CT, accuracy was 56.5%, sensitivity was 56%, and specificity 41.9%.[4]

In conclusion, EUS has the advantage of being portable, and often office-based, requiring only minimal preparation, and is well tolerated by the patient. Endorectal ultrasound has been shown to be the most accurate method for the determination of the depth of wall penetration, and is comparable for lymph node metastases. Therefore, it is an accurate method to preoperatively stage rectal cancers.

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Oxidative stress in patients with premature hair grayness

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Grayness of hair is usually a manifestation of the aging process, and is due to progressive reduction in melanocyte function. Premature grayness of hair (PGH) has been defined as onset of grayness before 20 years of age in Caucasoid, 25 years of age in Asian and 30 years of age in Negro.[1] The PGH is considered to be a variant of vitiligo that could be seen together or separately.[2] The systemic changes that could be seen in vitiligo, like elevated glycosylated hemoglobin, and abnormal lipoproteins, were also observed in PGH.[3] Several reports suggest that free radicals damage, and oxidative stress are involved in the etiopathogenesis of vitiligo.[4] The aim of the present study is to evaluate the oxidative stress, and free radicals damage in patients with PGH.

Sixty patients with PGH were included in this study, and were selected from outpatient clinic at the Department of Dermatology and Venereology, Baghdad Teaching Hospital, Iraq. The study conducted between April 2002 and April 2004. Their ages ranged between 11-29 years with a mean $\pm$ standard deviation (SD) of 24.55 $\pm$ 5.19. Of those 60 patients, 18 (30%) were females, and 42 (70%) were males. The severity of grayness of hair was graded according to the following: mild grayness: grayness of hair that can be noticed with difficulty, moderate grayness: grayness of hair that could be seen obviously with naked eye, severe grayness: grayness of hair that involved most of the scalp, and beard area in males. Eighteen patients (30%) had mild grayness, while 15 (25%) patients had moderate grayness, and other 27 (45%) patients had severe grayness. Patients with duration of grayness less than 2 years were considered as cases of acute PGH (15 patients), while patients with duration of grayness more than 10 years were considered as cases of chronic PGH (21 patients). A total of 60 healthy individuals were considered as control. They comprised of 16 (26.7%) females, and 44 (73.3 %) males. Their ages ranged between 15-30 years with a mean $\pm$ SD of 22.96 $\pm$ 3.86 years. Informed consent was obtained from all patients and controls.

Blood samples were collected from each patient, and control. Ten ml of venous blood was collected using 10 ml disposable syringes, after separation, the serum stored, and kept for further analysis. Serum glutathione, caeruloplasmin, malondialdehyde (MDA), copper, and zinc were measured. The significant difference between mean values was estimated by student's t-test. Pearson correlation coefficient (r) was used to test the relation between 2 parameters. The ANOVA test was used to compare the mean of parameters between grades. Serum MDA ($p<0.0001$), caeruloplasmin ($p<0.001$), and copper ($p<0.01$) were significantly higher in PGH than control while glutathione ($p<0.001$), and zinc ($p<0.001$) levels were significantly lower in patients than control. (**Table 1**). When patients with PGH were compared according to the severity of grayness (grades), serum glutathione were significantly higher in grade 3 (severe) PGH ($p<0.05$), while other parameters shows no significant difference between grades. When oxidative stress parameters were correlated

Table 1 - Oxidative stress parameters in chronic PGH in comparison with acute PGH and healthy controls.

Parameters	Acute PGH (3 months - 2 years)	Chronic PGH (more than 10 years)	Control	Patients
Malondialdehyde	2.11 ± 0.914*	1.27 ± 0.516†	1.18 ± 0.107	2.02 ± 0.947‡
Glutathione	0.52 ± 0.244*	0.84 ± 0.311†	0.85 ± 0.311	0.58 ± 0.482‡
Caeruloplasmin	29.38 ± 3.733**	27.11 ± 3.65†	26.93 ± 3.71	28.65 ± 3.57‡
Zinc	79.75 ± 3.63**	80.25 ± 3.21†	81.82 ± 3.63	79.90 ± 2.83‡
Copper	83.90 ± 2.29**	83.32 ± 2.18†	82.52 ± 2.10	83.68 ± 2.52‡

‡ - *p*-value in patients in comparison with control ($p< 0.05$), * - *p*-value in chronic PGH (21 patients) in comparison with acute PGH (15 patients) ($p< 0.05$), ** - *p*-value in chronic PGH (21 patients) in comparison with acute PGH (15 patients) (not significant)
† - *p*-value in chronic PGH (21 patients) in comparison with control (not significant),
PGH - premature grayness of hair.

with the duration of PGH, serum MDA ($p<0.001$), and caeruloplasmin ($p<0.001$) had a statistically significant negative correlation while serum glutathione levels showed a statistically significant positive correlation ($p<0.05$). Other parameters showed no significant correlation with duration. When oxidative stress parameters in patients with acute PGH (15 patients) were compared to that of chronic PGH (21 patients) MDA were significantly higher in acute PGH, glutathione were significantly lower in acute PGH. Other parameters (zinc, caeruloplasmin, and copper) showed no significant difference between acute, and chronic PGH. When oxidative stress parameters in patients with chronic PGH were compared to that of healthy control, there is no statistically significant difference in all parameters.

The PGH is a variant of vitiligo, and many biochemical changes (for example, elevated glycosylated Hb, and abnormal lipoproteins) that occur in vitiligo can occur in PGH.[4] Free radicals damage, and decreased antioxidants were reported in vitiligo.[4] These observation had encouraged us to conduct the present work. The present study showed that: MDA level (which reflect free radicals damage) was raised in mild PGH, and gradually decreased especially in severe grayness, also MDA level was high in early PGH, while it gradually declined as the grayness become chronic, and these finding were similarly reported in patients with vitiligo.[4] On contrary, antioxidant glutathione were low in early, and mild cases of PGH, and gradually raised to reach a normal level in chronic, and severe cases of PGH. Caeruloplasmin level were high in early cases of PGH, and gradually declined in chronic cases of PGH (reaching the level of healthy controls). These interesting results had been also reported in patients with vitiligo.[4]

In conclusion, the changes in free radicals damage, and antioxidants were comparable in patients with vitiligo, and PGH. Also these findings do confirm that the etiopathogenesis of vitiligo, and PGH is similar.[5]

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