destruction. Its prevalence in America is 14-20 per million population. Similar data are unavailable in India or Nepal; Grave's disease or Hashimoto's thyroiditis can involve thyroid. Sometimes, celiac sprue like chronic diarrhea is associated with it.

Strong corroborative clinical findings and laboratory results confirmed Schmidt's syndrome in our patient, she was put on life long replacement therapy with hydrocortisone and thyroxine.

Patients and their asymptomatic relatives need lifelong supervision and advice to ensure timely recognition of myxoedema coma or Addisonian crisis. In recent years, dramatic progress has been made in the identification of target antigens and epitopes involved in the autoimmune organ specific diseases. Intensive research for defective peripheral immune tolerance of these patients, is needed.

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