study parameters.

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Gp Capt TS RAGHU RAMAN

Senior Advisor (Pediatrics). Command Hospital (Air Force), Bangalore-560 007

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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Lt Col KEDARNATH DASH, Wg Cdr GURCHARAN SINGH, Sqn Ldr SANJIV GROVER

Command Hospital Air Force, Bangalore-560007

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