study parameters.

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SEZARY SYNDROME-CLINICO HISTOLOGICAL CORRELATION

Dear Editor,

This refers to the report on two cases of Sezary Syndrome (MJAFI, 1998;54: 76-78).

Sezary Syndrome (SS) comprises a triad of fiery red erythroderma, generalized lymphadenopathy and circulating atypical cells with cerebriform nuclei (Sezary cells) exceeding an absolute value of 1000/cumm or exceeding 10 percent of circulating cells [1].

Erythroderma, defined as an inflammatory skin disease which affects more than 90 percent of the body surface [2], is a characteristic feature of this syndrome. though 'multiple erythrodermic patches' with 'butterfly rash on face' in the first case and 'multiple erythematous scaly lesions' in the second case of the report do not indicate the extent of cutaneous involvement, these descriptions could well suggest the clinical impression of disseminated lupus erythematosus and psoriasis respectively; more so in the absence of generalised lymphadenopathy as was in both these cases. Cytomorphologically, Sezary cell is not pathognomonic of SS, as it can be seen in many benign dermatoses like chronic eczema, psoriasis, lupus erythematosus, parapsoriasis, atopic dermatitis, vasculitis, etc. [3].

SS, sometimes described as a leukaemic variant of Mycosis Fungoides (MF), is not only clinically and hematologically distinct from it, but also distinct from a histological view, thus discouraging the interchangeable use of both these terms. The infiltrate in SS is less pleomorphic than in MF; and eosinophils, plasma cells, macrophages and accessory cells are less frequent or lacking. [4]. Proliferation Kinetics of the dermal infiltrate using 3H-thymidine labelling (3H index) have revealed widely distinct patterns in both [5]. Lymph nodes in MF have a higher number of interdigitating reticulum cells and the ratio of mononuclear cells having cerebriform

nuclei to the interdigitating reticulum cells varies from 1:1 to 2:1, in contrast to a ratio of 5:1 to 10:1 in SS[6].

The case report serves to highlight the diagnostic dilemma a clinician might have to face when confronted with cutaneous markers of internal disease and also underscores the importance of multi disciplinary approach which is inevitable to overcome such situations.

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ECG DIAGNOSIS: ISORRHYTHMIC ARRHYTHMIA

Dear Editor,

A 52-year-old male patient was admitted with left sided hemiplegia caused by ischemic cerebral infarct along with hypertension. He was then placed on tab Atenolol and tab Disprin. On third day his electrocardiogram (ECG) revealed ventricular tachycardia. His subsequent ECG (Fig. 1) revealed AV junctional rhythm at the rate of 56 beats/min, inverted P-wave indicating retrograde atrial depolarisation, P-R interval 0.18 sec, and QRS duration 0.08 sec (Upper strip). This rhythm was interrupted by a normally conducted sinus beat indicating capture beat (first beat in second strip), a ventricular premature beat (third beat) followed by incomplete compensatory pause. It revealed AV dissociation with both atria and ventricle under control of separate pacemaker (Fig. 2). Rate of both pacemaker is 50 beats/min, however AV junctional rate is slightly faster than sinus rate hence P-wave is seen as marching through the QRS complexes. There is gradual shortening of R-P interval and there is no correlation between P-wave and QRS complexes. This is consistent with diagnosis of incomplete AV

Fig. 1: Electrocardiogram showing AV junctional rhythm

Fig. 2 : Second rhythm strip of ECG (initial 10 beats)

dissociation with interference-Isorhythmic arrhythmia.

AV dissociation occurs when atria and ventricle are under control of separate pacemakers[1]. The atria is under control of either sinus node, ectopic atrial or AV junctional rhythm. Incomplete AV dissociation is indicated when a single pacemaker establishes control of both atria and ventricle for either one beat (capture) or series of beats (sinus rhythm, AV junctional rhythm with retrograde atrial capture [2]. Our ECG reveals both the mechanism.

AV dissociation can occur by slowing of dominant pacemaker, acceleration of latent pacemaker, complete AV blocks and combination of above[2]. In our case it was due to combination of slowing of dominant pacemaker (second strip) and acceleration of latent

pacemaker (first strip). AV dissociation can develop with an AV junctional rhythm in response to severe sinus bradycardia; when the sinus rate and the escape rate are similar it is called isorhythmic arrhythmia[1]. It can occur in response to beta-blocker[1] which is probable cause in our case who was given tab atenolol.

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ATRIAL FIBRILLATION IN THYROTOXICOSIS

Dear Editor,

DPR, 45-year-old male, presented to peripheral hospital with signs and symptoms of thyrotoxicosis of one-month duration on 3rd March '97. He was not on any medication. His pulse was 140/minute irregularly irregular. Electrocardiogram confirmed presence of atrial fibrillation. there was no cardiomegaly on X-ray. There was no facility for thyroid function test, hence he was put on tab Neomercazole 10 mg tid and tab propranolol 40 mg tid on clinical ground. AF reverted to sinus rhythm on 7th day. He was referred to this centre for further evaluation on 27 March '97. His symptoms had improved but he was still clinically toxic and had sinus rhythm. His drugs were stopped to confirm diagnosis and evaluate for hyperthyroid state. While awaiting report of thyroid function test he developed palpitation and clinical examination and ECG revealed recurrence of AF. He was sent for thyroid scan, which showed diffuse thyromegaly with increased thyroid uptake-10%(normal 1-4%). His thyroid function confirmed thyrotoxicosis (Table 1). He was placed on tab Neomercazole 10-mg tid and tab propranolol 40-mg tid. Sinus rhythm was documented on third day. he was followed up for three months. He is presently euthyroid and maintained sinus rhythm.

TABLE 1
Thyroid hormone profile

Hormone	April 97	May 97	June 97	July 97
T3 (0.8 - 2.1 ng/ml)	2.95	2.61	2.56	2.19
T4 (4.5 - 13.5 ug/dl)	17.77	14.97	14.39	11.66
TSH (0.5 - 6.5 mIU/L)	0.13	0.12	0.18	0.32

Hyperthyroidism is major cause of paroxysmal AF. It is reported in about 9-22% patients of hyperthyroidism, occult or manifest [1]. The AF in thyrotoxicosis reverts spontaneously to sinus rhythm with

control of hyperthyroidism in 75% of patients within 13 weeks. The shortest duration of AF is reported for one-week [2]. Antithyroid drugs usually takes 2-6 weeks for achieving euthyroid status depending upon preformed and stored hormone level, and iodine content of the thyroid gland [3]. In present case AF reverted to sinus rhythm within 7 and 2 days after starting antithyroid drugs and beta-blocker. Euthyroid status was achieved after 12 weeks (Table-1), hence antithyroid drugs are unlikely to contribute to early reversion of AF to sinus rhythm. Beta-blockers are drug of choice for thyrotoxicosis induced arrhythmias. These drugs by reducing hyperadrenergic drive contribute largely to reversion of AF. The longer AF continues, more difficult it becomes to treat as it is said that AF begets AF [2]. It is therefore advised to treat AF early with beta-blockers in suspected cases of hyperthyroidism pending hormonal evaluation. Beta-blocker has effect on atrial action potential, increases its refractory period and acts as membrane stabilising agent, therefore is an agent of choice in atrial arrhythmias in hyperthyroidism.

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